Increasing propensity to mind-wander by transcranial direct current stimulation: A registered report

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Abstract

Transcranial direct current stimulation (tDCS) has been proposed to be able to modulate different cognitive functions. However, recent meta-analyses conclude that its efficacy is still in question. Recently, an increase in subjects' propensity to mind-wander has been reported as a consequence of anodal stimulation of the left dorsolateral prefrontal cortex (Axelrod et al., 2015). In addition, an independent group found a decrease in mind wandering after cathodal stimulation of the same region. These findings seem to indicate that high-level cognitive processes such as mind wandering can reliably be influenced by non-invasive brain stimulation. However, these previous studies used low sample sizes and are as such subject to concerns regarding the replicability of their findings. In this registered report, we implement a high-powered replication of Axelrod et al. (2015)'s finding that mind-wandering propensity can be increased by anodal tDCS. We used Bayesian statistics and a pre-registered sequential-sampling design resulting in a total sample size of

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N=192 participants collected across three different labs. Our findings show support against a stimulation effect on self-reported mind-wandering scores. The effect was small, in the opposite direction as predicted and not reliably different from zero. Using a Bayes Factor specifically designed to test for replication success, we found strong evidence against a successful replication of the original study. Finally, even when combining data from both the original and replication studies, we could not find evidence for an effect of anodal stimulation. Our results underline the importance of designing studies with sufficient power to detect evidence for or against behavioral effects of non-invasive brain stimulation techniques, preferentially using robust Bayesian statistics in pre-registered reports.

Keywords: mind wandering, tDCS, DLPFC, non-invasive brain stimulation

1. Introduction

Mind wandering can be tentatively defined as a shifting of the attentional focus from external task demands to internal thoughts (Smallwood & Schooler, 2006). Episodes of mind wandering are very common during activities of daily life (Killingsworth & Gilbert, 2010) and during experimental tasks. Depending on various factors such as task difficulty (Feng et al., 2013) and mood (Smallwood et al., 2009), the percentage of time we spend mind wandering is estimated to be between 30% and 50%. In recent years, much interest has focused on the neural basis of mind wandering (Mason et al., 2007; Christoff et al., 2009; Mittner et al., 2014). One consistent finding is that mind wandering involves the default-mode network (DMN; Raichle et al., 2001), a network of brain areas that are activated during internal mentation (Buckner et al., 2008; Andrews-Hanna et al., 2010; Andrews-Hanna, 2012). The finding that activity in these areas is increased has been replicated in several independent studies employing different tasks and methodologies (Weissman et al., 2006; Christoff et al., 2009; Mittner et al., 2014).

Less well understood is the role of the frontoparietal control network (FPN; Vincent et al., 2008; Spreng et al., 2010) which also seems to be involved in the initiation and sustenance of mind wandering (Smallwood et al., 2012). Several studies have linked perceptual awareness to the propagation of stimulus-induced neural activity to the FPN, representing a “global workspace” that provides conscious access to cognitive representations (for reviews see: Baars et al., 2013; Dehaene et al., 2006; Dehaene & Changeux,

24 2011). During mind wandering, [Smallwood et al. \(2012\)](#) argue that the
25 FPN might determine the contents of consciousness and serve as a common
26 workspace for both internally focused trains of thoughts (associated with
27 the DMN) and externally-guided cognition (operated by the dorsal attention
28 network; DAN). In this view, the FPN is a flexible network that contributes
29 to switches between different modes of the brain: An internally directed,
30 decoupled mode (DMN) and an externally-focused mode during which ac-
31 tivity in the DAN is increased. The dorsolateral prefrontal cortex (DLPFC)
32 is a key region of the FPN and has been hypothesized to be essential in
33 initiating and sustaining internal trains of thoughts, consequently leading to
34 attenuated processing of external stimuli (perceptual decoupling; [Smallwood
35 et al., 2012](#)). Based on this theory, it can be hypothesized that modulating
36 the excitability of the DLPFC could affect the frequency and/or length of
37 mind-wandering episodes. However, because the FPN is supposedly crucial
38 both for the maintenance of an externally-focused and an internally-focused
39 state, it is theoretically unclear whether mind wandering would be facilitated
40 or inhibited using neuromodulation.

41 Recently, three interesting studies ([Axelrod et al., 2015](#); [Kajimura & No-
42 mura, 2015](#); [Kajimura et al., 2016](#)) investigated this question empirically us-
43 ing transcranial direct current stimulation (tDCS). This non-invasive brain
44 stimulation technique is thought to be capable of inducing robust excitability
45 changes in the stimulated neural tissue ([Stagg & Nitsche, 2011](#)) by modulat-
46 ing synaptic efficacy and inducing synaptic plasticity. Intriguingly, [Axelrod
47 et al. \(2015\)](#) could show an increase in the propensity to mind wander (as
48 measured by self-reports) during a sustained attention task when anodal
49 tDCS was applied above the DLPFC relative to two control conditions, a
50 sham (inactive) stimulation and stimulation of the occipital cortex. This
51 finding would seem to support the theory reviewed above: Higher excitabil-
52 ity of the DLPFC (induced by anodal tDCS) in this framework could lead to
53 a better ability of the FPN to suppress distracting perceptual stimuli and/or
54 to maintain the ongoing train of internal thoughts. Furthermore, [Kajimura
55 & Nomura \(2015\)](#) and [Kajimura et al. \(2016\)](#) investigated similar questions in
56 a different experimental setup and found a pattern of results that is comple-
57 mentary in the sense that they observed reduced frequency of task-unrelated
58 thoughts after applying cathodal tDCS above the left DLPFC relative to
59 anodal stimulation. Together these findings appear to provide evidence for
60 [Smallwood et al. \(2012\)](#)'s theory and can be seen as a major advance in the
61 understanding of the neural correlates of mind-wandering episodes.

62 The result that mind-wandering propensity can be influenced by tDCS
63 has important implications both for basic neuroscience and in more applied
64 settings. In the scientific literature, the finding has attracted the attention
65 of several leading researchers (Fox & Christoff, 2015; Broadway et al., 2015),
66 with 51 independent citations so far. In their commentary on Axelrod et al.
67 (2015), Fox & Christoff (2015) argue that changes in meta-awareness in-
68 duced by the stimulation of DLPFC might be responsible for the observed
69 changes. Similarly, Broadway et al. (2015) are enthusiastic about Axelrod
70 et al. (2015)’s finding and argue that it “[...] marks a new era for re-
71 search into mind wandering and previews some of the insights that contin-
72 ued methodological advances will likely make possible”. We believe that such
73 strong endorsements from leading researchers in the field are likely to result
74 in a surge of research activity building on Axelrod et al. (2015)’s result. From
75 a more applied perspective, mind wandering has been, e.g., associated with
76 accidents in car driving (Yanko & Spalek, 2014; He et al., 2011) and avia-
77 tion (Wiegmann et al., 2005) and a technique that consistently and reliably
78 allows to manipulate the propensity to mind-wander has thus great poten-
79 tial to avoid many of these human errors. Furthermore, ruminations, which
80 may be seen as a special case of mind wandering, are core features of clinical
81 conditions such as major depression or obsessive-compulsive disorder. There-
82 fore, a technique to reliably influence such processes could open up exciting
83 avenues towards better treatment alternatives.

84 However, all of these considerations rest on the validity and most im-
85 portantly the replicability of the observed effects. Although the findings
86 summarized above have great potential influence, the evidence so far is in-
87 conclusive because it is based on clearly underpowered studies. Concretely,
88 the studies used a low sample size (about N=10-20 per group) such that the
89 results could very well be the result of random fluctuations. In addition,
90 even though Axelrod et al. (2015) replicated their main result in a second
91 experiment, Kajimura & Nomura (2015) and Kajimura et al. (2016) failed
92 to replicate Axelrod et al. (2015)’s findings when using anodal stimulation
93 of the DLPFC relative to a sham condition (though the effect was in the
94 expected direction and the replication was not a direct one). Based on these
95 arguments, we believe that a conclusive, high-powered replication of Axelrod
96 et al. (2015)’s finding is essential for establishing a sound basis on which
97 future researchers can advance the understanding and application of tDCS
98 in the setting of mind wandering (or avoid spending unnecessary resources
99 should the effect prove to be unstable).

100 Pre-registered replications are considered to be the best way to establish
101 a firm basis for the existence of an effect and they provide a rigorous way to
102 avoid the problems underlying the low replicability rate in psychology (Si-
103 mons et al., 2014; Chambers et al., 2014; Nosek & Lakens, 2014). The need for
104 rigorous replication may be further motivated by the recent meta-analytical
105 findings in the field of tDCS. After an enthusiastic explosion of studies apply-
106 ing tDCS to affect many cognitive functions and psychiatric diseases, recent
107 meta-analytic studies draw much more cautious conclusions (Tremblay et al.,
108 2014; Horvath et al., 2015a,b). In fact, Horvath et al. (2015b) question the
109 very existence of any effect of tDCS on cognition. However, stimulation pa-
110 rameters and tasks are diverse and strong conclusions cannot be made at
111 this point in time and Horvath et al. (2015b) conclude with an urgent call
112 for more direct replications in the field of tDCS. Finally, a review focusing
113 exclusively on stimulation of the DLPFC (the target region of Axelrod et al.,
114 2015) found very variable effects and “[...] sometimes apparent conflicting re-
115 sults” (Tremblay et al., 2014). Clearly, direct, pre-registered replications are
116 necessary to be able to identify findings that are reliable in this important
117 field.

118 Our project aimed to replicate the finding reported by Axelrod et al.
119 (2015). For this purpose, we conducted a multi-center study (measuring
120 in Tromsø, Amsterdam, and Göttingen) using identical experimental setups
121 following a pre-registered protocol in order to pool an appropriately large
122 sample size. We used Bayesian methods to estimate the effect size of anodal
123 stimulation and to establish success or failure of the replication attempt
124 (Verhagen & Wagenmakers, 2014).

125 2. Methods

126 All materials, simulations and analyses are available in a public repository
127 hosted by the Open Science Framework (OSF) at <https://osf.io/dct2r/>.
128 The repository was registered (frozen) before data collection such that none
129 of the materials can be covertly changed after data has been collected. The
130 link to the registered version of the project is <https://osf.io/bv32d/>.

131 2.1. Participants

132 Participants were collected from the respective subject-recruitment facil-
133 ities of three universities, the university of Tromsø (UiT), the university of

134 Amsterdam (UvA) and the university of Göttingen (UniGö). Ethical ap-
135 proval for the study was granted at all three universities. Based on our
136 design analysis (see below), we applied a sequential data collection protocol
137 (Schönbrodt & Wagenmakers, 2018; Schönbrodt et al., 2017) and set out to
138 collect between at least 120 and maximum 192 participants (a minimum of 20
139 and maximum of 32 participants per stimulation condition and study site).
140 Subjects who failed to provide a complete dataset for technical (e.g., failure
141 of the equipment) or other reasons (e.g., experiment not completed) were ex-
142 cluded from the analysis and replaced by new subjects. Specifically, in order
143 to be included in the experiment, all of the following conditions needed to
144 be satisfied for a participant:

- 145 • the participant did not have any neurological/psychiatric diseases (based
146 on self-report)
- 147 • participants did not have previous experience with tDCS (to increase
148 the efficacy of blinding)
- 149 • the participant was between 18 and 40 years old
- 150 • the participant completed the experimental session
- 151 • the stimulation equipment was functional across the complete session
- 152 • the data collected by the experimental computer was complete
- 153 • the participant complied with the instructions

154 After recruitment, participants were randomly allocated to either a sham
155 or an anodal DLPFC stimulation condition according to a randomization
156 list.

