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Modulating task outcome value to mitigate real-world procrastination via noninvasive brain stimulation

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eLife Assessment

This **valuable** cross-sectional longitudinal study leverages high-definition transcranial direct current stimulation to the left dorsolateral prefrontal cortex to examine its effect on procrastination behavior over an extended time span. The cross-sectional longitudinal study a testing of competing models provided **solid** evidence for how stimulating DLPFC impacts reveal-world procrastination behavior. Whether these results generalize to a larger population will be a significant future direction. This work will be of interest to those interested in cortical function, procrastination, and related states.

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Abstract

Procrastination represents one of the most prevalent behavioral problems associated with individual health and societal productivity. Despite its high prevalence and substantial impact on daily functioning, its underlying neurocognitive mechanisms remain poorly understood. A leading model posits that procrastination arises from imbalanced competing motivations: the avoidance of negative task aversiveness and the pursuit of positive task outcomes, yet this theoretical framework has not been fully validated in real-world settings and not applied effectively to guide interventions. Here, we addressed this gap with a double-blind, randomized controlled trial. We applied seven sessions of high-definition transcranial direct current stimulation (HD-tDCS) to the left dorsolateral prefrontal cortex (DLPFC) in chronic procrastinators. Using the intensive experience sampling method (iESM), we assessed the effect of anodal HD-tDCS on real-world procrastination behavior at offline after-effect (2-day interval) and long-term after-effect (6-month follow-up). We found that this neuromodulation produced a lasting reduction in real-world procrastination, with effects sustained at a 6-month follow-up. While the intervention is significantly associated with both decreased task aversiveness and increased perceived task outcome value, a mediation analysis indicated a disassociable mechanism: the increase in task outcome value (but not task aversiveness) showed a statistical pattern consistent with accounting for the observed behavioral improvement. In conclusion, these findings are consistent with the hypothesis that enhancing DLPFC function may reduce procrastination by selectively amplifying the valuation of future rewards, not by simply reducing negative feelings about the task. These results align with established decision-theoretic frameworks and suggest a targeted, theory-informed avenue for future behavioral interventions.

Introduction

Procrastination is increasingly becoming a prevalent behavioral problem around the world, which reflects the irrational voluntary postponement of scheduled tasks albeit being worse off for such delays (Blake, 2019; Steel, 2007). In epidemiological investigations, more than 15% of adults were identified as having chronic procrastination problems, and the situation for students was worse as 70-80% of undergraduates engaged in procrastination (American College Health Association, 2022; Ferrari et al., 2005). Moreover, the behavioral genetic evidence indicates a certain heritability of procrastination in human beings as well (Gustavson et al., 2017; Gustavson et al., 2014, 2015). In addition to its prevalence, the undesirable associations between procrastination behavior and health also warrant caution. There is cumulative evidence to show the close associations between procrastination behavior and work performance, financial status, interpersonal relationships, and subjective well-being (Ferrari, 1994; Pychyl & Sirois, 2016; Steel et al., 2021). Further, as prospective cohort studies indicate, many mental health problems emerge alongside procrastination, particularly sleep problems, depression, and anxiety (Hairston & Shpitalni, 2016; Johansson et al., 2023). Even worse, chronic procrastination has been consistently associated with poor general health conditions, such as immune system disruption, gastrointestinal disturbance, hypertension and cardiovascular disease (Sirois, 2015; Sirois, 2016). Thus, given these critical ramifications, considerable efforts have been devoted to delve into why we procrastinate irrationally.

To probe why we procrastinate irrationally, researchers have built upon theoretical bases of procrastination from different perspectives. For instance, Steel (2007) pioneered a promising temporal motivation theory (TMT) to explicate procrastination as the failure of self-regulation. This theory suggests that individuals would procrastinate as task utility is devalued in the far future (Steel & König, 2006). Based on insights into emotional regulation, the mood repair perspective provides another explanation to elucidate that procrastination is due to the failure of self-regulation to give priority to short-term mood repair caused by doing the task rather than long-term task reward (Sirois & Pychyl, 2013). Recently, the temporal decision model (TDM) of procrastination further provides an integrative framework to explain the procrastination decisions, which highlights that procrastination is contingent on the trade-off between task aversiveness and task-outcome value (Zhang & Feng, 2020; Zhang, Liu, et al., 2019). Task aversiveness reflects how unpleasant individuals perceive tasks to be, with more unpleasant feelings making procrastination more likely (Zhang & Feng, 2020). Task outcome value indicates how much it is worth as we evaluate the benefits it provides us (e.g., keeping body health) once we complete the task before the deadline (e.g., doing scheduled exercise) (Zhang & Feng, 2020). If the task aversiveness is overvalued in this trade-off, the decision to postpone tasks would be made consistently.

Theoretical frameworks posit that this trade-off and subsequent procrastination decisions may be modulated by individual differences in top-down regulatory capacity, with higher capacity typically associated with less procrastination (Blake, 2019; Ramzi & Saed, 2019; Zhao et al., 2021). Thus far, theoretical models have proposed three potential pathways through which regulatory processes might contribute to reduced procrastination: one for decreasing task aversiveness, the other one for increasing task-outcome value, and the last one for both (Zhang & Feng, 2020). As identified by both behavioral and neural evidence, procrastinators consistently report high task aversiveness when receiving scheduled tasks, and are more likely to postpone tasks so as to devalue negative task aversiveness (Blunt & Pychyl, 2000; Zhang et al., 2021). Meanwhile, priori literature suggests that top-down regulatory processes may facilitate the downstream modulation of negative emotional stimuli (Paschke et al., 2016; Tice & Bratslavsky, 2000). Therefore, one hypothesized pathway involves the downstream modulation of task aversiveness (Eckert et al., 2016). On the other hand, as a value-based decision, procrastination behavior is contingent on the evaluation of future outcomes (Rebetez et al., 2016; Zhang, Becker, et al., 2019). Existing evidence has shown that procrastinators generally underestimate the task-outcome value (H. Wu et al., 2016; Zhang et al., 2021). In this vein, it is hard to generate the motivation to take immediate action (Taura et al., 2015). Notably, increasing the value of future

rewards has been found effective in making individuals inclined to pursue future outcomes by potentially engaging prefrontal regulatory networks to prioritize future outcomes (Cho et al., 2015; Kelley et al., 2018). Thus, another pathway worth putting forward is that procrastination behavior could be shaped by increasing future task-outcome value. Also, given the integrative nature of prefrontal regulatory functions, we hypothesize a third pathway whereby both decreased task aversiveness and increased task-outcome value may jointly contribute to reduced procrastination, potentially reflecting coordinated downstream effects on valuation and affective processing.

Consistent with this framework, the left dorsolateral prefrontal cortex (DLPFC)—a region frequently implicated in value-based decision-making and top-down regulation—has been associated with procrastination. Structural and functional variations in the left DLPFC have been correlated with procrastination tendencies (Chen et al., 2020; Hu et al., 2018; Liu & Feng, 2017). In addition to structural brain hallmarks, the neurofunctional anomalies underlying procrastination were found in DLPFC-involved circuits (Y. Wu et al., 2016; Xu et al., 2021). Furthermore, the triple brain network theory provides network-based insights to highlight the neural signature of procrastination as the self-control neural network (e.g., left DLPFC; anterior cingulate cortex, ACC), emotional regulation network (e.g., insula and orbitofrontal cortex, OFC) and episodic prospection network (e.g., hippocampus and ventromedial prefrontal cortex, vmPFC, amygdala) (Chen & Feng, 2022; Chen et al., 2020; Schlüter et al., 2018; Wypych et al., 2019). In addition to the left lateralization, there is solid evidence indicating significant associations between top-down regulatory process and the right DLPFC indeed, particularly given that this region specifically functions in top-down regulation, future self-continuity representation and social decisions (Huang et al., 2025; Knoch & Fehr, 2007; Lin & Feng, 2024). Despite this case, Xu and colleagues demonstrated null effects of anodally stimulating the right DLPFC to modulate either value evaluation or emotional regulation for changing procrastination willingness (Xu et al., 2023). Moreover, a substantial amount of neural evidence supports this conclusion that DLPFC is involved in long-term reward evaluation and value encoding via top-down regulation circuits (Frost & McNaughton, 2017; Jimura et al., 2013; Smith et al., 2018). Using a neurocomputational model, Le Bouc & Pessiglione (2022) provided clear evidence indicating that dorsal PFC signaling expected effort values was significantly attenuated in procrastinators compared to healthy controls (Le Bouc & Pessiglione, 2022). In this vein, this evidence supports this conceptualization that the left DLPFC may be a domain-specific neural signature determining one's procrastination. In light of technical advances, high-resolution transcranial direct current stimulation (HD-tDCS) has been widely used to reveal the potential neurocognitive mechanism of problematic behaviors by modulating cortical excitability, blood-brain barrier permeability and even neuroplasticity (Cirillo et al., 2017; Woods et al., 2016), which is regulated by the NMDA (N-methyl-D-aspartate) system to either bolster LTP (Long-term potentiation) or LTD (Long-term depression) processes (Chrysikou et al., 2022; Shin et al., 2020). For instance, anodic HD-tDCS applied to the left DLPFC was found effective in inhibiting problematic behaviors caused by the lack of self-regulatory ability (Allenby et al., 2018), showing significantly amplified local neural oscillations (Chrysikou et al., 2022). Beyond regional neuromodulation in a dose-free protocol, cumulative evidence has well-documented that the effects of tDCS for neuromodulation are highly dose-dependent and are involved in network-wise covariance (Sabé et al., 2024; Soleimani et al., 2023; Woodham et al., 2025). Thus, this study aims to clarify the brain-behavior association between DLPFC neuromodulation and procrastination, and to test whether observed changes in task valuation and aversiveness are consistent with theoretical models of top-down regulation.

To empirically evaluate these theoretical pathways, we conducted a double-blind, randomized, multiple-session, placebo-controlled design, with a 2 (active HD-tDCS vs. sham control) × 2 (before first neural stimulation vs. after last neural stimulation) full factorial design (see Fig. 1). This HD-tDCS protocol consisted of 7 sessions spaced over 15 days, and each session was implemented every two days. To ensure sound ecological validity, each procrastinator was informed to report one REAL-LIFE task that he/she should complete the following day (e.g., Day 2) after the current neuromodulation session (e.g., Day 1), and was asked to report the ACTUAL performance for

completing this task in that day (Day 2) (see Fig. 1). Based on the temporal decision model of procrastination (Zhang, Liu, et al., 2019), we drew on the experience sampling method (ESM) to estimate the real dynamics of task aversiveness and task-outcome value by using a parameter-free model in each session. More importantly, to clarify which pathway best explains the neurocognitive mechanisms of procrastination, we built upon the mixed-effects general linear model and Quasi-Bayesian mediation model to test whether the changes in task aversiveness and task outcome value modulated by HD-tDCS could predict decreased procrastination. Finally, the follow-up investigation for actual procrastination was conducted for 6 months after the experiment to examine the long-term after-effect of this neural stimulation.

Materials and Methods

This study fully adhered to CONSORT reporting guidelines and was originally preregistered in the OSF repository (10.17605/OSF.IO/Y3EDT). However, due to the technical constraint related to OSF account service (see SI Methods), this OSF page is no longer accessible. For transparency and best practices of open science, based on the original protocol documentations, a preregistration statement has been reconstructed to clarify a priori hypotheses, sample size determinations, and analysis plans for this study (Table S1).