157 2.2. Apparatus

158 As the experiment was conducted across three separate locations, we
159 enforced similar conditions in the three labs by fixing specifications for the
160 apparatus and environment (see [experimental_setup.pdf](#)). These were set
161 up in collaboration with the authors of the original study to be as close to
162 the original experiment as possible. First, we required a quiet room free
163 from distracting elements. No one besides experimenter and participant was
164 allowed to enter the room during the study. In addition, optimal lighting
165 conditions was ensured (avoid, e.g., frontal lighting that may be disturbing).
166 Standard 19" flat-screen monitors were used in the study and the size of
167 the stimuli was adjusted by the experimental program to ensure that the
168 stimuli were presented in equal size on the retina. The experimental computer

169 ran identical versions of PsychoPy (release 1.83.04; [Peirce, 2007](#)) and the
170 experimental software and experimenters were encouraged to make sure that
171 the computer did not run any unnecessary background processes. Finally,
172 all participants wore earplugs to minimize the influence of environmental
173 noise, which they inserted once they read the instructions and possibly asked
174 questions.

175 We also provided comprehensive, standardized instructions for the ex-
176 perimenters (see [experimenter_instructions.pdf](#)) for running the experi-
177 ments. All experimenters were required to read the instructions and practice
178 testing on at least two pilot subjects before acquiring real data. Experimenter
179 interaction were kept at a minimum and instructions were delivered electroni-
180 cally to ensure a standardized procedure. There were, however, opportunities
181 for the participant to receive clarification and ask questions (prompted by
182 the experimental computer). A list of possible questions and standardized
183 answers that were given by the experimenters is available at [q_and_a.pdf](#).

184 The study used the Sustained Attention to Response Task (SART) which
185 is a variant of the Go/Nogo task that is very commonly used in mind wander-
186 ing research ([Smallwood & Schooler, 2006](#)). In this task, numbers between
187 0 and 9 were presented in the center of the screen in quick succession. The
188 participant was required to respond to each stimulus by pressing a button
189 (Go-trials) except when the target number “3” was displayed. In this case,
190 the response was to be withheld completely (Nogo-trials). No feedback about
191 the correctness of a response was given and the stimuli stayed on screen for
192 a fixed period of time, irrespective of the users’ response. In the context of
193 mind-wandering studies, brief self-reports (“thought-probes”) were presented
194 occasionally during the course of the experiment. These probes consisted
195 of a single question, “To what extent have you experienced task-unrelated
196 thoughts prior to the thought-probe?” and were answered on a scale from
197 “1” (minimal) to “4” (maximal).

198 In accordance with [Axelrod et al. \(2015\)](#), stimuli were presented in black
199 (RGB: [0,0,0]) on a gray background (RGB: [104,104,104]). The stimuli were
200 presented in the center of the screen and covered 3 degrees of visual angle.
201 The subject’s distance to the monitor was fixed at 60 cm and the maximum
202 length of the stimuli was readily determined to be 3.14 cm so as not to exceed
203 3 degrees. Stimulus duration was set to 1 s and an inter-stimulus interval
204 of 1.2 seconds was used. We provided scripts that test timing and size of
205 stimuli ([teststimsize.py](#)) and required the experimenters in each lab to
206 run these scripts before data acquisition to ensure comparability.

- 230 • the number of target trials (Nogo-trials) was fixed at 24, 12 per 20 min
231 period
- 232 • given these constraints and a total duration of 40 minutes, 1000 non-
233 target trials were presented: $24 \text{ thought-probes} \times 6 \text{ s} + 24 \text{ targets} \times$
234 $(1.0\text{s}+1.2\text{s}) + 1000 \text{ non-targets} \times (1.0\text{s}+1.2\text{s})=39 \text{ min}, 57 \text{ s}$
- 235 • trial-presentation was divided into 48 blocks (not known to the partic-
236 ipants) of unequal length
 - 237 – each block consisted of a variable number of non-target trials
238 (mean 20, sd 5.69, min 12, max 29)
 - 239 – non-target stimuli were independently drawn from the set $\{0, 1,$
240 $2, 4, 5, 6, 7, 8, 9\}$ with equal probability
 - 241 – each block ended either in a target-trial (stimulus “3”) or a thought-
242 probe
 - 243 – target-blocks and thought-probe blocks were presented in a pseudo-
244 random manner so that 3 blocks with target stimuli and 3 blocks
245 with thought-probes were appearing randomly in a set of 6 blocks
246 ensuring that thought-probes were not presented exclusively at
247 the beginning/end of the experiment, typically associated with
248 reduced/increased frequency of mind wandering, respectively
- 249 • the number of non-targets across blocks was in addition constrained
250 such that a total of 500 non-target trials were used across 24 blocks
251 (such that the durations of the two halves of the experiment were iden-
252 tical)
 - 253 – this was achieved by repeatedly drawing 24 samples from a trun-
254 cated normal-distribution (truncated to lie between 12 and 29)
255 until the sum of their rounded values equaled 500
 - 256 – this procedure was repeated for each half of the experiment

257 Before the start of the experiment proper, there was a short training
258 session of four blocks containing 2 targets and 2 probes (84 trials in total).

259 A Python-script using the PsychoPy library (Peirce, 2007) implementing
260 this procedure is available at [sart.py](#). Instructions were translated into
261 Dutch, German and Norwegian by native speakers (complete instructions
262 and the English template used to derive the local instructions can be found
263 in [instructions_en.py](#)).

2.3. Additional measures

After completing the experimental procedure, participants were required to complete three questionnaires: One measuring the mood of the participants, a state-mindfulness questionnaire and an own questionnaire referring to the content of the mind-wandering episodes that the participants experienced. The analyses (e.g., correlations between questionnaire scores and thought-probe responses or parameters of task performance) carried out on these additional measures were not pre-registered and are reported as exploratory.

Similar to the study by [Kajimura & Nomura \(2015\)](#), The Positive and Negative Affect Schedule (PANAS; [Watson et al., 1988](#)) was used for measuring the mood of our subjects. We used this scale, because of the link between prefrontal activity, task-unrelated thoughts and emotion regulation: First, there seems to be a bidirectional causal link between mind wandering and negative mood states ([Killingsworth & Gilbert, 2010](#); [Smallwood et al., 2009](#)). Second, there is converging evidence that the DLPFC plays a critical role in the top-down control of emotion ([Okon-Singer et al., 2015](#)), which is in accordance with the fact that symptom severity in major depression was quite consistently reduced by anodal tDCS applied over the left DLPFC (for reviews and controversies see: [Brunoni et al., 2012](#); [Berlim et al., 2013](#); [Shiozawa et al., 2014](#)). Finally, two recent study results showed that tDCS applied over the DLPFC can influence the frequency of ruminative thoughts of negative emotional content in healthy volunteers ([Kelley et al., 2013](#); [Vanderhasselt et al., 2013](#)). In this regard, monitoring mood changes in studies investigating the effects of non-invasive brain stimulation on mind-wandering propensity seems to be inevitable.

The PANAS scale consists of 20 items (10-10 describing positive or negative emotional states), which are to be rated from 1 (very slightly or not at all) to 5 (extremely). Positive and negative mood scores are calculated separately, and these values are used to assess the current or past mood states of the participants. We hypothesized that increasing intensity of negative feelings during the experiment would be associated with an increase in mind-wandering propensity in the anodal tDCS condition. Therefore, we asked our subjects to complete the PANAS twice: First for measuring their current (post-SART) mood (“how do you feel right now”), and second, to retrospectively measure their baseline (pre-SART) mood (“how did you feel at the beginning of the experiment”). Given that the completion of the PANAS in itself might induce subtle mood changes, we decided not to use

302 it before the main experiment in order to avoid interference with the repli-
303 cation attempt. The PANAS scale is available in the Dutch (Engelen et al.,
304 2006), German (Janke & Glöckner-Rist, 2014) and Norwegian (Gullhaugen
305 & Nøttestad, 2012) languages and the translated versions were used at each
306 of the three locations.

307 We also asked the participants to complete the Mindful Attention and
308 Awareness Scale (MAAS; Brown & Ryan, 2003), which is a 15-item scale
309 designed to measure an individual’s disposition to attend to the present
310 experience and overcome disrupting stimuli or internal states. It has pre-
311 viously been shown that MAAS scores negatively correlate with both the
312 frequency of self-reported mind wandering and behavioral measures (e.g. re-
313 sponse time variability, SART errors) of mind wandering (Mrazek et al.,
314 2012). Because low MAAS scores are considered to be indicative of an in-
315 creased mind-wandering trait that is stable over time (Brown & Ryan, 2003),
316 MAAS scores are expected to correlate with mind-wandering frequency in the
317 sham tDCS condition only. Moreover, the absence of correlations between
318 the MAAS and self-reported mind-wandering propensity in the anodal tDCS
319 condition would indicate that the effect of tDCS is independent of trait-like
320 inter-individual differences. The MAAS is available in Dutch (Schroevens
321 et al., 2008), German (Michalak et al., 2008) and Norwegian (Verplanken
322 et al., 2007).

323 Finally, because periods of mind wandering are not uniform in nature
324 and distraction from the task can be induced by disturbing external stimuli
325 (Stawarczyk et al., 2011) such as tDCS electrodes placed on the forehead,
326 we also asked the participants to freely report the content of their mind
327 wandering during the task. We also used 4 additional questions with 7-item
328 Likert scales (1: not at all, 4: to a medium degree, 7: extremely) to estimate
329 the degree to which participants were (1) thinking about task context (e.g.,
330 task difficulty, reflections on task performance, etc.), (2) distracted by tDCS
331 (e.g., skin itching, tingling, skin wetness, etc.), (3) distracted by other stimuli
332 (e.g., noises, visual stimuli, body sensations such as thirst or back pain, etc.)
333 and (4) thinking about personal issues (e.g., past memories, future plans,
334 etc.). Also, we asked the participants to guess whether they received real
335 or sham stimulation using a 7-item Likert scale (1: sham, 4: don’t know, 7:
336 real). With these questions we aimed to exclude the possibility that the effect
337 of tDCS on mind-wandering propensity was in fact related to the unpleasant
338 sensations caused by the stimulation or by the participants’ expectations
339 about stimulation-related effects (Turi et al., 2014). This questionnaire and

340 a translation into the three local languages can be found at [additional_](#)
341 [questions_English.pdf](#).

342 *2.4. Stimulation protocol*

343 The stimulation protocol adhered to the one reported in [Axelrod et al.](#)
344 (2015), with only minor modifications. All three labs used an identical model
345 of the NeuroConn DC stimulator (<https://osf.io/n4pbd/>). To deliver the
346 current, we used rubber electrodes (cathode: 7×5 cm; anode: 4×4 cm) with
347 conductive paste (Ten20; Weaver and Company, USA). One of the electrodes
348 was placed above position F3 (according to the International 10-20 system
349 used in electroencephalography, EEG), the other above the right supraorbital
350 area. The position of the stimulation electrode positioned at F3 was mea-
351 sured by applying the adequately sized EEG cap (circumference 56, 58 or 60
352 cm) on the participant’s head. The EEG cap was chosen based on measuring
353 the circumference of each participant’s head. After marking the F3 posi-
354 tion, the EEG cap was removed and the center of the stimulating electrode
355 corresponded to the F3 position. In addition, the edges of both electrodes
356 were precisely measured and marked which served as the landmark points
357 for preparing the electrode-skin interface. The skin in the predefined surface
358 regions were gently cleaned by using alcohol and cotton swab without over-
359 abrading the skin. A small amount of conductive paste was homogeneously
360 distributed over the previously cleaned skin surface and the rubber electrode
361 surface to ensure good contact between them. The electrodes were pressed
362 firmly with medium pressure to the head in order to adhere the electrodes
363 to the skin. To ensure that the conductive paste was distributed only over
364 the predetermined regions, the extra conductive paste was wiped-off. Con-
365 nector position was from anterior to posterior direction for the F3 electrode,
366 and from right supraorbital to right temporal lobe direction for the return
367 electrode. Impedance values were kept below 10 k Ω , subjects exceeding this
368 threshold were not included in the study.