Participants

Due to the lack of diagnostic criteria for clinical procrastinators, we recruited a large-scale sample (n = 1,682) to obtain a stable benchmark distribution. Thus, the procrastinators were captured once their procrastination scores were higher than 66 on General Procrastination Scale (GPS) (see Fig. 2A-B). Following this criterion, a total of 186 participants were included initially, which was in accordance with empirical evidence (i.e., 10 – 15% prevalence of procrastination) (Harriott & Ferrari, 1996). Subsequently, the semi-structured interview was performed to screen those suffering from problematic procrastination and volunteering for this study, thereby enrolling 53 participants (see SI Methods). Seven participants were eventually excluded from the analyses because they voluntarily dropped out before experimental completion. All the included participants were screened for depression and anxiety symptoms (see Tab. 1).

Table 1. Demographic information for included participants.

NM represents active neuromodulation group and sham indicates sham-control group. Anxiety symptoms were measured by State-Trait Anxiety Inventory (STAI). Depression symptoms were tested by Self-Rating Depression Scale (SDS). BF10 describes the Bayesian evidence strength to support alternative hypothesis, with > 3 for a strong evidence.

	active NM		SC		*P*-value (BF_{10})
	Male	Female	Male	Female	
Gender	3	20	3	20	.99 (-)
Age	19.61 ±0.78		22.22 ± 1.44		0.08 (1.03)
SES	2.17 ±0.65		2.34 ± 0.64		0.38 (0.41)
Anxiety	48.48 ± 6.52		47.30 ± 6.87		0.56 (0.33)
Depression	47.13 ±8.27		47.50 ± 9.28		0.88 (0.29)
Procrastination	71.00 ±5.47		72.07 ± 4.77		0.26 (0.48)

A full randomized block design was used to assign participants to both groups (active neuromodulation group, NM; sham-control group, SC) (see Fig. 2C). As the pilot study probing into the effect of single-session tDCS stimulation to change procrastination willingness indicated (t = 2.38, p = .02, 95% CI [0.14, 1.49]; Xu et al., 2023), statistical power was predetermined by G*Power at a relatively medium effect size (1-β err prob = 0.80, f = 0.25), indicating the total sample size at 34 (17 per group) to reach acceptable power. To account for potential attrition, we determined to recruit 36 participants, at least. (see SI Methods and Fig. S1). All the

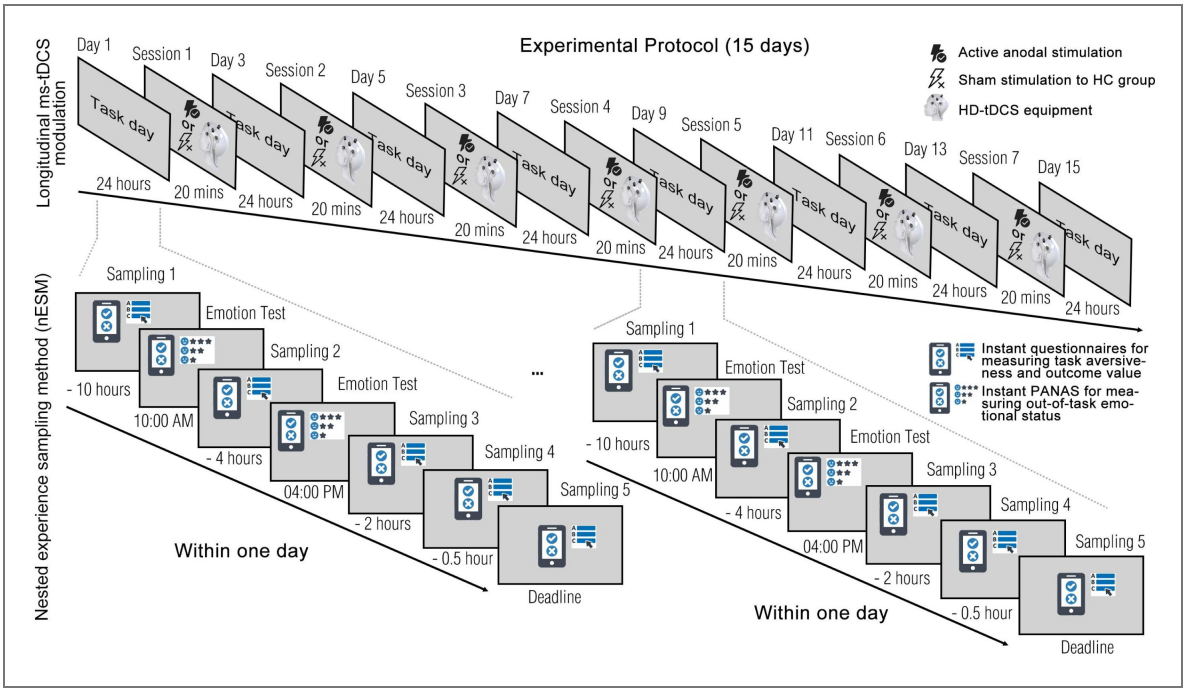


Figure 1. Experimental diagram of this study.

The upper sub-graph illustrates the whole multiple sessions tDCS neuromodulation pipeline including seven sessions (days) and eight task-demanded days. Here, the "flash" icon indicates conducting tDCS neuromodulation (active anodal stimulation for active NM group and sham stimulation for sham NM group). This "+" label means a task-demanded day, where no stimulation is required for participants but all the covariates of interest should be measured by experience sampling methods. The bottom sub-graph reflects specific pipeline in task-demanded days. Participants were required to provide response at five progressive time moments nearing deadline for task aversiveness and outcome value in task-demanded days. In this diagram, the icon of "clock" symbolizes ecological momentary assessment for measuring instant task willingness and outcome value. In addition, twice tests for daily emotions (labeled by "+") were added for participants at 10:00 and 16:00 as covariates of no interests to be adjusted.

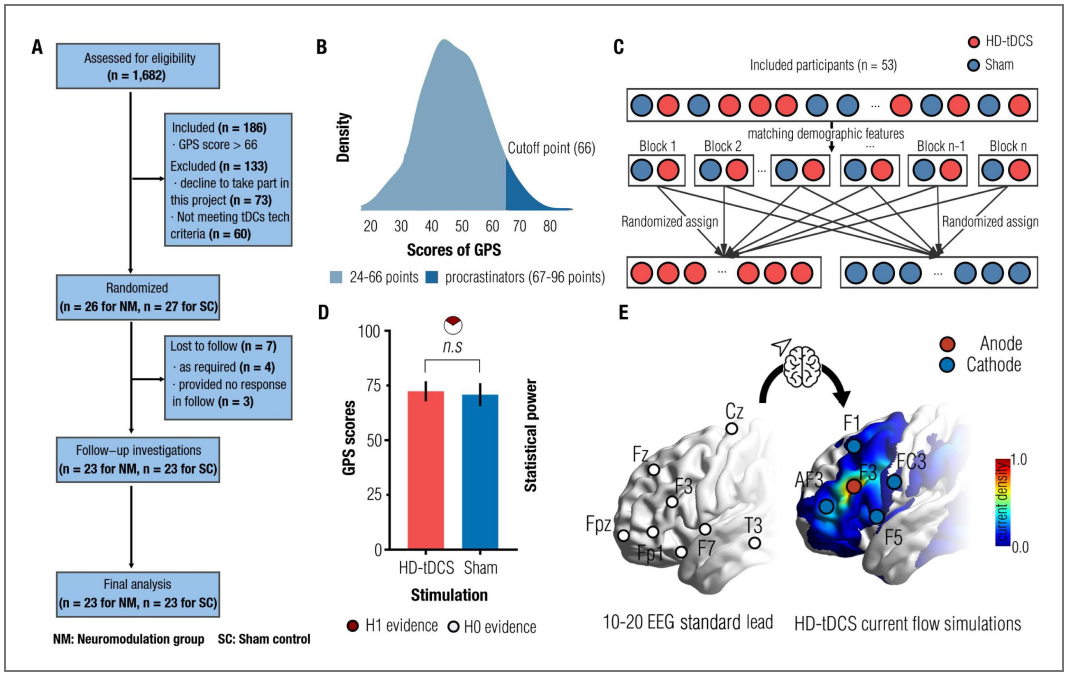


Figure 2. Flow diagram of CONSORT (A) and partial details of randomized groups (B-D) and neural locations of electric pole (E).

B plots the distribution of all the participants procrastination scores (GPS = general procrastination scale). C detailed what the full randomized block design is. D shows the comparison between active neuromodulation group and sham control for procrastination scores. E indicates the pipeline to determine the location of electric pole. The 10–20 EEG standard lead is used to locate the left dorsolateral prefrontal cortex (DLPFC) initially, and the neuronavigation is further utilized to locate the exact location of this targeting region (i.e., left DLPFC).

participants reported no history for HD-tDCS or neuromodulation. No significant differences were found between groups for any demographic characteristics (see Tab. 1). This study and protocol have been fully approved by the Institutional Review Board (IRB) of the School of Psychology, Southwest University (China, IRB200301108).

Measurement

The General Procrastination Scale (GPS) developed by Lay (1986) was used to quantify one's chronic procrastination symptom here (Lay, 1986). This scale was widely adopted in many cross-cultural contexts, and has been reported to have good psychometric properties (Klein et al., 2019). There were two additional items that we added for lie detection, including description of "the sky is red" and "I have never taken a shower". If either one was selected as "agree" by participant, his/her response would be discarded. Internal reliability of the GPS in the current study was found acceptable (Cronbach's ± = 0.890). No significant difference was found between groups for GPS scores (t = −1.08, 95% CI: −4.296 1.283, p = .283; Jeffreys-Zellner-Siow Bayesian factor, BF, BF10 = 0.455, error = 0.020 %).

Experimental design and procedure

Nested cross-sectional longitudinal design

This study used a nested cross-sectional longitudinal design to investigate whether the multiple-session anodal HD-tDCS targeting the left DLPFC could reduce actual procrastination behavior and to probe how this effect manifests. To assess procrastination in daily life, we implemented a 15-day protocol alternating between Neuromodulation Days (Days 2, 4, 6, 8, 10, 12, 14) and Task Days (Days 1, 3, 5, 7, 9, 11, 13, 15). On the Neuromodulation days, the 20-min anodal HD-tDCS neuromodulation targeting the left DLPFC was performed for the HD-tDCS active group at intervals of 2 days, while the shamcontrol group received sham HD-tDCS training. This HD-tDCS training was repeated for a total of seven sessions, and lasted 15 days (see Fig. 1). Crucially, to capture procrastination in ecologically valid contexts, priori to receiving either active or sham HD-tDCS (administered between 09:00-18:00), participants were instructed to specify a real-life task they were personally obligated to complete the following day, with a self-defined deadline strictly constrained to 18:00-24:00 to ensure ≥24 hours between stimulation offset and task deadline, thereby isolating offline after-effects. This task should meet the following three criteria: (a) it should be already assigned in the real-world settings; (b) deadline should be constrained to 18:00-24:00 (see above); (c) it should be more likely to induce procrastinate. By doing so, more than 300 real-life tasks were collected, spanning academic (e.g., "submit a statistics homework assignment"), occupational (e.g., "draft and email a project proposal"), administrative (e.g., "complete online application for Class C driver's license"), selfimprovement (e.g., "practice guitar for ≥30 minutes"), domestic (e.g., "do laundry"), and health-related (e.g., "running 2,000m for exercise"). Full task list has been tabulated in the Appendix 1. As primary outcomes, all the participants were required to report task-execution willingness (TEW) (Zhang & Feng, 2020; Zhang, Liu, et al., 2019), for a real-life task 24 hours post-neuromodulation. Thus, procrastination willingness was quantified as 100-TEW score (see underneath for details). Furthermore, we asked participants to report the actual task completion rate (CR) of the task at the deadline (e.g. participant A finished 90% homework at deadline and reported this situation to us at deadline). In this vein, the actual procrastination rate (PR) was quantified as 1-CR.

On the Task Day, we developed a mobile app to implement experience sampling method (ESM) for tracking one's real-time evaluation of task aversiveness and task outcome value (see Fig. 1). The task aversiveness describes how disagreeable one perceives performing a given real-life task to be, whereas outcome value refers to the subjective benefits of the task outcome brought about by completing the task before the deadline (Zhang & Feng, 2020). As theoretically conceptualized by the temporal decision model (TDM) of procrastination, the perceived task aversiveness is hyperbolically discounted when approaching deadline, showing sharply discounting when faring away from deadline but slowly discounting once nearing deadline (Zhang & Feng, 2020). Thus, considering this nonlinear dynamics inherent in this hyperbolic discounting, the five recording

moments of ESM were selected per task a priori by using a log-spaced temporal sampling scheme (Myerson et al., 2001), with increasing sampling density toward the deadline, such as moments of 10:00 (earliest), 16:00, 18:00, 19:30, 20:00 (deadline). The five sampling points could meet statistical prerequisite in the hyperbolic model fitting, requiring ≥ 4 points (Green & Myerson, 2004). To do so, recording moments of tasks were individually tailored for each task per participant in this ESM procedure. To obviate the confounds of daily emotions in task aversiveness evaluation, we used the averaged scores of PANAS at 10:00 (noon) and 16:00 (afternoon) as anchoring points to quantify one's daily emotions by using this ESM app. Before each session of HD-tDCS training, each participant was required to report a real-life task whose deadline is tomorrow. To obtain the long-term effect of HD-tDCS (i.e., the interval between HD-tDCS and task completion is at least 24 hours), the task deadline that participants reported was required to be between 18:00 – 24:00. Once a sampling time reached, this app would send a digital message to require participants to fill online form for data collection.

Quantification of covariates of interests

Outcome variables of this study were twofold: one is task-execution willingness and another is procrastination rate (PR). Task-execution willingness is used to evaluate one's subjective inclination to avoid procrastination (Zhang & Feng, 2020). In this vein, we used a 100-point scale to require participants to report their task-execution willingness (0 for "I will definitely procrastinate this task" and 100 for "I will take action to complete this task immediately"). This metric was recorded 24 hours after neuromodulation to examine its long-term effects. PR is used to quantify the extent to which one task has been procrastinated, and was calculated as 1 − CR (task completion rate). Critically, at the precise deadline, the app prompted participants to (a) indicate task completion status (yes/no), and if incomplete, (b) report the percentage completed (1-99%), defined as the Task CR, while simultaneously uploading objective evidence (e.g., screenshots of submitted files, photos of physical outputs, system-generated logs, or app-exported records). If the task was actually completed before the deadline, the CR would be 100% and the PR would be calculated as 0% (1-CR). PR was recorded at the actual task deadline for each participant. We were also interested in re-investigating their actual procrastination by using PR 6 months after the last neuromodulation to test the long-term after-effect of this neuromodulation.

From what has been mentioned above, task aversiveness and outcome value were considered key factors to explain the effect of neuromodulation on reducing procrastination in the current study. To quantify one's task aversiveness, participants were required to rate their feelings towards the task by using a 100-point visual analog scale (i.e., How do you feel in the current moment when you need to complete this task before deadline, with 0 for "extremely unpleasant", 50 for "totally neutral" and 100 for "extremely pleasant"). Likewise, participants are also required to rate their outcome value by using a 100-point visual analog scale (i.e., How much do you desire to obtain incentive outcome of this task, with 0 for "extremely weak", 50 for "medium" and 100 for "extremely strong"). As articulated temporal decision theoretical model above, the task aversiveness evoked by executing a task was temporally dynamic in a hyperbolic discounting pattern, with sharply discounting in faring away from deadline but slowly discounting in nearing deadline (Zhang & Feng, 2020). To quantitatively characterize the task aversiveness with consideration for its dynamics, the model-free area under the curve (AUC) was calculated. Specifically, based on the log-spaced temporal sampling rule, task aversiveness was measured by 100-point visual analog scale at the five sampling moments. Then, the task aversiveness discounting (A) was calculated as 1- (A(t) / A(earliest)), where t(earliest) was the earliest sampling point, serving as the reference for immediate execution. Subsequently, using the GraphPad Prism software (v9, 525), the AUC was computed as the trapezoidal integration between task aversiveness discounting and time across five data points, basing on the Myerson algorithm (Myerson et al., 2001). By doing so, a higher AUC reflects stronger temporal discounting of task aversiveness along with nearing deadline, which means that participants experience a faster decline in subjective aversiveness as execution is delayed, yielding lower effective aversiveness and reduced avoidance behavior. As for the task outcome value, it was theoretically posited as a

relatively stable evaluation of the task (Zhang & Feng, 2020). Therefore, it was quantified by the selfreported 100-point visual analog scale after neuromodulation at least 12 hours later, to ensure no online effect.

HD-tDCS protocol

The HD-tDCS suit (stimulater and 4 × 1 multichannel stimulation adapter, MSA) that this study used was produced by the Soterix Medical Inc., and has been widely verified safe, effective and reliable for public (Villamar et al., 2013). Based on advanced properties of 4 × 1 MSA, the targeted areas for current flow can be constrained within 2.5 cm^2 (Villamar et al., 2013).

To position electrodes into targeted areas (left DLPFC), the 10-20 international system for EEG was initially used to mark potential nodes, and determined Cz as reference point. There is compelling evidence to claim that the left F3 could be used as target node for modulating the left DLPFC (Seibt et al., 2015; Tsukuda et al., 2025). In this vein, the central anodal electrode was determined onto F3, and four return electrodes surrounded central electrode at outside of 7.5 cm, including F5, AF3, FC3 and F1 (see Fig. 2E). Ramp-up and ramp-down durations were set to 30 seconds. To further locate the targeted areas, the high-definition neuronavigation system (ANT Neuro Inc., Welbergweg, Germany) was performed. Results indicated the accurate position that we pre-determined by showing a high overlap probability over the left DLPFC (MNI Coordinate: −51 40 18, 94.33 % overlapping probability) (see SI Method and Fig. 2E). In addition, the coordinates of this targeted area were retrieved from the Brede Database (*http://neuro.imm.dtu.dk/services/*), and showed highly pertinent functions related to DLPFC.

Before stimulation, participants were informed to clean the scalp to reduce resistance. Then, cotton swabs were used to separate hair until the scalp surface become visible. Subsequently, the electrically conductive gel (about 1.5 ml) was introduced into the plastic casing facilitating constraint of current flow. Next, the Ag/AgCI sintered ring electrodes were placed onto plastic casing and covered with a cap to lock them in the right positions. To reduce discomfort, the electrode cables were taped elsewhere. Further, the above processes would be re-adjusted if any electrode resistances were found larger than 1.5 units (Villamar et al., 2013). Once all the processes had been completed, the stimulator would be launched.

Participants in the HD-tDCS training group underwent constant electric current of 2.0 mA targeting the left DLPFC for 20 minutes. Results from the simulation of electric density showed a peak current of ~0.5mA/cm^2 at the central electrode and of ~0.125 mA/cm^2 at the four return electrodes, thereby indicating the safety and effectiveness. As for the sham-controlled group, the stimulator would deliver current flow with 2.0 mA during the first and last 30 seconds to elicit sense of electric stimulation for blinding of them. To obtain the pure offline effect, these measures for task-execution willingness, task aversiveness, and outcome value were conducted after stimulation at least 12 hours (Bikson et al., 2016).

Statistics

All the statistics were implemented by *R* (*https://www.rstudio.com/*) and *R*-dependent packages.

To clarify whether multiple-session HD-tDCS neuromodulation can reduce procrastination, the general linear mixed-effects model (LMM) was constructed for subjective procrastination willingness (i.e., self-reported visual analog scores) and actual procrastination behavior (i.e., real-world task-completion rate before deadline). Here, sex, age and socioeconomic status (SES) were modeled as covariates of no interest. As the National Bureau of Statistics (China) issued (*https://www.stats.gov.cn/sj/tjbz/gjtjbz/*), on the basis of per capita annual household income, the SES was divided into seven hierarchical tiers from 1 (poor) to 7 (rich). To obviate subjective rating bias stemming from individual daily mood, we separately measured participants' daily emotional fluctuation at 10:00 and 16:00 using a self-rating visual analog item (i.e., "How do you feel today?", 0 for "completely uncomfortable" and 100 for "definitely happy"). By doing so, the averaged score of those self-rating emotions at the two time points was modeled into the LMM as a covariate of no interest. Given the risks of convergence failure with the two correlated random-effects structure (i.e., treatment days and self-reported emotions), we hypothesized that the random effects are

independent, leading to model simplification. Consistent with this assumption, model comparisons favored the simplified independent structure over the full correlated structure for both outcomes. For the actual procrastination model, the simplified model showed lower AIC (Δ = −1.0) and BIC (Δ = −11.8), with a non-significant likelihood ratio test ($\chi^2(3) = 5.04$, $p = .17$). For the task-execution willingness model, the simplified model was also preferred (ΔAIC = −5.5, ABIC = −16.4; LRT: $\chi^2(3) = 0.51$, $p = .91$). This analysis was implemented using the "lme4" and "lmerTest" packages. Employing "emmeans" package, simple effects were also tested at baseline and post-last-intervention using Tukey-adjusted pairwise comparisons of estimated marginal means from the full LMM, controlling for covariates and random-effects structure. To validate statistical robustness, instead of continuous outcomes for parametric tests, we also conducted a between-group comparison for the number of tasks that procrastination emerges by using the nonparametric x^2 test with ϕ correction or Fisher exact test. Regarding the 6- month follow-up investigation, this LMM was also built to examine the long-term after-effect of neuromodulation on reducing actual procrastination. To examine whether our findings were sensitive to the distributional assumptions of the dependent variables, we re-analyzed the main models using an alternative distributional specification. Given that both procrastination rates (ranging from 0% to 100%) and task-execution willingness (measured on a 0–100 visual analog scale) are bounded continuous outcomes, and that procrastination rates frequently reached the lower boundary (0%) in the present study, a Beta regression model with a logit link function was employed for a sensitivity analysis.