369 In the anodal stimulation condition, participants received 20 minute-long
370 continuous stimulation at 1.0 mA intensity with 30 s fade-in and 30 s fade-
371 out periods, whereas the sham protocol applied the fade-in and fade-out
372 periods and the minimum possible stimulation duration of 15 s. As the
373 study uses double-blind design, the stimulators ran in study-mode where each
374 stimulation protocol was arbitrarily linked to a letter and secured with a 5-
375 digit code. The Neuroconn DC stimulator has certain hardware limitations,
376 that did not allow standard blinding using the 5-digit codes if the exact

stimulation parameters described by [Axelrod et al. \(2015\)](#) were to be used. More specifically, the pseudo-stimulation mode accessible by the 5-digit codes produces a sham protocol with a stimulation duration of 40 s in addition to the fade-in and fade-out periods, which was not desirable. Therefore, part of the stimulator’s display was covered with non-transparent tape to avoid the experimenter getting feedback about which condition was currently been run. Details about preparing and using the stimulator are available at [experimental_setup.pdf](#) and [experimenter_instructions.pdf](#). The mapping between stimulator code and stimulation mode were only accessible to a single researcher from each lab that was also responsible for programming the device but not involved in data-acquisition.

2.5. Statistical Methods

We used exclusively Bayesian statistics because of their many advantages compared to the more commonly used null-hypothesis testing (NHST) approach (see e.g., [Gelman et al., 2013](#); [Kruschke, 2014](#)). In addition, we report standard frequentist statistics for comparability with the original study.

All pre-registered analyses discussed in the following were implemented as scripts in the R programming language ([R Core Team, 2015](#)) using the BayesFactor package ([Morey & Rouder, 2015](#)) and Stan ([Carpenter et al., 2017](#)) as the modeling backend and R-packages `rstan` ([Stan Development Team, 2016](#)) and `brms` ([Bürkner et al., 2017](#)) for interfacing Stan from R. The replication and meta-analytic Bayes factors were calculated using code provided by [Verhagen & Wagenmakers \(2014\)](#) on their webpage (http://www.josineverhagen.com/?page_id=76). A listing of the exact version of R and all packages used are provided in the file `versions_used.txt` as generated by script `print_versions.R`. The analysis scripts were developed using data generated by pilot subjects using the final experimental software. After the data was collected, these scripts were supposed to be executed without changes (only the pilot data-files exchanged with the real ones) and the results reported. However, several minor adjustments to the analysis scripts were necessary because of coding errors and changes in the analysis-packages used. All such changes are summarised in the Appendix and details are available in the form of difference files in our OSF repository. Both the raw data and all output of the analysis scripts were stored and uploaded to OSF and the quantities described in the following sections reported in the results section of this paper.

413 2.5.1. *Effect of anodal stimulation on self-reported mind wandering*

414 The main result of this study concerns the comparison of the groups re-
 415 ceiving sham and anodal stimulation of the left prefrontal cortex in terms of
 416 their mean self-reported thought-probe scores. The original study (Axelrod
 417 et al., 2015) found that propensity to mind-wander (as measured by the mean
 418 of a subjects’ responses to all thought-probes presented during the experi-
 419 ment) was increased for subjects receiving anodal stimulation. We tested this
 420 prediction using a directed Jeffreys-Zellner-Siow (JZS) Bayes Factor (Rouder
 421 et al., 2009) that tests the hypotheses that (1) the effect is in the expected
 422 (positive) direction against the hypothesis that (2) the effect is either zero or
 423 in the unexpected (negative) direction. We supplemented the analysis with
 424 BFs quantifying the evidence in support of the hypothesis that the effect is
 425 positive or negative compared to exactly zero and an interval estimate for
 426 the effect size.

427 In particular, we first calculated a directed Bayes Factor, BF_{directed} , test-
 428 ing the hypothesis that the result of subtracting the mean thought-probe
 429 responses of the anodal group from that of the sham group is larger than
 430 zero against the hypothesis that it is less or equal to zero (Morey & Rouder,
 431 2015). We used a prior with an r-scale parameter of $\sqrt{2}/2 = 0.707$ that
 432 assumes that effect sizes are distributed according to a Cauchy-distribution
 433 with scale 0.707. This choice of prior was motivated by the fact that observed
 434 effect-sizes in tDCS studies are mostly small or medium (e.g., the absolute
 435 value of effect-sizes for cognitive effects of DLPFC stimulation reported by
 436 Horvath et al. (2015b) were on average 0.4). In case this BF is larger than 1,
 437 we found evidence for a positive effect of anodal stimulation. Values smaller
 438 than 1 quantify evidence for a negative effect. In case the real underlying
 439 effect-size is zero, the BF_{directed} is likely to be inconclusive because there is
 440 similar amount of evidence for a positive or a negative effect, respectively.

441 Therefore, to better evaluate evidence for zero effect of stimulation, we
 442 calculated two BFs testing the hypotheses that the effect is zero, against the
 443 existence of a positive ($BF_{\text{null}+}$) or negative effect ($BF_{\text{null}-}$). We used the
 444 same prior distribution as before. BFs larger than one quantify evidence for
 445 the hypothesis that the effect is zero while a BF lower than one indicates
 446 evidence for a positive ($BF_{\text{null}+}$) or negative effect ($BF_{\text{null}-}$). Thus, while
 447 the previous BF_{directed} directly tests the hypothesis predicted by the original
 448 study, this BF tests for the absence of any effect.

449 In addition, we used a final, undirected model (comparing any effect

450 against a null-effect) to extract an estimate for the posterior distribution
 451 of effect sizes which we quantified by its mean and highest-density interval
 452 (HDI). This estimate produced a range of values that contains the real ef-
 453 fect size with 95% probability given that the model is correct and assigns
 454 probabilities to each of those values. Therefore, we can exclude values falling
 455 outside of the 95% HDI with high probability.

456 The four measures described so far are quantifying slightly different as-
 457 pects of the data but are, of course, not independent. If the directional
 458 BF_{directed} is large, we expect the posterior HDI to be mostly or completely
 459 positive, the $BF_{\text{null}+}$ to be well below one and $BF_{\text{null}-}$ to be inconclusive.
 460 Conversely, in case of high BFs in favor of the null-hypothesis, we expect a
 461 lower BF in favor of a positive effect and a posterior distribution (HDI) that
 462 includes zero.

463 In addition to these analysis, we calculated the replication Bayes Fac-
 464 tor developed in [Verhagen & Wagenmakers \(2014\)](#). This Bayes Factor,
 465 $BF_{\text{replication}}$, pitches two competing theories against one another: A theory
 466 that a proponent of the original study might hold (i.e., that the replication
 467 effect size will be in line with the distribution of effect sizes implied by the
 468 original study) and a skeptic’s null-hypothesis that the effect size does only
 469 deviate randomly from zero. The advantage of this BF is that it directly
 470 tests the question whether or not the results of the original study have been
 471 replicated or are more likely the result of random fluctuations. However, the
 472 test is likely to be inconclusive when the effect size observed in the replication
 473 is much lower than that from the original study (which is often likely, given
 474 the “significance filter” ensuring that published effect sizes that are based
 475 on low sample size are large; [Gelman & Carlin, 2014](#)). This is in line with
 476 the finding that underpowered studies might be unfalsifiable per se ([Morey
 477 & Lakens, 2016](#)). For this reason, we calculated this $BF_{\text{replication}}$ only as a
 478 secondary measure of replication success as it was likely to be inconclusive.
 479 Only when the difference between the original effect size and the obtained
 480 one is large enough compared to that between zero and the replication effect
 481 size, the replication BF favors the null-hypothesis instead of the presence of
 482 an effect.

483 Finally, we were interested in the total amount of evidence for the pres-
 484 ence of an effect when pooling both the original study and the replication
 485 attempt (because the two studies are very similar, data can be assumed to
 486 be exchangeable). For this purpose, the fixed-effect meta-analytic Bayes fac-
 487 tor BF_{meta} ([Rouder & Morey, 2012](#)) has been developed which merges the

original and the new data. The original study showed strong support for the presence of an effect, possibly because of the significance filter that ensures large effect-sizes of significant findings (Gelman & Carlin, 2014). Therefore, we expected the BF_{meta} to be biased in favor of a positive effect (Nuijten et al., 2015) and the results from the BF_{meta} received less weight when drawing conclusions from our analyses.

The script for the analyses described here is available at [anodal_mw.R](#).

2.5.2. Design Analysis

The previous section described our main analyses that determine success or failure of this replication attempt. Based on these primary analyses, we conducted a design analysis based on simulations to find a sampling plan that would allow to find conclusive evidence for these measures.

In order to determine an appropriate sample size that allows to find an effect with high probability, we are required to specify a realistic effect size estimate. It is a well-known fact that published effect sizes that are based on small sample sizes and the criterion of statistical significance are inflated because of the “significance filter” (Gelman & Carlin, 2014): For an effect to become significant at low sample-sizes the effect must be large. We therefore thought it likely that the very strong effect of $d = 1.59$ reported by Axelrod et al. (2015) was an overestimate and that the real effect-size would be much lower. We note here, that the effect size reported in Axelrod et al. (2015) used a non-standard estimate of the pooled variance that accounts for differences in means and therefore results in the lower (though still huge) estimate of $d = 1.24$ that was reported in their study. In the field of tDCS, observed effect sizes are usually of small or medium size. The absolute value of effect-sizes for cognitive effects of DLPFC stimulation reported by Horvath et al. (2015b) were on average 0.4 (SD=0.59; median=0.29, meta-analytic mean=0.31, SD=0.41) and a recent preregistered tDCS study (which does not suffer from the significance filter) found an effect-size of $d = 0.45$ (Minarik et al., 2016).

We therefore designed our study to be able to detect effects in this range with appropriate probability and report a design analysis for a wide range of effect sizes. It has recently been proposed that underpowered studies are unfalsifiable (Morey & Lakens, 2016). These authors convincingly argue that even large discrepancies between an original, underpowered study and a (direct) replication study cannot be detected with high probability even if the replication study has infinite sample size. Accordingly, we choose to

base our power calculations not on the goal to replicate (or not-replicate) the original study but rather focus on estimating the real effect and of excluding the possibility of a zero effect while also analysing the expected distributions of the BFs.