To ascertain the neurocognitive mechanism of tDCS in reducing procrastination, the Quasi-Bayesian mediation analysis was used to model the association between the effects of tDCS, task aversiveness/outcome and decreased procrastination. To build upon this model, the tDCS treatments were inputted as independent variables, and the task aversiveness/outcomes were modeled as mediating variables by using the "Mediation" package (*https://cran.r-project.org/web/packages/mediation/*) (Imai et al., 2010). We estimated these pathway effects (i.e., average mediation effects, δ; average direct effect, ζ; total effects, ρ) by using Markov Chain Monte Carlo (MCMC) sampling. To improve the statistical reliability, the sequential ignorability assumption was tested by using sensitivity analysis. Details for the statistical principals and basis could be found elsewhere (Imai et al., 2010). As these mediation analyses are based on observational measures rather than experimentally manipulated mediators, they do not establish causal pathways. Simulation studies have shown that such analyses can suffer from inflated Type 1 error rates. Results should therefore be interpreted as hypothesis-generating and statistically consistent with the proposed theoretical model, rather than as confirmatory evidence of causal mechanisms.

Results

Blinding

In both groups, almost all participants reported perceiving acceptable pain stemming from current stimulation, and believed they were receiving treatment, with 91.30% (21/23) for active neuromodulation group (NM) and with 86.95% (20/23) for sham control group (SC) ($X^2 = 0.224$, $p = .636$). All the participants were engaged in the identical experimental procedures excepting stimulation's type (active *vs* sham). In addition, statistical models excluded Session 1 and Session 4 because participants reported additional unexpected events that uncontrollably disrupt task execution in both groups (see SI Result and Tab. S2).

Multiple-sessions HD-tDCS (ms-tDCS) can alleviate procrastination

To identify whether ms-tDCS targeting the left DLPFC can alleviate subjective procrastination willingness and actual procrastination behavior, a general mixed-effects linear model (LMM) with Satterthwaite's method was built, with task-execution willingness and actual procrastination rates (PR) as primary outcomes, respectively. For procrastination willingness, results showed a statistically significant interaction effect between multi-session neuromodulations and groups ($\beta = -7.84$, SE = 1.80, $t = -4.36$, DF = 45.6, $p < .001$; Fig. 3A). In the post-hoc simple effect analysis, it

demonstrated a significantly increased task-execution willingness (i.e., decreased procrastination willingness) after neuromodulation in the active neuromodulation group (NM-before: 35.65 ± 30.21, NM-after: 80.43 ± 19.92, Mean Diff = 41.79, SE = 7.58, DF = 103.4, *t*.ratio = 5.51, $p < .0001$, Tukey correction), but no such effects were identified in the sham control group (SC-before: 37.57 ± 26.46, SC-after: 47.35 ± 30.49, Mean Diff = 2.58, SE = 7.56, DF = 96.8, *t*.ratio = 0.34, $p = .73$, Tukey correction) (Fig. 3B-C). A linear uptrend for task-execution willingness was further observed across multiple sessions in the active NM group, indicating gradually increasing neuromodulation effects (Fig. 3D; $p < .01$, Mann-Kendall test). For actual procrastination behavior, changes to actual procrastination rates across all the sessions have been detailed in the Fig. 3E. Similarly, a statistically significant interaction effect was identified here ($\beta = -7.37$, SE = 2.40, $t = -3.02$, DF = 46.6, $p = .004$), and the simple effect analysis further revealed decreased actual procrastination rates after ms-tDCS in the active neuromodulation group (NM-before: 56.74 ± 39.10, NM-after: 0.00 ± 0.00, Mean Diff = 44.40, SE = 9.36, DF = 110.0, *t*.ratio = 4.74, $p < .0001$, Tukey correction), but no such prominent changes found in the sham control group (SC-before: 46.47 ± 40.76, SC-after: 33.35 ± 37.82, Mean Diff = 7.53, SE = 9.28, DF = 102.0, *t*.ratio = 0.81, $p = .42$, Tukey correction) (Fig. 3F-G). Also, a significant downtrend for procrastination rates across all the sessions was identified in the active NM group (Fig. 3H; $p < .01$, Mann-Kendall test).

Furthermore, the nonparametric X^2 test of R × C contingency table was conducted for the count of procrastinated tasks, by treating this outcome (i.e., whether a participant actually procrastinates the task in real-world settings) as an ordinal variable. Results showed a significant reduction in group-averaged procrastination frequency in the active neuromodulation group after last-session HD-tDCS, but not in the sham control group (NM-before: 69.56% (16/23 participants), NM-after: 0.00% (0/23 participants); SC-before: 69.56% (16/23 participants), SC-after: 56.52% (13/23 participants), $X^2 = 10.08$, $p < .001$), partly indicating a high statistical robustness. To systematically test whether such effects are biased by extreme data points or patterns, we reran the main LMMs by iteratively removing data from the last two sessions, which showed extraordinarily high effectiveness from neuromodulation (e.g., all the participants in the neuromodulation group had no actual procrastination behavior in session #6 and #7). Results showed the significant group*neuromodulation sessions interaction effects across all those nested models (removing session #6, #7 or both, all $p < .05$; see SI Results and Tab. S3–4), potentially indicating a statistical robustness from the data pattern. Furthermore, as a sensitivity analysis, we reran the main LMMs using Beta regression distribution with a logit link function, which is appropriate for both bounded outcomes mentioned above (i.e., procrastination rate and procrastination willingness). The main effects (i.e., Group and Treatment day) and their interaction remained significant for both procrastination rate and willingness (see SI Results and Tab. S5), confirming that our findings are robust to alternative distributional assumptions. In brief, these findings provided empirical evidence to support that ms-tDCS neuromodulation targeting the left DLPFC can be an effective way to reduce both procrastination willingness and actual procrastination.

Ms-tDCS changes task aversiveness and task-outcome value

Both task aversiveness and task outcome value serve as key pathways determining whether one would procrastinate. To this end, we further utilized a generalized linear mixed-effects model to examine the effects of ms-tDCS on changes in task aversiveness and task outcome value. Task aversiveness changes across all the sessions are shown in the Fig. 4A and 4C. We demonstrated a statistically significant decrease in task aversiveness and an increase in task outcome value via ms-tDCS in the neuromodulation group (Task aversiveness: interaction effect, $\beta = -0.12$, SE = 0.04, DF = 46.69, $t = -3.28$, $p = .002$; simple effect, NM-before$_{(AUC)}$: 1.13 ± 0.54, NM-after$_{(AUC)}$: 1.95 ± 0.84, Mean Diff = 0.81, SE = 0.16, DF = 104.5, *t*.ratio = 4.91, $p < .001$, Tukey correction; Outcome value: $\beta = -6.76$, SE = 1.74, DF = 46.2, $t = -3.89$, $p < .001$; simple effect, NM-before: 35.87 ± 27.83, NM-after: 73.09 ± 23.34, Mean Diff = 39.36, SE = 7.25, DF = 103.7, *t*.ratio = 5.43, $p < .001$, Tukey correction; see Fig. 4B), but not in the sham control group (Task aversiveness: SC-before$_{(AUC)}$: 1.07 ± 0.51, SC-after$_{(AUC)}$: 1.28 ± 0.46, Mean Diff = 0.17, SE = 0.17, DF = 98.3, *t*.ratio = 1.06, $p = .29$, Tukey correction; Outcome value: SC-before: 34.00 ± 25.17, SC-after: 40.13 ± 28.94, Mean Diff = 5.54, SE = 7.26, DF =

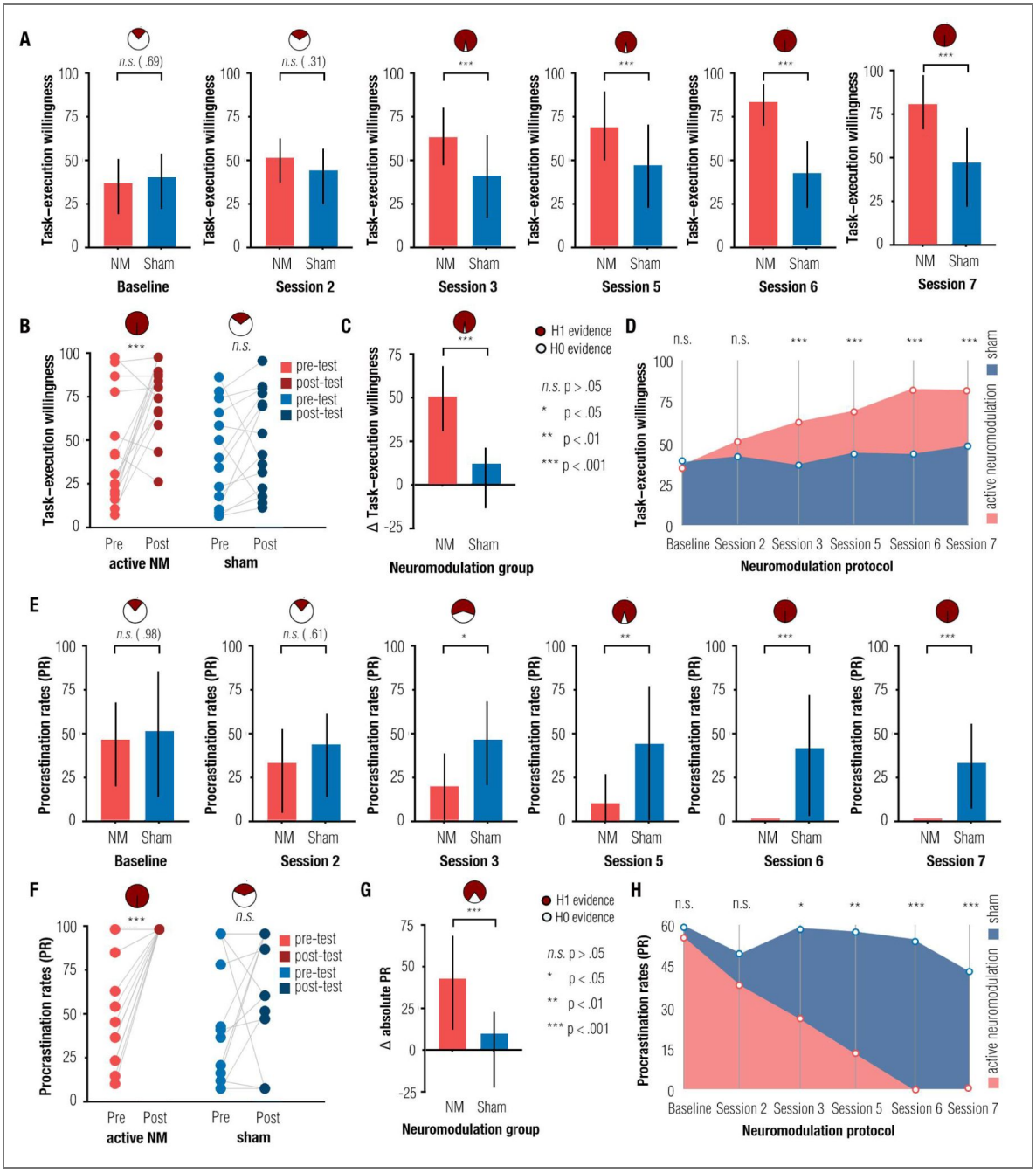


Figure 3. Results of neuromodulation effects to task-execution willingness and procrastination rates (PR).