Following (Kruschke, 2014), we ran a Bayesian power analysis where our primary goal was to exclude the null-hypothesis of an effect-size of $d = 0$ from the posterior 95% highest-density interval in the positive direction. Practical reasons did not allow us to exceed a sample size of $N=192$, such that each lab committed to collecting a maximum of $N=64$ subjects (32 per condition). In addition, we did not want to collect more data than necessary for ethical reasons. Therefore, we chose to apply a sequential design with a specified maximum sample size of $N=192$ (Schönbrodt & Wagenmakers, 2018; Schönbrodt et al., 2017). In order to avoid spurious rejections of the existence of an effect, we chose to first collect a minimum sample size of $N=120$ (20 per lab and condition). If the 95% posterior highest density interval (HDI) did not exclude zero at this point, we continued sampling until a maximum of $N=192$ had been reached. Once the initial 120 subjects were collected, we stopped after each batch of 18 subjects (3 per lab and condition) and evaluated whether the lower bound of the 95% HDI was larger than zero. If that would have been the case, we would have stopped data-collection; otherwise we would continue until the designated maximum (this was the case in our study, see Results). Note, that this was a directional stopping rule: We would only stop collecting data in case the HDI was fully positive. If it would have been fully negative, we would have continued sampling up to the full sample-size. The reason for this asymmetry was that a negative effect would have been surprising (given that we expected a positive effect) and we would have wanted to collect as much evidence for that as possible. The final posterior HDI was not biased in either direction, though.

In Figure 2, we provide a simulation-based analysis of this design. The simulation underlying this analysis proceeded as follows:

1. Pick an effect-size estimate d (we ran this simulation for effect sizes ranging between 0 and 1 in steps of 0.05)
2. For each d , run $n_{\text{rep}} = 10000$ simulations as follows:
 - generate a random dataset with an effect-size of d
 - following the sampling plan described above, calculate

- 560 a) the posterior HDI from the (undirected) Bayesian t-test de-
 561 scribed by Rouder et al. (2009) and implemented in Morey &
 562 Rouder (2015)
- 563 b) the Bayes Factors discussed above, BF_{directed} , $BF_{\text{null+}}$ and
 564 $BF_{\text{null-}}$

565
 566 and return the first N for which the lower bound of the HDI is
 567 above zero (or N_{max} if this did not happen), the associated BFs,
 568 the associated width of the HDI and whether or not the HDI
 569 excluded zero

570 3. Summarize/visualize the results for each effect-size estimate

571 The code for running this analysis and to produce Figure 2 is available
 572 at [power_sequential_hdi.R](#).

573 Given this sampling plan, the probability of obtaining a false-positive,
 574 concluding that the HDI excludes zero even if $d = 0$, is 4.02%. The probabil-
 575 ity to find a conclusive HDI that excludes zero (power) is a function of the
 576 underlying real effect size (Fig. 2 a). For realistic estimates of the effect-size
 577 around $d = 0.4$, we have a power between 0.8 ($d = 0.39$) and 0.9 ($d = 0.46$).
 578 We could also determine the expected size of our sample (Fig. 2 b): With
 579 a real effect-size of 0.4, we had a probability to stop after the initial sample
 580 of $N=60$ per group of 0.54 and the probability to go to the maximum was
 581 0.18. This illustrates the efficiency of this sampling plan as we had a good
 582 chance of being able to stop data-collection at an earlier stage. Figure 2 c)
 583 and d) show the distribution of the expected BF_{directed} , $BF_{\text{null+}}$, $BF_{\text{null-}}$ and
 584 the expected width of the posterior HDI. At $d = 0.4$, the expected directional
 585 BF is around 86 and the expected width of the HDI around 0.7 (see Table
 586 1). In case of a zero underlying effect size, the design is less efficient: the
 587 BFs in favor of the null-hypothesis were only expected to be of moderate size
 588 (around 6).

589 The analyses described so far used a Cauchy-distribution with scale pa-
 590 rameter $r = \sqrt{2}/2$ as the prior distribution on the effect-size. The expected
 591 results for both the HDI and the BFs are not sensitive to the choice of this
 592 prior parameter. We re-ran the simulation described above for two other
 593 common choices of the scale-parameter, $r = 1$ and $r = \sqrt{2}$ and the effect
 594 on the outcome variables was minimal. This is due to the rather large sam-
 595 ple even with the lowest possible sample size allowed by our sampling plan
 596 because the likelihood eventually overwhelms any reasonable choice of prior.

Figure 2: Design analysis for a sequential design with a maximum N of 192, an initial N of 120 and optional stopping after batches of 18 subjects in case the 95% HDI excluded zero. (a) Probability that the HDI excludes zero as a function of the real underlying effect-size. Dashed lines show the effect-size for which our sampling plan has 80% and 90% power, respectively. (b) Probability to collect samples of different sizes as a function of real effect-size. In case of a low real effect size, collection of the full sample of $N=96$ per group is highly likely while only the minimal $N=60$ per group will likely be collected if the effect size is large. (c) Distribution of BFs (both BF_{directed} and BF_{null}) we are likely to find given the underlying effect size. Horizontal dashed line indicates $BF=6$. (d) The expected width of the posterior HDI given the underlying effect-size. Because needed sample size decreases with increasing effect-size, the width of the HDI increases as well. Colored and grey ribbons show 80% and 95% HDI for the respective parameter.

Table 1: Summary of the sampling plan in case of two hypothetical scenarios: The null-hypothesis is true ($d = 0$, left) and the real effect has an effect-size of $d = 0.4$ (right). If the null-hypothesis is correct, the directional BF, $\text{BF}_{\text{directed}}$, will be inconclusive as there is about the same amount of evidence for the effect being negative or positive, while both $\text{BF}_{\text{null}+}$ and $\text{BF}_{\text{null}-}$ are likely to be of moderate size. In the case of a small-to-medium effect size of $d = 0.4$, the $\text{BF}_{\text{directed}}$ results in compelling evidence while the $\text{BF}_{\text{null}+}$ is less compelling (median $1/\text{BF}_{\text{null}+}$ only moderately in support of positive effect). The $\text{BF}_{\text{null}-}$ shows compelling evidence for the null and is not easy to interpret when the real underlying effect is positive as it only compares evidence for negative and zero effect-sizes. The expected width of the HDI is about 0.55 in case of $d = 0$ but only 0.69 for the case of $d = 0.4$. This effect exists because sample size is maximal when $d = 0$.

	$d = 0$			$d = 0.4$		
	median	$P(\text{BF} > 6)$	quantiles	median	$P(\text{BF} > 6)$	quantiles
$\text{BF}_{\text{directed}}$	1.02	0.13	[0.06, 21.4]	86.2	0.96	[6.97, 7473.6]
$\text{BF}_{\text{null}+}$	6.3	0.52	[0.78, 16.11]	0.20	0.003	[0.003, 1.88]
$1/\text{BF}_{\text{null}+}$	0.16	0.01	[0.06, 1.28]	4.89	0.44	[0.53, 310.5]
$\text{BF}_{\text{null}-}$	6.45	0.53	[0.93, 16.0]	17.9	0.99	[13.11, 24.1]
$1/\text{BF}_{\text{null}-}$	0.16	0.006	[0.06, 1.07]	0.06	0	[0.04, 0.08]
HDI width	0.55		[0.53, 0.56]	0.69		[0.54, 0.73]
$P(\text{HDI} > 0)$	0.043			0.81		

597 2.5.3. Hierarchical ordered probit model

598 In addition to the aforementioned analysis, we analyzed the data using a
599 novel analysis method that has not been used previously to analyze thought
600 probe data. We used a hierarchical Bayesian model developed for analyzing
601 rank-ordered data. In the previous analyses and in most if not all of the
602 literature, mind-wandering thought-probes are first averaged within-subject
603 before this average is submitted to the final between-subject analysis. This
604 kind of analysis is problematic in at least three ways: First, it constitutes a
605 “waste” of data because information about within-subject variability of re-
606 sponses to thought-probes is lost. Second, treating thought-probe responses
607 as a metric variable is problematic because assumptions underlying the em-
608 ployed methods are likely not to be met. Finally, interesting and known
609 effects on responding are ignored. Most prominently, an effect that is visible
610 in all mind-wandering studies we have seen so far, is the time-on-task effect
611 that is well-known to affect how likely subjects are to respond positively to
612 mind-wandering probes (Thomson et al., 2014).

613 These points can be improved upon by using an appropriate model.

614 The first point, modeling within- and between-subject variability, can be
615 accounted for by a hierarchical modeling approach where subject-level pa-
616 rameters are separately estimated while constraining these estimates by a
617 group-level distribution. The second point (treating ordered variables as
618 metric) can be improved upon by using an ordered probit model. A Bayesian
619 implementation of such a model is described in [Kruschke \(2014\)](#) (Ch. 23).
620 Basically, the assumption of an underlying metric (normal) variable is made
621 which is thresholded by the participant into discrete response bins. In this
622 setting, both the threshold and the parameters of the underlying distribu-
623 tion are estimated separately. Finally, covariates (e.g., time-on-task) can be
624 easily integrated using this method.

625 To justify the need for these advanced analysis methods, we compared
626 models of different complexity on a thought-probe dataset. Because we did
627 not have access to [Axelrod et al. \(2015\)](#)’s original data, we used data from
628 an unpublished study collected in our lab. In this study, we also used the
629 SART paradigm (though using slightly different parameters, such as number
630 of trials and targets). We also employed the same 4-point scale as used in
631 the current study and 20 thought-probes spread out across the experiment
632 were collected from each of 19 participants. A detailed description of this
633 study can be found in [bsc_christian_fossheim.pdf](#). We believe that this
634 data, while not identical to the current study, could give an indication of the
635 magnitude of within-/between-subject variation in responding to thought-
636 probes.

637 In preparation of the analysis, we analyzed these data using a range of
638 models of increasing complexity (code for fitting and diagnosing these models
639 is available at [analysis/ordered_probit](#)). We compare the models based
640 on their predictive performance using leave-one-out cross-validation (LOOC)
641 and Watanabe’s information criterion (WAIC) implemented in the `loo` pack-
642 age ([Vehtari et al., 2015](#)) which are the state-of-the-art model-selection cri-
643 teria for hierarchical Bayesian models ([Gelman et al., 2014](#)). These criteria
644 are reported on the deviance scale and differences of about 10 units are con-
645 sidered strong ([Spiegelhalter et al., 2002](#)). In general, LOOC is the preferred
646 criterion, while WAIC can be a viable and computationally easier approx-
647 imation to LOOC ([Gelman et al., 2014](#)) when calculation of the LOOC is
648 not possible. For all reported models, LOOC and WAIC produced identical
649 results and we therefore only report the former.

650 The first model uses a basic analysis strategy as a baseline, treating MW
651 probes as metric and interchangeable across trials and subjects. Next, we im-

plemented an ordered-probit model where individual responses were treated independently. The comparison of these two models determined whether treating the data as metric was justified. The third and fourth model implement a hierarchical version of the first two models, where subject-level means are constrained by a group-level distribution. Comparing these two models to the first two can help to determine whether the explicit modeling of within- and between-subject variation is necessary. Finally, we added time-on-task as a covariate to the hierarchical ordered probit model. Table 2 lists the LOOC criterion (standard error in parentheses) for each of the models. It is clear that the ordered probit model more appropriately models the data than a model treating the data as metric both in the basic ($\Delta\text{LOOC}=34.1$, $\text{SE}=6.0$) and the hierarchical case ($\Delta\text{LOOC}=31.9$, $\text{SE}=5.9$). Finally, adding the covariate time-on-task strongly improves predictive accuracy, $\Delta\text{LOOC}=12.5$, $\text{SE}=5.0$.