A shows the effects of neuromodulation to increase task-execution willingness for both active group and sham control across sessions that included in formal analysis (session 0 (baseline), 2, 3, 5, 6, 7). B illustrates the effects of whole neuromodulation round to task-execution willingness for both groups. C plots the changes of task-execution willingness for both groups after neuromodulation. D provides a line chart to show the changes of task-execution willingness across each session that included in formal analysis. E shows the effects of neuromodulation to reduce PR for both active group and sham control across sessions that included in formal analysis. F illustrates the effects of whole neuromodulation round to PR for both groups. G plots the absolute changes of PR for both groups after neuromodulation. H provides a line chart to show the changes of PR across these sessions that included in formal analysis. Pie graph for each comparison represents the corresponding result of Bayesian factor inference, with brown piece for supporting H1 evidence and white piece for supporting H0 evidence. Each bar indicates mean value, and each line placed onto the bar reflects standard deviation (SD).

98.7, *t*.ratio = 0.76, p = .44, Tukey correction; see Fig. 4D). In the neuromodulation (NM) group, task aversiveness steadily decreased with the cumulative number of stimulation sessions, while perceived task outcome value increased significantly (see Fig. 4E-F, p < .05, Mann-Kendall test). Thus, these findings are consistent with the view that neuromodulation of the left DLPFC is associated with reduced task aversiveness and increased task-outcome value.

Increased task outcome value but not decreased task aversiveness predicts reduced procrastination

Given the dual neurocognitive pathways identified above—reduced task aversiveness and increased task-outcome value—we proposed that these changes may reflect downstream consequences of prefrontal neuromodulation on value-based decision processes, statistically consistent with theoretical models of top-down regulation. To this end, we utilized a general linear model to regress decreased task aversiveness and increased task outcome value to changes in task-execution willingness. In this model, increased task outcome value ($\Delta_{\text{Outcome value}}$) significantly predicted increased task-execution willingness ($\Delta_{\text{task-execution willingness}}$) ($X^2$ = 15.95, p <.01, R^2 = .40; $\Delta_{\text{Outcome value}}$: β = 0.61, S.E. = 0.12, p < .001,95% CI: 0.37 – 0.86), whereas no significant effect was observed for predicting task-execution willingness through decreased task aversiveness ($\Delta_{\text{Task aversiveness}}$:$\beta$= 0.10, S.E. = 0.12, p = .41, 95% CI: −0.14 – 0.34). Likewise, for actual procrastination behavior in real-world settings, increased outcome value ($\Delta_{\text{Outcome value}}$) was identified to be significantly predictive, whereas decreased task aversiveness showed null effects (see Tab. 2). Collectively, these findings provide statistical evidence consistent with the hypothesis that the outcome-value pathway may contribute to procrastination reduction.

Table 2. Summary for general linear model in predicting changes of task aversiveness and outcome value to actual procrastination.

S.E. means standard error. * p < .05.

	β	S.E	*P* value	Odd Ratio (OR)	adj R^2
Δ task aversiveness	0.62	0.42	0.13	1.85	
Δ outcome value	0.85*	0.42	0.04	2.34	.29

Increased task outcome value is specifically associated with reduced procrastination in the contexts of neuromodulation

To explore the potential neurocognitive pathways of procrastination, the Quasi-Bayesian mediation analysis was undertaken, with increased task outcome value as a statistically mediated variable. As an exploratory analysis, results indicated that increased task outcome value was associated with changes in the task-execution willingness (δ = 21.73, p < .01; ζ = 11.25, p = .07, ρ = 32.99, p < .01, simulation = 1,000; see Fig. 5A) and real-world procrastination (δ = 30.75, p < .01; ζ = 3.05, p = .52, ρ = 33.81, p <.01, simulation = 1,000; see Fig. 5B), in the context of ms-tDCS neuromodulation. To ensure the statistical robustness and specificity of these findings, the sensitivity analysis was implemented by changing sampling parameters and outcome variables. By doing so, those findings were validated statistically robust, as shown by replicated observations across bootstrapping sampling subsets (see SI Results and Tab. S6–7). Moreover, the results of the control analysis further validated the specificity of these findings by showing a null statistically mediated effect of this model to predict one's task aversiveness (see SI Results and Tab. S8). In summary, these findings identified a statistical pathway consistent with the theoretical model: neuromodulation of the left DLPFC was associated with increased task-outcome value, which in turn was associated with reduced procrastination. Nevertheless, all mediation findings are now explicitly labeled as "exploratory" and framed as quantifying statistical associations consistent with the TDM pathway, not causal mediation.

Figure 4. Results of neuromodulation effects to task aversiveness and outcome value.

A shows the effects of neuromodulation to increase AUC of task aversiveness for both active group and sham control across sessions that included in formal analysis (session 0 (baseline), 2, 3, 5, 6, 7). Higher AUC indicates lower task aversiveness as a given task is increasingly executed. B plots the changes of AUC of task aversiveness for both groups after neuromodulation. C shows the effects of neuromodulation to increase outcome value for both active group and sham control across sessions that included in formal analysis. D plots the changes of outcome value for both groups after neuromodulation. E provides a line chart to show the changes of AUC of task aversiveness across these sessions that included in formal analysis. F provides a line chart to show the changes of outcome value in the same manner. Pie graph for each comparison represents the corresponding result of Bayesian factor inference, with brown piece for supporting H1 evidence and white piece for supporting H0 evidence. Each bar indicates mean value, and each line placed onto the bar reflects standard deviation (SD).

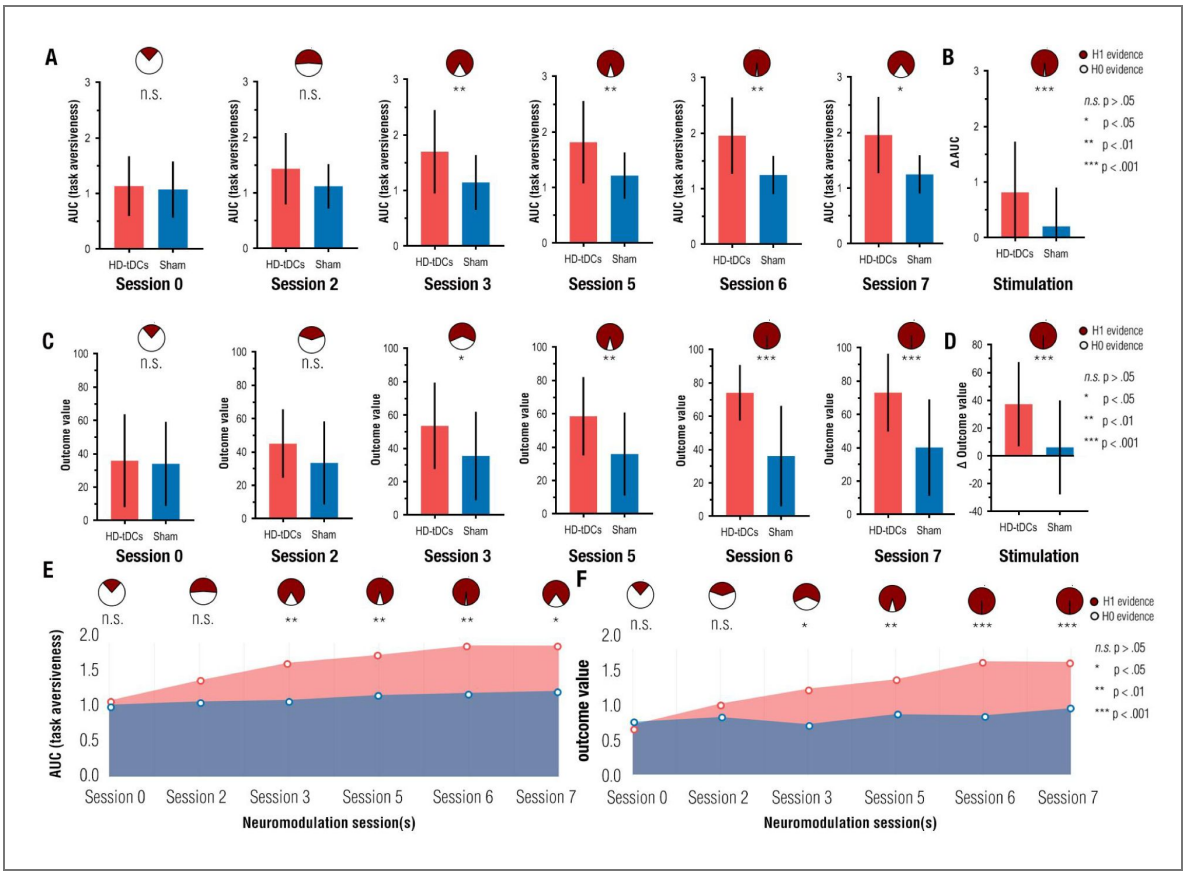
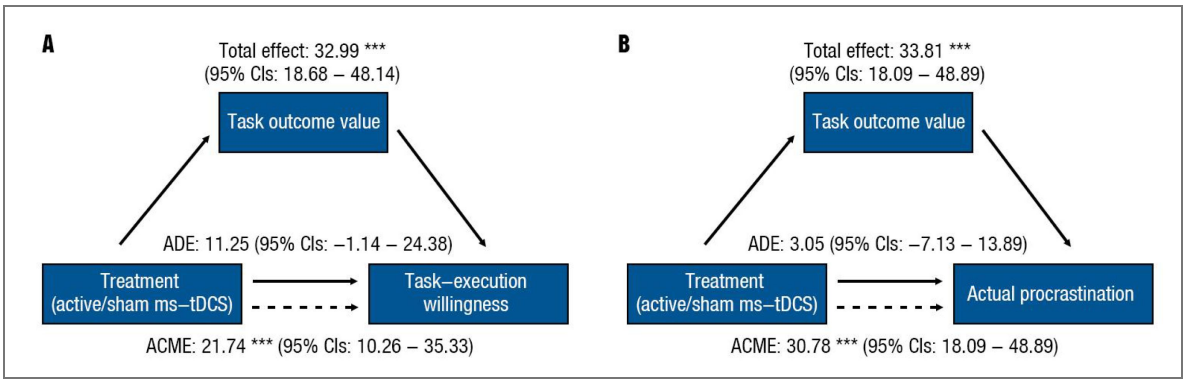


Figure 5. Results of Quasi-Bayesian model for the mediated role of increased task outcome value in the association between neuromodulation and task-execution willingness (A) and actual procrastination rates (B).

ADE = average direct effect, ACME = average causal mediation effect, CI = confidence interval.



Long-term effects of ms-tDCS

We have also attempted to conduct a follow-up investigation to test the long-term after-effect of ms-tDCS in reducing actual procrastination. Almost all the participants had undergone follow-up except one in the neuromodulation group after last neuromodulation for 6 months (N_{NM} = 22, N_{SC} = 23). Thus, the LMM was constructed, with the PR before first neuromodulation vs. PR after last neuromodulation for 6 months as covariates of interest. Results showed the statistically significant group*time interaction effects (β = 16.5, SE = 9.9, p = .049). Simple-effect model demonstrated a decrease in actual procrastination rates in the active neuromodulation group after last stimulation for 6 months compared to baseline (β = - 22.05, SE = 10.0, p= .038, Tukey correction; NM-before: 40.68 ± 37.96, NM-after$_{6\text{-months}}$: 18.63 ± 29.80), and revealed null effects in the SC group (β= 1.26, SE = 9.78, p= .99, Tukey correction; SC-before: 46.47 ± 40.75, SC-after$_{6\text{-months}}$: 47.73 ± 39.18) (see Fig. 6). Furthermore, using a nonparametric X^2 test to compare differences in the number of procrastinated tasks, we still found a statistically significant reduction in procrastination frequency in NM group after neuromodulation for 6 months compared to baseline (X^2 = 3.30, p = .035, NM-before: 68.19% (15/22), NM-after$_{6\text{-months}}$: 40.91% (9/22)), while no significant changes were observed in the SC group (X^2 = 0.11, p = .74, SC-before: 69.56% (16/23), SC-after$_{6\text{-months}}$: 73.91% (17/23)). Therefore, beyond short-term effects, the benefits of ms-tDCS neuromodulation on reducing procrastination were still detectable at a 6-month follow-up, providing preliminary evidence consistent with long-term after-effects.