Table 2: Model selection criteria for models of increasing complexity. The hierarchical ordered probit-model including a time-on-task covariate is the most appropriate of the models. weights=posterior probability that each model has the best expected out-of-sample predictive accuracy; LOOC=leave-one-out cross-validation criterion. The model with the lowest LOOC is preferred.

Model	Description	LOOC (SE)	weight
1	metric	1116.8 (17.7)	0.0
2	ordered probit	1048.6 (6.3)	0.0
3	hier. metric	992.8 (22.6)	0.0
4	hier. ordered probit	929.1 (18.3)	0.0
5	hier. ordered probit + time-on-task	904.2 (20.2)	1.0

Based on these considerations, we chose the hierarchical ordered probit model that included a time-on-task covariate as the final analysis model. The model is mathematically fully specified in Appendix 1, including choice of the prior distribution, and implemented in the R-script [hier_ordered_probit.R](#). We report and interpret all coefficients in terms of posterior mean and HDI.

2.5.4. Effect of location (lab)

Despite the uniform study design applied at all locations (UiT, UvA, UniGö), unknown contextual factors might cause substantial variability in effect sizes between the three labs. Therefore, we compared the tDCS effects resulting from the data from all three labs independently by calculating

677 independent estimates per lab for the full hierarchical ordered probit model
678 presented in the previous section. These estimates in terms of posterior mean
679 and HDI are presented side-by-side for comparing the variability in the dif-
680 ferent variables across labs. We also augmented the model with covariates
681 for study location (UiT, UvA, UniGö). Comparing the posterior means for
682 the location-coefficients and their HDI as well as a model-comparison anal-
683 ysis of the augmented vs. the non-augmented model enabled us to rule out
684 or quantify location-specific effects. For details see Appendix 1. The script
685 implementing these analyses is available at [location_effects.R](#).

686 2.5.5. *Frequentist analyses*

687 For comparabililty with the previous literature, we also conducted stan-
688 dard two-sample t-tests on mean thought-probe responses for sham vs. anodal
689 stimulation (both directed and undirected). We also report standardized ef-
690 fect sizes (Cohen’s d) for these effects. These analyses are only conducted
691 because they correspond directly to the analytical strategy chosen by the
692 authors of the original study ([Axelrod et al., 2015](#)). Unfortunately, our se-
693 quential sampling scheme prevents us from calculating these statistics for the
694 final sample as the stopping-rule invalidates the p-values. We therefore use
695 only the guaranteed initial sample size of N=60 per group for this analysis.
696 The script implementing these analyses is available at [frequentist.R](#).

697 2.5.6. *Exploratory analyses*

698 To further assess whether mind wandering or other task-related measures
699 were influenced by tDCS, we conducted five Bayesian repeated-measures
700 analyses of variance (ANOVA) tests along with their frequentist equivalents
701 with time (2 levels: first vs. second parts of the task, associated with online
702 vs. offline effects, respectively) as within-subject and stimulation (2 levels:
703 anodal vs. sham tDCS) as between-subject factors. This analysis design is
704 identical to that used by the original study ([Axelrod et al., 2015](#)), which
705 focused on three measures of interest, each entered as dependent variable
706 in separate ANOVAs: Thought-probe ratings, mean reaction times for Go
707 stimuli (GoRT) and mean error rates for Nogo stimuli (commission errors).
708 We extended this analysis with two additional parameters: Reaction time
709 coefficients of variation (RTCV) and error rates for Go stimuli (omission er-
710 rors). RTCV was quantified as dividing the standard deviation by mean RT
711 scores, calculated for both parts of the task and for each participant sepa-
712 rately. Both RTCV and omission errors were proposed to index lapses of

attention during the SART, and therefore, are regarded as behavioral indices of mind wandering (Cheyne et al., 2009). All analyses within this section were done using JASP 0.9 (JASP Team, 2018). Bayesian tests were run with default prior scales of JASP (r scale fixed effects: 0.5). Interaction terms were assessed by comparing models including the effect to equivalent models without the effect ($BF_{\text{inclusion}}$). Based on the recommendation by Jeffreys (1961), we report results with BF values providing moderate evidence for either the alternative ($BF > 3$) or null-hypothesis ($BF < 0.33$). Depending on the type of variable (continuous vs. ordinal), correlations between behavioral measures were assessed by calculating either Pearson’s or Kendall’s correlation coefficients. To demonstrate effect size for frequentist ANOVAs, we report partial η^2 values. Given the exploratory nature of correlation analyses performed herein, the reported p values are not corrected for multiple comparisons and findings should be treated with caution.

3. Results

3.1. Demographics

Our sample consisted predominantly of females (70%, 134/192) who were young adults (M=22.2 yrs, SD=3.19 yrs, range 18-35 yrs). There were no strong differences in these characteristics between labs, see Table 3. During data acquisition, three subjects in Tromsø had to be excluded due to missing electrode contact after the first half of the experiment (two subjects) and a technical malfunction of the electrode cables (one subject). In Amsterdam, two subjects had to be excluded, one because of an interruption of the experimental session and one that turned out not to fulfill the inclusion criteria after the session. No subjects were excluded in Göttingen.

Table 3: Demographics across the three labs.

Lab	Proportion male	Mean/SD Age	Min/Max Age
AMS	10/64	20.66 (2.35)	[18, 31]
GOE	28/64	23.30 (2.66)	[18, 34]
TRM	20/64	22.75 (3.77)	[19, 35]
all	58/192	22.2 (3.19)	[18, 35]

738 3.2. Pre-Registered Analyses

739 In agreement with our sequential-sampling plan, we tested several times
 740 during data acquisition whether our stopping criterion was fulfilled. This
 741 criterion was that the 95% HDI of the posterior effect-size estimate would
 742 exclude zero in the positive direction. This did not turn out to be the case
 743 and therefore the maximum sample size was collected resulting in $N = 64$
 744 subjects per lab and a total of 192 participants. In summary, the mean
 745 posterior effect size was consistently estimated to be slightly negative and
 746 the HDIs all included zero, see Table 4 and Figure 3.

747 3.2.1. Effect of anodal stimulation on self-reported mind wandering

748 With our final sample size, the effect-size estimated according to our
 749 pre-registered analysis plan was $d = -0.11$, $\text{HDI} = [-0.38, 0.17]$. Negative
 750 effect-sizes indicate that subjects in the anodal stimulation condition were
 751 less likely to respond off-task on the thought-probes than subjects in the sham
 752 stimulation condition. Accordingly, the directional Bayes Factor, $\text{BF}_{\text{directed}}$,
 753 which compared the hypotheses that the effect was positive to the hypothesis
 754 that it was zero or negative was in support of negative effect-sizes ($\text{BF}_{\text{directed}} =$
 755 0.29) but only slightly so. According to this test, it is about 3.4 times as
 756 likely that the effect-size was zero or negative when compared to a strictly
 757 positive effect. We also pre-specified several BFs that would test the null-
 758 hypothesis of a zero effect against several alternatives (against a positive,
 759 $\text{BF}_{\text{null}+}$, a negative, $\text{BF}_{\text{null}-}$, or any effect, BF_{null} , respectively). All of these
 760 Bayes Factors were in support of the null-hypothesis with varying degrees of
 761 strength. When comparing the null-hypothesis to the a-priori hypothesized
 762 positive effect, the null-hypothesis was about 10.65 times more likely to be
 763 true, $\text{BF}_{\text{null}+} = 10.65$. When comparing the null-hypothesis to any non-zero
 764 effect-size, the null-hypothesis was less strongly supported, $\text{BF}_{\text{null}} = 4.79$ and
 765 even when comparing the null against a negative effect-size (that was unlikely
 766 a-priori but seems more plausible given the observed negative effect-size), the
 767 null was slightly favored, $\text{BF}_{\text{null}-} = 3.09$.

768 Finally, we also calculated the replication Bayes Factors, $\text{BF}_{\text{replication}}$, and
 769 the meta-analytic BF, BF_{meta} (Verhagen & Wagenmakers, 2014). The repli-
 770 cation BF tests the hypothesis that the observed data from our replica-
 771 tion study is consistent with the originally reported effect-size against the
 772 alternative that it is not. We found strong support for the alternative
 773 ($\text{BF}_{\text{replication}} = 0.002$) indicating that it is about 500 times as likely that the
 774 effect was not consistent with the originally reported effect-size, i.e., that the

Figure 3: Results of the sequential sampling plan. Target statistics for increasing sample size (per lab) are plotted. Dots represent the pre-registered timepoints at which data-collection could have been stopped should the HDI have excluded zero in the positive direction. (a) Scatter-plot of individual subjects' mean thought-probe responses together with a density estimate and mean and confidence interval (red). (b) Effect-size and 95% HDI for the effect of anodal stimulation on mean thought probes. All HDI's included zero at all times. The final mean effect-size was in the opposite direction than hypothesized. (c) Bayes-factors quantifying evidence in support of various hypotheses (see text for details).

Table 4: Results at the pre-registered stopping points. The criterion for stopping the data-collection was that the 95% HDI around the effect-size would exclude zero in the positive direction. The effect-size was consistently negative and all HDIs included zero and therefore the complete sample was collected.

N	Cohen's d	$BF_{\text{null}+}$	$BF_{\text{null}-}$	BF_{null}	BF_{directed}	$BF_{\text{replication}}$	BF_{meta}
120	-0.09 [-0.44, 0.24]	7.46	3.21	4.48	0.43	0.002	0.34
138	-0.06 [-0.38, 0.25]	7.27	3.91	5.08	0.54	0.003	0.28
156	-0.05 [-0.35, 0.25]	7.30	4.44	5.52	0.61	0.003	0.25
174	-0.07 [-0.36, 0.22]	8.65	3.93	5.41	0.45	0.003	0.32
192	-0.11 [-0.38, 0.17]	10.65	3.09	4.79	0.29	0.002	0.48

effect did not replicate. The meta-analytic BF was calculated to judge overall support for the presence of any effect of anodal stimulation on thought-probes when pooling both the original and the replication study. Also this BF supported the null but only weakly so ($BF_{\text{meta}}=0.48$) which was expected given that the original study reported a huge, and most likely overestimated, effect-size ($d_{\text{original}} = 1.24$) which would bias the result of the meta-analytic BF in favor of a positive effect.

3.2.2. Hierarchical ordered probit model

The pre-registered hierarchical ordered probit model was fit to the final dataset. The posterior mean and HDIs are reported in Table 5. We ran 12 parallel chains for 2000 iterations each, treating the first 1000 samples as warmup resulting in final of 12000 independent samples from the posterior distribution. We used that many samples in order to properly estimate the tails of the distribution which were needed for accurately reporting the 95% HDI. The Gelman-Rubin diagnostic (Gelman & Rubin, 1992) was calculated to ensure that all reported results had an $\hat{R} \leq 1.05$. We also visually inspected the traceplots for all variables and no anomalies were spotted.

In order to show the appropriateness of the model, we conducted posterior predictive checks (Gelman et al., 1996). We generated $n_{\text{rep}} = 100$ complete datasets by drawing coefficients randomly from the posterior distribution and simulating datasets according to the model specification. The distribution of summary statistics from these posterior simulations can be compared to the actually observed data to evaluate model fit. Figure 4 shows the result of these checks. Model fit is excellent on the group-level but not all individual differences are picked up by this model.

Table 5: Results of fitting the hierarchical ordered probit model. As expected, there is a positive effect of trial number (time on task). However, contrary to our hypothesis, the coefficient coding for the effect of anodal stimulation is negative (with the HDI including zero).