Discussion

In the current study, by performing anodal ms-tDCS neuromodulation on the left DLPFC, both procrastination willingness and actual procrastination behavior were significantly decreased in real life. Additionally, a 6-month follow-up investigation revealed the long-term retention of such effects. Furthermore, this neuromodulation was found to decrease task aversiveness and increase outcome values; notably, only increased task-outcome value could predict decreased procrastination. On balance, our findings provide evidence consistent with the hypothesis that neuromodulation of the left DLPFC is associated with reduced procrastination, primarily through increasing task-outcome value rather than merely reducing task aversiveness. In addition, this study provided an effective way to reduce actual procrastination by using ms-tDCS neuromodulation.

One contribution of this study is to provide empirical evidence partially consistent with the temporal decision model (TDM), showing that increased task-outcome value—rather than decreased task aversiveness—was statistically associated with reduced procrastination following DLPFC neuromodulation. Neurobiological substrates of procrastination have been investigated in recent years, and have demonstrated the crucial roles of the left DLPFC in predicting procrastination (Chen & Feng, 2022; Chen et al., 2021; Chen et al., 2022; Hu et al., 2018; Liu & Feng, 2017; Zhang et al., 2017; Zhang et al., 2016). Not only the brain functional anomalies of the left DLPFC but also the neuroanatomical disruptions of self-regulatory brain network constituted by the left DLPFC were found to be linked with more procrastination behaviors (Chen & Feng, 2022; Zhang et al., 2016). Notwithstanding this, it still remains unclear to claim their brain-behavior relationship - that is - no known evidence existed to clarity whether changes of the left DLPFC lead to procrastination or vice versa. The current study demonstrated the role of the left DLPFC in procrastination by showing that the neuromodulation of the left DLPFC indeed manipulated procrastination, and thus provided straightforward and powerful evidence to fill this gap.

It has long been acknowledged that the left DLPFC is consistently implicated in top-down regulatory processes and value-based decision-making, such as patience to wait long-term gratification for a delay, inhibition of impulsiveness, and control of game addiction (Cohen & Lieberman, 2010; Lin & Feng, 2024). Furthermore, the increased activation of the left DLPFC has been observed during the exertion of self-regulation which modulates value signals (Hare et al., 2009; Harris et al., 2013). Meanwhile, procrastination has been argued to be the

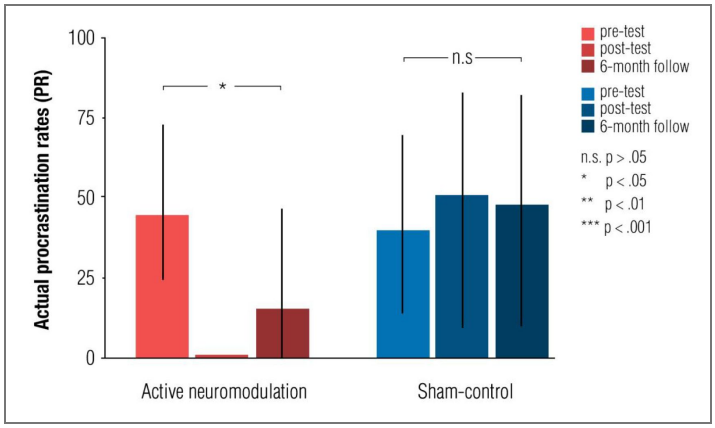


Figure 6. Changes of actual procrastination rates among pre-test, post-test and 6-month follow-up for active neuromodulation group and sham-control group.

Pre-test means to test the actual procrastination rates before first HD-tDCS neuromodulation. Post-test means to test the actual procrastination rates after last neuromodulation. The 6-month follow-up means to re-investigate the actual procrastination rates after last neuromodulation for 6 months.

consequence of self-regulation failure for a long time (Ariely & Wertenbroch, 2002; Rebetez et al., 2018; Rozental & Carlbring, 2014). Supporting this, both the brain morphological disruptions in the DLPFC and anomalies in the functional coupling of the DLPFC-based regulatory network have been correlated with procrastination tendencies (Xu et al., 2021; Yang et al., 2021). Moreover, it was worth noting that the increased activation of the left DLPFC was found to be involved in outcome value evaluation through self-control regulation (Chen et al., 2018; Zha et al., 2019). There is more straightforward evidence to substantiate the role of manipulating the DLPFC in changing one's subjective value evaluation (Huang et al., 2017; Minati et al., 2012). Moreover, the theoretical explanations and empirical evidence have increasingly converged into one line for claiming that the increased task outcome value would prompt more motivation to drive one to take action immediately and thus reduce procrastination (Zhang et al., 2019). Building on this foundation, among several theoretical interpretations and cognitive pathways, our study showed the one plausible neurocognitive mechanism of procrastination: the cortical excitability of the DLPFC produced by active neuromodulation may engage prefrontal regulatory networks to increase task outcome value, which in turn is associated with reduced procrastination behavior, statistically supporting the theoretical accounts of temporal decision model (TDM, Zhang et al., 2019). TDM uniquely posits procrastination as contingent on the trade-off between task aversiveness and taskoutcome value; our observation that only increased outcome value—not decreased aversiveness—statistically predicted reduced procrastination aligns precisely with TDM's hypothesis that value-based processes may dominate affective-avoidance processes in driving behavioral change. However, neither TMT (which emphasizes temporal discounting of utility per se) nor the mood repair perspective (which prioritizes short-term affect regulation) explicitly predicts this dissociable pattern. Despite statistically supporting the TDM, we acknowledge that alternative neurocognitive mechanisms could contribute to the observed reductions in procrastination. For instance, repeated exposure to the experience-sampling protocol may have enhanced participants' awareness of task progress or facilitated feedbackbased learning, thereby increasing the subjective value of goal completion independent of DLPFC neuromodulation. Similarly, improvements in working memory for task maintenance, attentional allocation to reported goals, or strategic shifts in goal selection across sessions could plausibly mediate the intervention effects. While our double-blind, sham-controlled design and inclusion of daily emotional covariates help mitigate some non-specific confounds, the present study did not incorporate direct measures of these alternative processes. Consequently, we cannot definitively isolate the value-based pathway posited by the TDM from concurrent contributions of attention, learning, or executive functions.

In addition, another contribution of the current study is to provide an effective way to reduce both procrastination willingness and actual procrastination in real-life tasks. As mentioned above, despite the fact that multifarious behavioral interventions and evidence have been massively studied for overcoming procrastination, they have shared a common aim - that is - reducing the intention-action gap (Miao et al., 2024; van Hooft et al., 2005). Eerde and Klingsieck (2018) put forward an insightful standpoint established by meta-analytic evidence: procrastination is characterized as an intention-action gap rather than an intention to postpone (van Eerde & Klingsieck, 2018). This notion has been partly supported by showing that cognitive behavior therapy (CBT) for goal-directed behaviors may outperform other interventions focusing on time management (Rozental et al., 2018). Moreover, the trans-theoretical model of procrastination has shown that the behavioral intervention may be effective in changing one's motivation to overcome procrastination but not in actual behaviors (Grunschel & Schopenhauer, 2015). Thus, the ms-tDCS protocol employed here may offer a promising avenue for reducing procrastination by attenuating the intention-action gap, though further validation in larger, clinically screened cohorts is warranted. Furthermore, both 2-day-interval long-term effects and the 6-month long-term retention of the effects of ms-tDCS on reducing actual procrastination have been revealed as well. Thus far, the trends in adopting tDCS neuromodulation techniques in many aspects of behavioral therapies have emerged, but concern for a long-lasting effect of single session stimulation has continued (Brunoni et al., 2013; Brunoni et al., 2012). To tackle this concern well, instead of single-session tDCS, the current study adopted multiple-session stimulation to

implement neuromodulation on the left DLPFC, which facilitates long-term effects (Au et al., 2017; Tedesco Triccas et al., 2016). Existing neurobiological theories and empirical evidence have demonstrated that multiplesession tDCS stimulation could boost cumulative effects of consolidation for activitydependent LTP (long-term potentiation), which is crucial to neurobehavioral learning, and thus produce robust long-term after-effects (Agboada et al., 2020; Au et al., 2017). Intriguingly, the activity-dependent LTP process produced by multiple-session consolidation was found to contribute to long-term cortical plasticity, especially in the DLPFC (Jannati et al., 2023; Siebner & Rothwell, 2003). Thus, the detectable effects at 6 months are consistent with the hypothesis that repeated neuromodulation may induce neuroplastic changes in the DLPFC that support sustained behavioral change. However, we explicitly note that a single follow-up timepoint cannot establish the stability or trajectory of these effects; future studies with multiple longitudinal assessments are required to substantiate claims about long-term retention. On balance, this study provided an effective way to help procrastinators overcome actual procrastination in real-life.

Limitations

While the use of a multi-session design and the long-term assessment can be considered a strength of the present study, it also has several limitations. Even though various tDCS effects have been demonstrated so far, they also tend to be difficult to replicate and sensitive to not yet fully understood context conditions and interindividual differences, which also applies to transcranial magnetic stimulation (TMS) (Valle et al., 2009). To overcome this shortcoming, it will be necessary to establish an individually tailored tDCS protocol to improve the sensitivity of corresponding interventions (Chew et al., 2015). Thus, future research could further improve the effects of tDCS on reducing procrastination by adopting more individualized tDCS protocols. Another limitation is the lack of real-time functional neuroimaging measures to better monitor the impact of our intervention. In the absence of such measures, we had to rely on behavioral indicators to assess the success of the tDCS training. In addition to technical limitations, given the apparently limited size of the sample (total $N = 46$), it warrants caution in generalizing these findings elsewhere, and necessitates further validations in a large-scale cohort. Also, considering the lack of medical screening for psychiatric conditions (e.g., ADHD or depression) in this sample, it remains unclear whether these training effects are domainspecific for procrastination. Notably, we explicitly acknowledge that exploratory Quasi-Bayesian mediation analyses, based on observational measures rather than experimentally manipulated mediators, cannot establish causal pathways and are susceptible to inflated Type 1 error rates as demonstrated in recent simulation studies. These findings should be interpreted strictly as hypothesis-generating and statistically consistent with the proposed theoretical model, rather than as confirmatory evidence of causal mechanisms. Substantiating the precise neurocognitive pathway will require future studies employing stronger causal designs, such as experimental manipulation of task valuation or longitudinal cross-lagged modeling. Moreover, this study did not collect data for assessing participants' self-control at either baseline or post-neuromodulation. Accordingly, we explicitly note that self-control was not directly measured or manipulated in this study; the observed associations between DLPFC neuromodulation, task-outcome value, and procrastination are consistent with theoretical models positing a role for top-down regulatory processes, but do not constitute direct evidence that self-control mechanisms were engaged. This limitation precludes definitive conclusions about the unique contribution of self-control-related pathways versus alternative neurocognitive mechanisms. In addition, despite instructing to report valid real-life tasks with high probabilities to procrastinate, we did not measure the task difficulty and consistency across sessions for each participant. Consequently, interpreting the effects of neuromodulation to mitigate procrastination as "unique contributions" warrants caution. Given the absence of cathodal HD-tDCS stimulation as a contrast condition in inference, it also warrants caution that the increased DLPFC excitability may not be the exclusive neural mechanism for procrastination. Finally, while our 6-month follow-up provides preliminary evidence for sustained effects, the use of a single follow-up timepoint limits our ability to characterize the temporal

trajectory of retention. Future studies incorporating multiple followup assessments (e.g., 1-month, 3-month, 6-month, 12-month) would strengthen evidence for the robustness and durability of this intervention.