Variable	Coefficient (Mean and 95% HDI)
Intercept (μ_g)	2.25 [2.14, 2.35]
trial (β_1)	0.20 [0.18, 0.23]
stimulation (β_{anodal})	-0.09 [-0.24, 0.07]
threshold (θ_2)	2.53 [2.51, 2.56]
probe-level variance (σ)	0.78 [0.76, 0.80]
group-level variance (σ_g)	0.62 [0.57, 0.68]

Figure 4: Posterior predictive distribution of average responses to thought-probes (left) and for four randomly selected subjects (right). Grey bars represent data, black dots and error bars represent mean and 95% HDI for simulated data.

800 The results of this analysis show a clear positive effect of time-on-task
801 as previously reported, $\beta_1 = 0.20 [0.18, 0.23]$, indicating that subjects were
802 more likely to report being off-task later in the experiment (about 0.67
803 units on the 4-point Likert-scale comparing the end to the beginning of
804 the experiment). The results also show that anodal stimulation did not
805 appear to increase the likelihood to answer off-task on the thought-probes,
806 $\beta_{\text{anodal}} = -0.09 [-0.24, 0.07]$. While the mean coefficient estimate is neg-
807 ative, its 95% HDI includes zero and therefore does not provide evidence
808 against the null-hypothesis.

Figure 5: Coefficient estimates independently for each lab and from a combined model. Colored lines are estimates from individual lab data and the black line and grey area corresponds to posterior mean and 95% HDI from the combined model.

3.2.3. Effect of location (*lab*)

In order to test whether the lab in which each of the three subsets of data were collected would have an impact on the estimation of the effects, we pre-registered to fit the model from the previous section separately to the data from the three locations. In addition, we estimated a pre-registered extended model where lab was entered as a covariate (see Appendix for details). The same model-fitting and -checking procedure as detailed above was used to ensure that the model-fits were reliable.

Results for these analyses are presented in Figure 5. The estimates of the relevant coefficients are in good agreement between labs: Coefficients are estimated to be of a similar magnitude and the HDIs of the separately estimated coefficients overlap in almost all cases. The combined model, treating lab as a fixed-effect covariate seems to provide a good compromise between the independent estimates. The only exception is the coefficient for the time-on-task effect, β_1 . The HDIs estimated for the Amsterdam sample $\beta_1 = 0.13$ [0.088, 0.18] does not overlap with those from the Tromsø $\beta_1 = 0.26$ [0.22, 0.31] or the Göttingen $\beta_1 = 0.22$, [0.18, 0.27] samples. This finding indicates that participants in the AMS lab showed a lesser time-on-task effect on thought-probes than those in GOE or TRM.

We hesitate to provide an interpretation of this finding as it is quite possibly a spurious result: Analyzing the result from Figure 5 involves 18 comparisons. Therefore using 95% HDIs and decision by non-overlap of these intervals, we would already expect to see 1 or 2 positive results due to chance

832 alone (given that the models were fit on independent datasets).

833 We also pre-registered a model-comparison between the ordinal probit-
834 regression model with and without the lab-covariate based on the LOOIC and
835 the WAIC. This analysis can provide evidence for or against the suitability
836 of including lab as a covariate in the model, i.e., whether a considerable
837 amount of the variation in the data is being explained by this factor or not.
838 The model that does not have any information about which lab the data
839 was collected in resulted in a LOOIC of 10093.2 (SE= 83.1) and a WAIC
840 of 10091.8 (SE= 83.0) while the extended model had a LOOIC of 10092.7
841 (SE= 83.1) and a WAIC of 10091.6 (SE= 83.0). These are virtually identical
842 ($\Delta\text{LOOIC} = -0.3$, SE= 0.8; $\Delta\text{WAIC} = -0.1$, SE= 0.8) and therefore these
843 criteria do not prefer any of the two models.

844 Even though the extended model did not provide a better model fit,
845 we can check the regression coefficients corresponding to the different labs.
846 Analyzing the extended model further, these coefficients were estimated as
847 $\beta_{\text{AMS}} = -0.17, [-0.35, 0.02]$ and $\beta_{\text{GOE}} = -0.29, [-0.47, -0.10]$. According
848 to this model, participants at the university of Göttingen were therefore less
849 likely to respond to be off-task when compared to participants in Tromsø. As
850 before when investigating the data from the labs separately, participants from
851 Amsterdam were slightly less likely to respond with off-task than participants
852 from Tromsø but slightly more likely to response off-task than subjects from
853 Göttingen (though these HDIs did overlap).

854 We did not expect a priori to find any differences between the estimates
855 from the three different labs. Since there were some indications of possi-
856 ble differences in the data, we chose to run several exploratory analyses to
857 investigate possible reasons for this finding (see section 3.3.2).

858 3.2.4. *Frequentist analyses*

859 In accordance with our pre-registered analysis plan, we performed inde-
860 pendent t-tests on individually calculated mean thought-probe scores. Note
861 that only the initial sample of $N = 120$ is used in these tests as the stop-
862 ping rule would invalidate p-values calculated for the complete sample since
863 these would have to be corrected for the intermediate looks at the data. The
864 two-tailed t-test exploring whether anodal tDCS resulted in altered (i.e.,
865 either increased or decreased) mind-wandering propensity relative to sham
866 stimulation was not significant ($t(117.68) = -1.01$, $p = 0.312$, Cohen's $d =$
867 -0.102). Also, the one-tailed t-test assessing directional effects indicated that
868 anodal tDCS was not associated with increased propensity of mind wandering

869 ($t(117.68) = -1.01$, $p = 0.843$).

870 3.3. Exploratory Analyses

871 3.3.1. Influence of brain stimulation on other task measures

Table 6: Summary statistics of different outcome variables split by stimulation and online (part 1) and offline (part 2). Mean $\pm$ standard deviations are reported.

	1 st part	1 st part	2 nd part	2 nd part
	Anodal	Sham	Anodal	Sham
Thought-probes	2.08 $\pm$ 0.56	2.15 $\pm$ 0.49	2.30 $\pm$ 0.62	2.36 $\pm$ 0.63
RT (ms)	393.4 $\pm$ 71.6	381.5 $\pm$ 61.8	380.6 $\pm$ 87.2	368.5 $\pm$ 55.6
RTCV	0.29 $\pm$ 0.13	0.28 $\pm$ 0.08	0.30 $\pm$ 0.12	0.29 $\pm$ 0.11
Commission errors (%)	35.7 $\pm$ 19.8	38.4 $\pm$ 18.8	43.1 $\pm$ 23.6	42.9 $\pm$ 20.6

872 In accordance with the well-known time-on-task effect on mind wander-
873 ing (i.e., more attentional lapses in later parts of the task) that we already
874 reported in our pre-registered analyses, we found compelling evidence for
875 the effect of time ($BF_{10} = 7.03 \times 10^8$; $F(1,190) = 52.421$; $p < 0.001$; $\eta^2 =$
876 0.216), although this effect was numerically rather small (first part: $M =$
877 2.12 ; $SD = 0.52$; second part: $M = 2.33$; $SD = 0.62$). In addition, partic-
878 ipants became faster ($BF_{10} = 106.46$; GoRT: $F(1,190) = 14.714$; $p < 0.001$;
879 $\eta^2 = 0.072$) and made more key presses on Nogo trials (commission errors:
880 $BF_{10} = 1958.5$; $F(1,190) = 21.409$; $p < 0.001$; $\eta^2 = 0.101$) in the second part
881 of the experiment. This finding indicates a change in the speed-accuracy
882 tradeoff with task progress (Pearson’s correlation between GoRT and com-
883 mission errors for the whole task: $BF_{10} = 4.07$; $r(190) = -0.199$; $p = 0.006$),
884 and might be related to more mind wandering during the second part of the
885 task (Kendall’s correlation between thought-probe ratings and GoRT for the
886 whole task: $BF_{10} = 3.55$; $\tau(190) = 0.131$; $p = 0.008$; between thought-probe
887 ratings and commission errors: $BF_{10} = 554.09$; $\tau(190) = 0.203$; $p < 0.001$).
888 Finally, response times were more variable in the second part of the SART
889 (RTCV: $BF_{10} = 5.83$; $F(1,190) = 8.352$; $p = 0.004$; $\eta^2 = 0.042$), an effect
890 that can also be attributed to increasing mind wandering propensity with
891 time spent on the task (Kendall’s correlation between thought-probe ratings
892 and RTCV: $BF_{10} = 3639.73$; $\tau(190) = 0.224$; $p < 0.001$; Pearson’s correla-
893 tion between GoRT and RTCV: $BF_{10} = 1411.99$; $r(190) = 0.312$; $p < 0.001$;
894 between commission errors and RTCV: $BF_{10} = 1.08 \times 10^8$; $r(190) = 0.446$; p

895 < 0.001). Although omission errors on Go trials were not affected by time-
896 on-task ($BF_{10} = 0.11$), they correlated positively both with mind wandering
897 ($BF_{10} = 10.99$; $\tau(190) = 0.150$; $p = 0.004$) and with other task measures
898 (GoRT: $BF_{10} = 101.1$; $r(190) = 0.268$; $p < 0.001$; RTCV: $BF_{10} = 5.42 \times 10^{27}$;
899 $r(190) = 0.711$; $p < 0.001$).

900 With respect to the effect of tDCS on mind wandering or task perfor-
901 mance, neither the main effect of stimulation (BF_{10} between 0.23 and 0.53;
902 $F < 1.59$, $p > 0.208$), nor its interaction with time ($BF_{inclusion}$ between 0.15
903 and 0.28; $F < 1.241$, $p > 0.265$) were significant for either of the five measures
904 of interest.

905 3.3.2. Exploratory analysis of location effects

906 In order to further investigate the effects of lab in which each of the
907 three datasets was collected on thought-probe responses reported earlier,
908 we extended the hierarchical probit regression model described in Appendix
909 1 by introducing interaction effects for lab $\times$ stimulation and lab $\times$ trial
910 treating Tromsø as the baseline. The resulting model produced a better
911 fit in terms of model-selection criteria (LOOIC= 10077.2, SE= 83.4) than
912 the model with only lab as a main effect ($\Delta LOOIC = 7.3$, SE= 4.3). Us-
913 ing this model, the HDIs for the main effect of lab no longer exclude zero,
914 $\beta_{AMS} = -0.19, [-0.45, 0.07]$, $\beta_{GOE} = -0.24, [-0.50, 0.02]$ even though they
915 are still indicating reduced off-task reports in both Amsterdam and Göttingen
916 when compared to Tromsø. There is no evidence that the brain stimu-
917 lation affected the thought-probe reports differentially in the three labs,
918 $\beta_{GOE \times stimulation} = -0.09, [-0.45, 0.27]$, $\beta_{AMS \times stimulation} = -0.06, [-0.29, 0.42]$.
919 Finally, the time-on-task effect seems to be reduced in subjects from Ams-
920 terdam as compared to Tromsø, $\beta_{AMS \times trial} = -0.13, [-0.18, -0.08]$ but not
921 in Göttingen, $\beta_{GOE \times trial} = -0.04, [-0.09, 0.01]$. This finding agrees with the
922 results from the pre-registered analysis which found that the time-on-task
923 effect was reduced in Amsterdam in independent analyses for each lab.