Conclusions

In conclusion, this study potentially provides an effective way to reduce both procrastination willingness and actual procrastination behavior by using neuromodulation on the left DLPFC. Furthermore, such effects have been observed for 2-day-interval long-term after-effects, and were also found for 6-month long-term retention in part. More importantly, this study identified that the ms-tDCS neuromodulation could decrease task aversiveness and increase task outcome value, and further demonstrated that the increased task outcome value could predict decreased procrastination, a relationship that is conceptually consistent with theoretical models of top-down regulation and value-based decision-making. In this vein, the current study enriches our understanding of neurocognitive mechanism of procrastination by showing the prominent role of increased task outcome value in reducing procrastination. Also, it may inform the development of theory-driven, neuromodulation-informed strategies for behavioral interventions, pending further validation in diverse populations.

Data availability

We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study, and the study follows JARS (Appelbaum, et al., 2018). Data, analysis code, and research materials are available at Science Data Bank (ScienceDB, *https://doi.org/10.57760/sciencedb.35140*). Data were analyzed using R, version 4.4.1 (R Core Team, 2020) and the package ggplot, version 3.5.2, as well as MATLAB (2021, MathWork Inc.) and GraphPad Prism.

Supplementary Information (SI) Text

SI Methods

Reconstructed preregistration statement

This study has been originally preregistered in the Open Science Framework (OSF, 10.17605/OSF.IO/Y3EDT) preregistration service on September, 2020. However, the creator's OSF account was suspended and further disabled due to an automated system flag, rendering the original HTML preregistration page inaccessible, including to the authors. To ensure the accessibility and transparency in full adherence with open science best practices, we have reconstructed the preregistration content herein, This reconstructed statement is not *post hoc*, but reflects the exact hypotheses, sample size, and analysis plan as executed (Table. S1).

Randomised participants

To randomise participants into neuromodulation and sham-control group, the full randomised block design (FRBD) was conducted here. Furthermore, it could be beneficial from FRBD that the within-group variances can be minimized. In detail, the whole sample would be portioned into k blocks, which were manipulated for high homogeneity. For each block, the pairs of participants were then randomly designated to neuromodulation and sham-control group. Thus, adopting FRBD for randomised processes can reap huge fruits to keep within-group homogeneity for each group than do of that full randomised design (1). The randomised codes came from "*www.random.org*", which produced random labels by using atmospheric.

Estimation for statistical power

To determine adequate statistical power beforehand, we capitalized on the G*Power software to estimate the minimum sample size obtaining the medium effect size (α err prob = 0.05, 1-β err prob = 0.80, f = 0.25) by building a repeated measures, within-between, and interaction effect ANOVA model (2). In this vein, the medium effect size can be reached once total sample size n = 18,

noncentrality λ = 15.75, critical F = 2.1945, numerator Df = 6, and denominator Df = 96. In addition, owing to the repeated measures for each participant, the 2-dimensional plot, so-called Power Contours estimation, was used to visualize and calculate the acceptable sample size by corresponding experimental design (3). Thus, according to the design this study made, the Power Contours estimation was done up front and indicated the total sample size of this study can attain the adequate statistical power (see Fig. S1). More detail for how to estimate and plot this map can be found elsewhere (3).

How to determine one's socioeconomic status (SES)

In line with Human Connectome Project (HCP) project proposed by National Institutes of Health (NIH), this study also acquired demographic information for each participant, including gender, age, nationality, educational level (years), family's economic status (poor, modest, middle and affluent class), family structure (FS), and status of birth (SB). In terms of criteria endorsed by the Food and Agriculture Organization (FAO) of the United Nation (UN) and the financial statement published by National Bureau of Statistics (NBS) of China in 2019 (*http://www.stats.gov.cn/tjsj/*), the economic status of the family was rated as poor, modest, middle and affluent class according to family incomes, with < ¥ 45 thousands per year for poor class, ¥ 45–65 thousands per year for modest class, ¥ 65–105 thousands per year for middle class, and > ¥ 105 for affluent class.

To determine homogeneity between the HD-tDCS group and sham-controlled group, non- / parametric and Jeffreys-Zellner-Siow Bayesian examinations were adopted to identify significant differences between demographic variable.

Semi-structural interview questionnaire

To ensure the high ecological validity, we developed a semi-structural interview questionnaire to test whether participants were eligible for the current study. There were major four profiles to investigate them, including evaluation of how they perceived pain for procrastination, whether their social functions were disrupted by procrastination, whether their attitudes are to change procrastination and whether they accepted our protocol. The outlines of this questionnaire has been provided as below:

1. Please details your identity and investigation proposal; 2. Do you perceive you have procrastination symptoms, and how severe it is do you think; 3. Are you ever assumed you have "procrastination disorder"; 4. Do you feel pain due to procrastination and how it impacts your daily life; 5. What types of procrastination are do you think in yourself; 6. Do you think you would suffer from procrastination all the life; 7. Do you want to get rid of procrastination; 8. Have you try to stop procrastination; 9. Do you know neuromodulation technique, such as tDCS or TMS; 10. Do you feel panic or worrisome for medical technique, especially in electric medical technique; 11. Do you feel panic or worrisome for electric current; 12. Do you willing to receive electric medical technique for getting rid of procrastination despite limited skin pains.

High-definition neuronavigation system

To ensure the accuracy of targeting location (left DLPFC), this neuronavigation system was used to estimate the location from skull. This system reported the MNI coordinate of what we predetermined for the first, and would re-adjust automatically twice to self-verify the estimation accuracy. Subsequently, the brain labels for these coordinates were reported by using AAL atlas and Broadman atlas, respectively. Results indicated that the F3 node overlapped the left DLPFC with 94.33% probability, which was cross-checked automatically.

Temporal difference-to-difference model

In the current study, in addition to the estimation for the model including all the repeated measures, we still aimed to do a random-sham pre-post tests for clarifying whether this HD-tDCS by using multiple sessions protocol was effective. In this vein, the difference coefficient (Δ) was computed as difference between post-test after last session and pre-test before first session, one-by-one for participants. In this vein, the pre-post within-participant differences upon outcome variables can be estimated as the HD-tDCS training effect.

SI Results

Unexpected event effects

In the Session 1, a significant unexpected life event effect occurred by this fact that almost all the participants in both group do not procrastinate (Neuromodulation group, NM, Procrastination rate (PR): 4.34 % (1/23), Sham control group, SC, PR: 8.69 % (2/23)). In the follow-up investigation, all the participants reported a strong expectation for this neuromodulation as they received such treatment for the first time. In the Session 4, the PR was observed to be increased dramatically in both groups (Neuromodulation group, NM, Procrastination rate (PR): 86.95 % (20/23), Sham control group, SC, PR: 86.95 % (20/23)). In the follow-up investigation, all the participants reported that taking action immediately to complete task was hampered significantly due to weekend effect (10, 11). In this vein, data for these sessions were excluded in the analysis. Here, the follow-up investigation required participants to report whether their performance for completing task was influenced by additional effects. If in this case, they should report what unexpected events occur here. The x^2 test was used to examine whether those reported unexpected events confounding task execution posed significantly differences between groups across all the sessions (see Table. S2).

Robustness check for interaction effects

To examine whether Group*Neuromodulation_Session interactions are statistically robust, we reanalyzed interaction effects by removing data from session #6, #7 and both, respectively in which those sessions showed extraordinary high effectiveness of neuromodulation (i.e., outliers). The findings derived from those nested models showed that all the interaction effects are all retained, indicating a high statistical robustness (Table. S3–4).

Results of linear probability panel model

Given the panel data pattern (7 longitudinal sessions × task-execution willingness), this study has drawn upon the linear probability panel model (PLM) fitting the task aversiveness and outcome value to task-execution willingness. Specifically, the Fisher's Augmented Dickey-Fuller Test (ADF) tests were performed to examine whether these data are suitable for this model. Results indicated the stationary properties for all the variables (task aversiveness, DF = -5.78, $p < .05$; outcome value, DF = -5.83, $p < .05$; task-execution willingness, DF = -5.83, $p < .01$). Further, the findings derived from Breusch-Godfrey/Wooldridge test maintained the decision to reject the stability of pool model ($X^2 = 22.75$, $p < .0001$). Also, the Hausman test was done for determining what types of models would be accepted. Results indicated that the random effect model is unstability ($X^2 = 12.92$, $p < .0015$). Thus, the fixed effect model controlling both time and individual variants was adopted as final one.

Results of LMM adjusted for baseline

To minimize false-positive risks, rather to take time and group as predictors meantime, we reanalyzed these data by remodeling post-neuromodulation procrastination as dependent and remodeling pre-neuromodulation procrastination, group and other covariates as predictors. Results showed the significantly predictive of pre-neuromodulation procrastination willingness ($\beta = 24.48$, SE = 6.24, $p = .0018$) and procrastination rate (PR, $\beta = 30.66$, SE = 8.48, $p = .0015$) to post-neuromodulation ones. Moreover, for those covariates of interests on cognitive mechanisms, these findings are validated as well, showing statistically significant predictions from preneuromodulation cognitive processes to post-neuromodulation ones (task aversiveness, $\beta = 0.58$, SE = 0.23, $p = .018$; outcome value, $\beta = 22.86$, SE = 5.11, $p = .0005$). This replicated effect has been observed in predicting post-neuromodulation PR by pre-neuromodulation one for 6 month ($\beta = 34.79$, SE = 11.1, $p = .005$). Taken together, using stringent statistical constrain to adjust baseline, we revealed the same findings compared to traditional LMM.

Results of sensitivity analysis to LMM

To assess whether the primary findings were sensitive to the Gaussian distribution assumption, we re-analyzed the main models using Beta regression with a logit link function, which is appropriate for bounded continuous outcomes . Models were fitted using the glmmTMB package (Brooks et al., 2017) in R, retaining the same fixed and random effects structure as the primary models: (1 + day_c + Emotions_c || SubjectID).

The results of the Beta regression models corroborated the primary findings. For procrastination rate, the Group × day_c interaction was significant (b = -0.182, SE = 0.091, z = -2.01, p = .045). Fortask-execution willingness, the Group × day_c interaction was also significant (b = -0.375, SE = 0.086, z = -4.35, p < .001). The main effects of Group and day_c remained significant across both outcomes (all p < .001). Complete results are presented in Table S5.