924 Furthermore, we were interested in whether the apparent effect of lab
925 might not actually be due to a gender effect. Previous research has reported
926 gender differences in mind-wandering propensity (Bertossi et al., 2017) and
927 given that we sampled a slightly higher proportion of females in Amsterdam
928 than in the other labs (see Table 3), the observed lab-effect might actually be
929 due to differences in mind-wandering in males and females. We investigated
930 this possibility by augmenting the probit-regression model that includes lab
931 as covariate with an additional covariate coding for the gender of the partic-

932 ipant. Assuming that any differences between the labs were due to gender
933 effects, we would therefore expect the lab-coefficients to be estimated near
934 zero and the coefficient coding for gender to show an effect. This augmenta-
935 tion of the model did not improve the model-fit (LOOIC= 10091.8, SE= 83.1;
936 $\Delta\text{LOOIC} = -0.4$, SE= 0.2). The coefficients for the lab-variables were simi-
937 lar to the ones estimated from the model not including gender as a covariate,
938 $\beta_{\text{AMS}} = -0.16, [-0.35, 0.01]$ and $\beta_{\text{GOE}} = -0.27, [-0.45, -0.08]$ and the coef-
939 ficient for gender was spread wide around zero, $\beta_{\text{male}} = -0.06, [-0.22, 0.11]$
940 indicating that gender was not likely to be responsible for the aforementioned
941 lab effect.

942 3.3.3. Questionnaires

943 When analyzing changes in self-reported mood states during the task,
944 both Bayesian and frequentist repeated-measures ANOVA revealed a main
945 effect of time for positive, but not negative mood scores (PANAS-positive:
946 $\text{BF}_{10} = 8.37 \times 10^{14}$; $F(1,190) = 92.480$; $p < 0.001$; $\eta^2 = 0.327$; PANAS-
947 negative: $\text{BF}_{10} = 0.32$; $F(1,190) = 2.236$; $p = 0.136$; $\eta^2 = 0.012$), indicating a
948 significant reduction in positive mood by the end of the task (pre-task rating:
949 $M = 29.35$; $SD = 6.26$; post-task rating: $M = 25.09$; $SD = 7.22$). Neither
950 the main effect of stimulation nor its interaction with time was significant
951 for the PANAS scores. Furthermore, since mind wandering has been associ-
952 ated with negative mood states (Killingsworth & Gilbert, 2010; Smallwood
953 et al., 2009), we hypothesized a correlation between mind-wandering propen-
954 sity (subjective thought-probe reports) and changes in mood scores measured
955 by the PANAS. Despite our expectations, thought-probe responses did not
956 correlate with pre- vs. post-SART difference scores for PANAS-negative (an-
957 odal tDCS group: $\text{BF}_{10} = 0.36$; $\tau(94) = 0.099$; $p = 0.179$; sham tDCS group:
958 $\text{BF}_{10} = 0.13$; $\tau(94) = 0.009$; $p = 0.908$) or PANAS-positive items (anodal
959 tDCS group: $\text{BF}_{10} = 0.36$; $\tau(94) = 0.98$; $p = 0.052$; sham tDCS group:
960 $\text{BF}_{10} = 0.15$; $\tau(94) = 0.035$; $p = 0.622$).

961 Using the MAAS questionnaire, we have also collected self-reported scores
962 on the individual’s inherent ability to attend to the present experience and
963 remain undistracted. Higher MAAS scores indicate higher level of concen-
964 tration, and therefore, we anticipated that MAAS scores would negatively
965 correlate with thought-probe scores. However, in contrast to our hypothesis,
966 neither group showed a relationship between MAAS scores and mind wan-
967 dering, albeit the correlations were in the expected direction (anodal tDCS
968 group: $\text{BF}_{10} = 0.36$; $\tau(94) = -0.098$; $p = 0.166$; sham tDCS group: $\text{BF}_{10} =$

969 0.29; $\tau(94) = -0.088$; $p = 0.214$).

970 4. Discussion

971 The aim of the study was to replicate the findings reported by [Ax-](#)
972 [elrod et al. \(2015\)](#) about the potential effect of anodal tDCS on mind-
973 wandering propensity. Mind-wandering propensity was assessed by self-
974 reports (thought-probes) while participants were engaged in a sustained at-
975 tention task. Building upon the findings of the original publication, we tested
976 the hypothesis that anodal tDCS over the left DLPFC would increase mind-
977 wandering propensity relative to an inactive (sham) stimulation. The present
978 replication study was performed as a fully pre-registered, multi-center study
979 utilizing a sequential sampling plan with equal sample size across laborato-
980 ries.

981 Contrary to our hypothesis and the findings from [Axelrod et al. \(2015\)](#),
982 we found that the participants receiving anodal stimulation were numerically
983 less likely to respond being off-task when compared to the group receiving
984 sham stimulation over the left DLPFC. Overall, however, our findings show
985 support in favor of a null-effect of stimulation on self-reported thought-probe
986 scores as shown by an analysis based on Bayes Factors. When comparing
987 a null-effect to an effect in the positive direction as hypothesized *a priori*,
988 there was strong evidence for a null effect ($BF_{\text{null}+} = 10.65$). Also, when
989 testing the hypothesis of the effect being zero against the full range of pos-
990 sible non-zero effects, there was moderate evidence for a null effect ($BF_{\text{null}} =$
991 4.79) and even when comparing against a purely negative effect, the null
992 was somewhat favored ($BF_{\text{null}-} = 3.09$). In addition, there was extreme evi-
993 dence ($BF_{\text{replication}} = 0.002$) that the original study was not replicated using
994 a special Bayes Factor designed to indicate replication success ([Verhagen &](#)
995 [Wagenmakers, 2014](#)). When pooling data from both the original and repli-
996 cation study there was strong evidence ($BF_{\text{meta}} = 0.059$) for the absence of
997 an effect of anodal stimulation. We conclude from these results that there is
998 no support for the supposition that bipolar anodal tDCS in the form used in
999 our and the original study ([Axelrod et al., 2015](#)) can influence the propensity
1000 to mind-wander. On the contrary, we found substantive evidence *against* the
1001 existence of such an effect.

1002 Our failure to replicate the original study is perhaps not particular sur-
1003 prising when viewed in the context of previous replication failures in the field
1004 of psychology (e.g.; [Open Science Collaboration et al., 2015](#); [Klein et al.,](#)

2014; Wagenmakers et al., 2016) in general and brain stimulation in particular (Learmonth et al., 2017; Horvath et al., 2016; Vannorsdall et al., 2016). Typically, a result obtained in an initial, often low-powered study fails to be reproduced in large-sample replication attempts (Boekel et al., 2015). Replications are the cornerstone of empirical research and crucial for scientific progress. Even though this is a well-known fact, replication attempts are still rare (Makel et al., 2012). Several reasons for this problematic state of affairs have been pointed out by many authors (Simmons et al., 2011; Chambers, 2017) which comprise factors on many different levels. We conclude that the original result by Axelrod et al. (2015) was most likely a false positive finding caused by strong variability and low sample size. We believe that it is crucial that future studies aiming to establish a specific experimental effect should be required to (a) employ sample sizes that are adequate to find effects of a reasonable magnitude, and (b) to either pre-register their study from the outset or provide a pre-registered replication of their own result. Such requirements would go a long way to protect the literature from the omnipresent false-positives, even though replication by independent, if possible multiple, labs is the ultimate goal (Simons, 2014).

It is important to point out, however, that our failed replication of the study by Axelrod et al. (2015) does not imply that tDCS is an ineffective tool for modulating mind wandering propensity. On the contrary, we are aware of four other studies that reported evidence for active stimulation either increasing or reducing the mind-wandering propensity during various tasks. In three studies, Kajimura and colleagues showed that anodal stimulation of the right inferior parietal lobule (rIPL) reduces mind wandering propensity (Kajimura & Nomura, 2015; Kajimura et al., 2016, 2018). In their first two reports (Kajimura & Nomura, 2015; Kajimura et al., 2016), the cathode was placed above the left DLPFC, rendering the contribution of left DLPFC vs. rIPL to the observed effect impossible to distinguish. However, in their most recent study, the authors used an extracephalic return electrode, providing evidence for rIPL stimulation being primarily responsible for the mind wandering-reducing effect (Kajimura et al., 2018). Interestingly, analysis of effective connectivity patterns revealed that the behavioral effect of anodal tDCS on decreased mind wandering propensity was mediated by weaker afferent connections from the medial prefrontal cortex (MPFC) to the posterior cingulate cortex, highlighting the MPFC node within the DMN as a key mediator for inducing and/or maintaining task-unrelated thoughts (Kajimura et al., 2016). The role of the MPFC in influencing mind wandering is also

supported by another study showing that cathodal tDCS targeting the left MPFC reduces attentional lapses during a choice reaction time task in males (Bertossi et al., 2017). Given the negative results of the current study, however, it is important to replicate any of these positive effects before accepting them as facts.

As detailed in the introduction, several neuroimaging studies and theoretical accounts attribute an important role to the FPN (and, more specifically, to the DLPFC) in regulating mind wandering episodes under various circumstances (Christoff et al., 2009, 2016; Dumontheil et al., 2010; Smallwood et al., 2012). In this regard, the positive finding by Axelrod et al. (2015) fits well in this framework, seemingly providing direct evidence for the causal (rather than correlational) involvement of the left DLPFC to regulating mind wandering propensity. However, the poor spatial focality of bipolar tDCS montages is well-known (Csifcsák et al., 2018; Laakso et al., 2016; Opitz et al., 2015), with stimulation-induced electric fields (EFs) spreading well beyond the area of scalp electrodes, most probably influencing neural excitability in a wide range of cortical areas (Keeser et al., 2011). Using high-resolution realistic head models of healthy adults, we have recently demonstrated that tDCS protocols targeting the left DLPFC show substantial inter-individual variability in the spatial distribution of tDCS-induced EFs (Boayue et al., 2018). Using our previously described and publicly available pipeline (Boayue et al., 2018), we now present new modelling results to gain insight into the potential underlying neural effects that were induced by our tDCS protocol. We focused on the normal component of the EF, that is, on the component perpendicular to the cortical surface, either entering (positive values) or leaving the cortex (negative values). Previous work identified these currents as being excitatory or inhibitory in nature (Rahman et al., 2013), enabling us to assess the direction of the expected effect. In Figure 6 (left panel), we show that despite targeting the left DLPFC, this montage induces EFs in both the medial and lateral aspects of the two hemispheres. Moreover, the right and left MPFC receives excitatory and inhibitory stimulation, respectively, which is particularly interesting as both the enhancement and reduction of MPFC activity by tDCS was associated with changes in mind wandering propensity (Bertossi et al., 2017; Kajimura et al., 2016). Based on these, we argue that stimulation of the MPFC could just as well be responsible for the effect reported by Axelrod et al. (2015) than that of the left DLPFC. In addition, the variability maps shown in Figure 6 (right panel) clearly indicate that the magnitude of EFs in the bilateral DLPFC is highly variable between

Figure 6: Simulation of transcranial direct current stimulation-induced electric fields (EFs) in the cortex of 18 head models for the montage used in our study and by [Axelrod et al. \(2015\)](#). Group-averaged mean values are presented on the left side, whereas the variability of effects across individuals is presented on the right side. For these simulations, we focused on the normal component of the EF, manifesting in positive (anode-like) and negative (cathode-like) values in the mean maps. Across-subject variability was quantified as the EF coefficient of variation ($\frac{\text{standard deviation}}{\text{mean}} \times 100$). Simulation parameters and methods were as described in [Csifcsák et al. \(2018\)](#).

participants.

The tDCS protocol employed in our and the original study even though standard in the field has some drawbacks: First, the protocol used a weak stimulation intensity (1 mA) resulting in electric-field magnitudes of about 0.1-0.2 V/m in the target area (see Figure 6). These estimates are based on computational models that have also been validated by intracranial measurements ([Opitz et al., 2016](#)). It is unclear whether the electric field induced by transcranial electric stimulation is robust and strong enough to cause any physiological effect ([Huang et al., 2017](#)), let alone manifest at the behavioural level. Therefore, it is possible that the stimulation intensity of 1 mA with the present bipolar montage is just not potent enough for the tDCS-induced electric field to have an effect on neural excitability ([Vöröslakos et al., 2018](#)). Second, the bipolar tDCS protocol produces diffuse electric fields resulting in a lack of specificity and the unintended stimulation of other regions ([Csifcsák et al., 2018](#)). The result is a diffuse stimulation of the target region. A better approach might be the use of recently developed high-definition brain stimulation protocols, e.g., 4×1 ring protocols, which allows for more targeted

stimulation (Datta et al., 2009). These protocols allow a much more precise targeting of a region of interest while minimizing the electric field in other parts of the brain. However, this increased focality comes at the price of possibly influencing different regions in different subjects because of substantial differences in brain anatomy (Opitz et al., 2015). It is therefore desirable to use individualized montages based on head models from high resolution magnetic resonance (MR) images to guide optimal electrode placement to result in comparable electric field distributions in individual brains. Taken together, routine usage of this approach could in the future help to increase focality of stimulation and to reduce between-subject variance of the results.

As part of our exploratory analysis, we found that anodal tDCS was not associated with either online or offline effects on task performance. Still, we found robust time-on-task effects regarding thought-probes, accuracy and reaction time measures, which are in line with previous findings (Bastian & Sackur, 2013; Cheyne et al., 2009; McVay & Kane, 2012; Smallwood & Schooler, 2006). Interestingly, although the negative correlation between response times and commission error rates are indicative of a speed-accuracy tradeoff, these parameters were inversely influenced by mind wandering propensity on a between-subject level: Participants reporting more mind wandering were characterized by higher error rates, but also by longer (rather than shorter) reaction times. Response time slowing has been associated with task-unrelated thoughts previously, and it was also found to be predictive of omission errors, as in our study (McVay & Kane, 2012; Smallwood & Schooler, 2006). Nevertheless, these data strengthen views that there is a complex relationship between self-reported mind wandering intensity and performance patterns on the SART (McVay & Kane, 2012), since the latter can be influenced by factors other than mind wandering per se (e.g., impulsivity or response strategy; Helton et al., 2010). Finally, it is worth mentioning that RT variability (RTCV) showed the strongest correlation with thought-probes, highlighting this measure as the most promising objectively quantifiable SART performance index for estimating the prevalence of off-task periods (Bastian & Sackur, 2013).

Rather surprisingly, we did not find a relationship between mind wandering propensity and the participants' mood scores. Despite the often described link between negative mood and task-unrelated thoughts (Killingsworth & Gilbert, 2010; Smallwood et al., 2009), the causal relationship between these phenomena might be too subtle to be detected by our relatively simple questionnaires and thought-probes. Moreover, to avoid inducing mood changes

prior to tDCS, we asked our participants to rate their pre-task mood retrospectively, which most probably restricted the reliability of our mood data. The individual’s predisposition to mindfully attend to the present has been regarded as a personality attribute that is opposed to the propensity to mind wander (Mrazek et al., 2012). However, in our dataset, we did not observe a negative correlation between thought-probe responses and MAAS scores. Interestingly, recent work pointed out that rather than merely being in contrast, these phenomena can interact in a very complex, and at times synergistic way (Seli et al., 2015; Agnoli et al., 2018). For example, it was suggested that the deliberate vs. spontaneous nature of mind wandering is differently related to certain factors of mindfulness (Seli et al., 2015). Thus, the fact that our thought-probes were not enquiring about this aspect of mind wandering might have rendered our analysis insensitive to unveiling the relationship between these phenomena.

We also found indications for differences in mind-wandering propensity between the labs. Even though the results were not very strong (0.2-0.3 units on the 4-point Likert scale) and did not increase the model fit in terms of the model-selection criteria, participants from the university of Amsterdam were generally less likely to respond off-task to the thought-probes than participants from Tromsø. This finding may have several possible explanations. For example, subtle differences in how the thought-probes are being expressed in the three languages (German, Dutch and Norwegian) may have caused participants to give slightly different interpretations to the meaning of the scale. This is a common issue when comparing scales across languages and it is often recommended to disregard any cross-language main effects, assuming that the scales still have metric equivalence but may have a shifted origin (van de Vijver, F. J. R. & Leung, K, 2011). Another possibility are national differences in acceptability of deviations from task-conform behaviour. Recently, researchers have begun to look more closely into boundary conditions of the thought-probe technique (Weinstein et al., 2018; Weinstein, 2017). This finding is a first indication that it may be important to consider language- or nationality-specific effects as well.

In summary, in a high-powered, pre-registered multi-center study, we were not only unable to detect an effect of anodal transcranial direct current stimulation on mind wandering propensity, but we actually found evidence for the absence of such an effect. Our findings further emphasize the significance of direct replications for the further advancement of the field of cognitive neuroscience in general and brain-stimulation in particular.

1174 5. Appendix

1175 5.1. Hierarchical ordered probit model

1176 The model is fully specified as follows: Each response to a thought-probe
 1177 (one of the set $\{1, \dots, K\}$) given by subject j in trial t , is modeled as a
 1178 categorical variable with probability K-simplex p (a K-simplex is a set of K
 1179 positive numbers that sum to one)

$$\text{probe}_{j,t} \sim \text{Categorical}(p).$$

1180 The probabilities for each of the responses are calculated by assuming an
 1181 underlying, continuous, normally-distributed “mind-wandering” variable y
 1182 with parameters $\mu_{j,t}$ and σ that is thresholded into the discrete responses
 1183 at thresholds $\theta_1, \dots, \theta_{K-1}$. The probabilities to give each of the responses
 1184 is the area under the normal curve of y that falls into the K response-bins
 1185 $[-\infty, \theta_1], \dots, [\theta_{K-1}, \infty]$. Therefore, the probabilities are calculated as

$$p_k = \Phi\left(\frac{\theta_k - \mu_{j,t}}{\sigma}\right) - \Phi\left(\frac{\theta_{k-1} - \mu_{j,t}}{\sigma}\right)$$

1186 where Φ is the cumulative standard normal distribution (see [Kruschke, 2014](#),
 1187 for a comprehensive presentation of this model).

1188 The underlying distribution is modeled with a hierarchical linear model

$$\mu_{j,t} = \beta_{0,j} + \beta_1 z(t) + \beta_{\text{anodal}} \text{anodal}_j \quad (1)$$

1189 where $z(t)$ is the z-transformed trial number and anodal_j is an indicator
 1190 variable specifying whether a subject was in the control group (0) or in the
 1191 anodal stimulation group (1). The subject-level intercepts are constrained
 1192 by a group-level distribution

$$\beta_{0,j} \sim \text{Normal}(\mu_g, \sigma_g).$$

1193 Priors are set to be vague as recommended in [Kruschke \(2014\)](#):

$$\mu_g \sim \text{Normal}\left(\frac{1+K}{2}, K\right),$$

1194

$$\sigma_g \sim \text{Uniform}(K/1000, 10K),$$

1195

$$\sigma \sim \text{Uniform}(K/1000, 10K)$$

1196 and

$$\beta_1 \sim \text{Normal}(0, K).$$

1197 The test of the hypothesis that anodal stimulation can increase mind-
1198 wandering is whether the distribution for the β_{anodal} coefficient will be larger
1199 than zero.

1200 For analyzing the effect of lab where the data for a specific subject was
1201 collected, we run three instances of this model with the datasets from the
1202 three universities and present the resulting posterior distribution side-by-
1203 side. In addition, we augment this model with a covariate for lab, modifying
1204 Eq. 1 to read

$$\mu_{j,t} = \beta_{0,j} + \beta_1 z(t) + \beta_{\text{anodal}} \text{anodal}_j + \beta_{\text{labAMS}} \text{AMS}_j + \beta_{\text{labGOE}} \text{GOE}_j$$

1205 where AMS and GOE are indicator variables coding for whether a subject
1206 was recorded in Amsterdam or Göttingen, respectively (with Tromsø serving
1207 as the baseline). This augmented model will be compared to the model
1208 without these covariates using the LOO and WAIC indicators to evaluate
1209 whether the inclusion of this information would improve the fit of the model.

1210 5.2. Changes to the original protocol

1211 The changes detailed here are part of our OSF protocol and can also be
1212 found under <https://osf.io/37kfj/>.

1213 5.2.1. Changes made after pre-registering with EJN but before any data was 1214 collected

1215 The changes documented here have been made before the first dataset
1216 was collected. It is part of a registration at OSF that has been made on
1217 November, 2nd 2017, <https://osf.io/bv32d/>.

1218 Additional instructions for experimenter.

- 1219 • added three more questions (the last three) to the Q&A sheet with
1220 standardized answers to questions that the data-collectors from the
1221 three labs are using in case there are questions from the participants;
1222 those were added purely for preventive reasons because of experiences
1223 during piloting

1224 *Adapted translated instructions.*

- 1225 • adapted the German instructions to reflect the English template; this
1226 was because of an oversight in which only the English template was
1227 adjusted during preparation of the study while the translations were
1228 forgotten. This oversight was spotted by our German collaborators and
1229 we fixed this before any data-collection

1230 *Expanded instructions to avoid accidental unblinding.*

- 1231 • during the course of the pilots at our partnering institutions, we became
1232 aware of the fact that our previously detailed protocol could result in
1233 accidental unblinding of the experimenter. This is due to the fact that
1234 the impedance measurement on the stimulator reflects the ramp-down
1235 period which is earlier in the sham as compared to the real stimulation
1236 condition. We account for this by requiring the experimenters to cover
1237 the stimulation device after recording the initial impedance measure-
1238 ment and to turn it off without lifting the cover before turning it on
1239 again for the final post-stimulation measurement of impedance. This
1240 is reflected in updated portions of the experimenter instructions.
- 1241 • we added a note to the datasheet where the experimenter should input
1242 the number of times the impedance measurement had to be repeated
1243 to come below the required 10 kOhm

1244 *Screen size.* We became aware of an error in our pre-registration where we
1245 specified that we would be using 12" flatscreen monitors. The actual screen
1246 size in the three labs was 19". This difference in screen sizes had no impact
1247 on the size of the displayed stimuli as those were adjusted to cover 3 degrees
1248 of visual angle independently for each lab.

1249 *5.2.2. Changes made after starting the data collection but before any analysis*
1250 *was conducted*

1251 None.

1252 *5.2.3. Changes made after finished data-collection*

1253 It was necessary to adapt several of the pre-registered analysis scripts.
1254 There were two reasons for these changes:

- 1255 1. There were updates to some of the used analyses packages which re-
1256 quired changes to the code in order to run as intended

1257 2. There were errors in the original analysis-script that were only spotted
1258 when confronted with real data.

1259 At our OSF-repository <https://osf.io/dct2r/>, we store a copy of the
1260 updated analysis files and we also keep the output of the `diff` utility that
1261 stores any changes made to the original scripts in an easily readable format.
1262 These files are called `<scriptname>.diff` where `<scriptname>` is replaced
1263 with each of the changed script files. The original script files can be retrieved
1264 from the pre-registration at <https://osf.io/bv32d/>.

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