Results of sensitivity analysis to mediation model

To examine the robustness and specificity of this mediation model, we conducted several sensitive analyses. Thus, the we attempted to re-do this mediation model by replacing the sampling method from bootstrap-based bias-corrected and accelerated (BCa) intervals to “bca” sampling at 5000 simulations. Results demonstrated the robustness of this model by showing same findings (see Table. S6–7).

Further, we inputted the age and gender as outcome variables into this model for testing whether this mediation model is specific to predict decreased procrastination. As hypothesized, no significant effects were found to predict task aversiveness by using this model, and thus supported the specificity of these findings (see Table. S8).

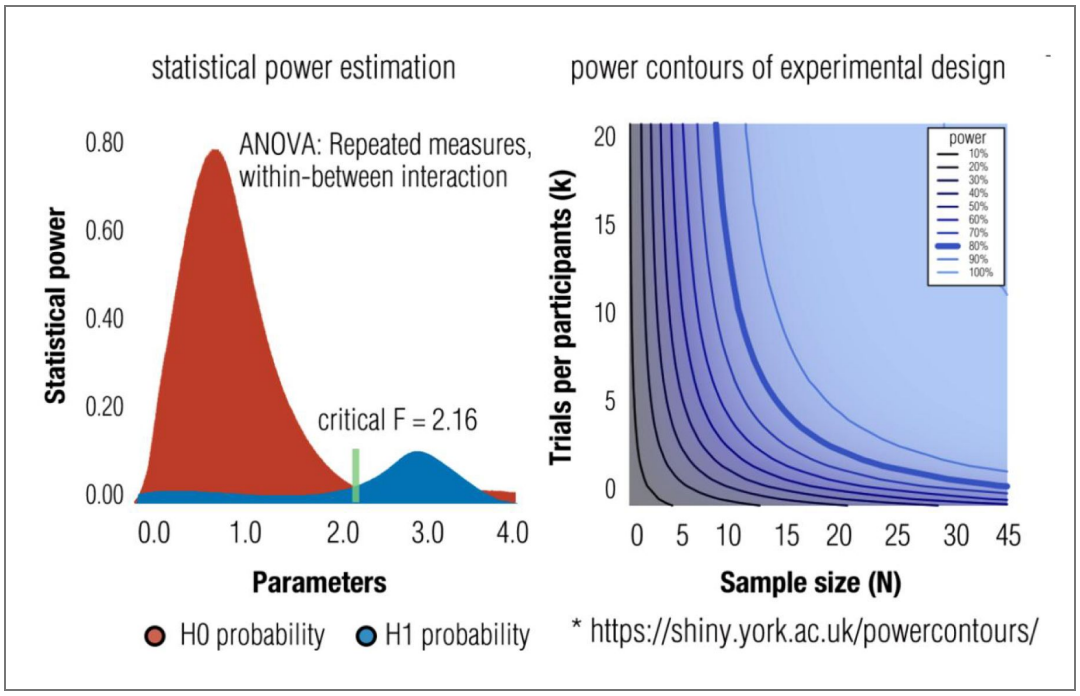


Figure S1. **Statistical power estimation by using ANOVA model and power contours according to experimental design**

Table S1. Reconstructed preregistration statement table.

Question	Hypothesis	Sampling plan (e.g. power analysis)	Analysis Plan	Interpretation given to different outcomes
Whether the multi-session HD-tDCS over the left DLPFC could reduce real-world procrastination?	This HD-tDCS neuromodulation could increasingly mitigate real-world procrastination	We capitalize on the G*Power software to estimate the minimum sample size obtaining the medium effect size (α err prob = 0.05, 1-β err prob = 0.80, f = 0.25) by building a repeated measures, within-between, and interaction effect ANOVA model (2). In this vein, the medium effect size can be reached once total sample size n = 18, noncentrality λ = 15.75, critical F = 2.1945, numerator Df = 6, and denominator Df = 96. In addition, owing to the repeated measures for each participant, the 2-dimensional plot, so-called Power Contours estimation, would be used to visualize and calculate the acceptable sample size by corresponding experimental design (3)	(a) The generalized mixed-effect linear model (GLMM) would be constructed using 2 (active vs. sham) × 2 (before first neuromodulation vs. after last neuromodulation) full factorial design for procrastination willingness (i.e., selfreported scores) and actual procrastination rate (1- self-reported task completion rate); (b) Markov Chain Monte Carlo Generalised linear mixed effects model (MCMCglmm) would be built to reestimate results that probed above for validation; (c) Temporal difference-to-difference model would be used to obtain the difference coefficient (Δ) individually, and independent t-tests with Jeffreys-Zellner-Siow Bayesian examinations for between-group differences upon procrastination willingness (i.e., selfreported scores) and actual procrastination rate (1 - self-reported - task completion rate) would be conducted; (d) Between-group comparison for the counts of reporting task procrastination across participants are conducted by X^2 test with ϕ correction or Fisher exact test would be carried out; (e) The joint model of longitudinal and survival data (JM-LAD), in conjunction of machine learning algorithm, was adopted to capture multi-session effects individually	-

Whether the multi-session HD-tDCS over the left DLPFC could reduce task aversiveness and increase outcome value?	Multi-session HD-tDCS could decrease task aversiveness and increase outcome values meanwhile	See above	The generalized mixed-effect linear model (GLMM) would be constructed using 2 (active vs. sham) × 2 (before first neuromodulation vs. after last neuromodulation) full factorial design for task aversiveness (i.e., AUC of five task time points) and outcome value (i.e., AUC of five task time points)
Why multisession HD-tDCS could ameliorate procrastination willingness and real-world procrastination behavior?	(a) Multi-session HD-tDCS could increase procrastination willingness and decrease real-world procrastination rate by undermining task aversiveness; (b) Multi-session HD-tDCS could increase procrastination willingness and decrease real-world procrastination rate by increasing outcome value;	No suitable method to make this sample size estimation	(a) Generalized linear model to correlate $\Delta_{\text{Task aversiveness}}$ and $\Delta_{\text{Outcome value}}$ to $\Delta_{\text{Procrastination willingness}}$; (b)Generalized linear model to correlate $\Delta_{\text{Task aversiveness}}$ and $\Delta_{\text{Outcome value}}$ to $\Delta_{\text{Real-world Procrastination}}$; (c)Quasi-Bayesian causal mediation analysis to model the task aversiveness as the mediator for interpreting association of HD-tDCS intervention to changes of procrastination willingness and real-world procrastination; (d) Quasi-Bayesian causal mediation analysis to model the task aversiveness as the mediator for interpreting association of HD-tDCS intervention to changes of procrastination willingness and real-world procrastination; (e) Quasi-Bayesian causal mediation analysis to model the outcome value as the mediator for interpreting association of HD-tDCS intervention to changes of procrastination willingness and real-world procrastination;

Does this multisession HD-tDCS neuromodulation has lasting effect?	This effect can be tested in the 6-month followup	(a) The repeated-measure ANOVA model would be built to test real-world procrastination rates across three time points (Pre-, Post-, and 6-month followup test); (b) Jeffreys-Zellner-Siow Bayesian factor model would be used to validate above results derived from repeated-measure ANOVA	-

Table S2. The number of reporting unexpected event effects in both group across all the session.

Here, we conducted a post-neuromodulation investigation to require participants to report whether their performance for completing task was influenced by additional impacts outside normal conditions, such as "get a flu", "get a fever", "mandatory assignment for other tasks" and " unexpected emergency events". If in this case, they should report what unexpected events occur to uncontrollably disrupt task execution. In the S1, all the 44 participants reported expected effects from the neuromodulation. In the S1, one participant reported to be in flu. In the S3, both participants in NM group reported a mandatory meeting assignment, whist one participant in the SC reported a bicycle accident and additional two reported mandatory meeting assignment. In the S4, all the 39 participants reported that their task performances are fully disrupted by the weekends. S5–7 reported the similar additional AE mentioned previously.

	S0	S1	S2	S3	S4	S5	S6	S7
NM	0/23	21/23	1/23	2/23	20/23	0/23	3/23	5/23
SC	0/23	23/23	0/23	3/23	19/23	1/23	0/23	3/23
X^2	-	.023	-	.200	.026	-	-	.500

Table S3. LMM to robustness check with outcome as subjective procrastination willingness.

Model	*β* (group*treatment_day)	Std. Error (SE)	*T* value	*P* value
Full model	-7.78	1.79	-4.35	< .001
Removing Session #7	-9.78	2.25	-4.34	< .001
Removing Session #6	-7.27	1.83	-3.98	< .001
Removing Session #6 and #7 both	-10.02	3.38	-2.96	.005

Table S4. LMM to robustness check with outcome as real-world procrastination rates.

Model	*β* (group*treatment_day)	Std. Error (SE)	*T* value	*P* value
Full model	-7.38	2.45	-3.02	.004
Removing Session #7	-10.34	3.16	-3.27	.002
Removing Session #6	-6.55	2.53	-2.59	.011
Removing Session #6 and #7 both	-11.07	4.36	-2.54	.013

Table S5. Sensitivity Analysis Results Using Beta Regression.

DV	Effect	*b*	SE	*z*	*p*	Consistency with Gaussian model
Actual procrastination rate	Group	-0.760	0.202	-3.77	< .001	Yes (p < .001)
	Treatment Day	0.225	0.064	3.50	< .001	Yes (p < .001)
	Group × Day interaction	-0.182	0.091	-2.01	.045	Yes (p = .004)

Task-execution Willingness	Group	-1.081	0.282	-3.83	< .001	Yes (p < .001)
	Treatment Day	0.446	0.064	7.00	< .001	Yes (p < .001)
	Group × Day interaction	-0.375	0.086	-4.35	< .001	Yes (p < .001)

Table S6. Summary for mediation model in predicting task-execution willingness by using treatment (active ms-tDCS v.s. sham) from mediated effect of increased task outcome.

	Estimate	95% Lower	95% Upper	P value
ACME	21.95 ***	10.67	34.90	.0001
ADE	11.14	-2.20	25.10	.10
Total Effect	33.10***	19.00	47.80	.0001

Table S7. Summary for mediation model in predicting actual procrastination by using treatment (active ms-tDCS v.s. sham) from mediated effect of increased task outcome.

	Estimate	95% Lower	95% Upper	P value
ACME	30.91 **	11.93	49.63	.002
ADE	2.77	-7.12	13.38	.61
Total Effect	33.68**	18.10	49.74	.002

Table S8. Summary for mediation model in predicting task aversiveness by using treatment (active ms-tDCS v.s. sham) from mediated effect of increased task outcome.

	Estimate	95% Lower	95% Upper	P value
ACME	0	0.00	0.00	1.000
ADE	25.09 **	8.29	42.00	0.0036
Total Effect	25.09 **	8.29	42.00	0.0036

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Author Contributions

Zhiyi Chen: Conceptualization, Methodology, Software, Writing - Original Draft and Visualization; ZhenZhen Huo, Chenyan Zhang, Bernhard Hommel: Writing - Original Draft, Formal analysis and Validation; ZhuangZheng Wang, Ye Liu, Bowen Hu, Wanting Chen: Writing - Review & Editing, Methodology, or Validation; Tingyong Feng, Conceptualization, Supervision, Project administration and Funding acquisition. CZY, RZL and LW contributed equally to this work.

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References

Agboada D, Mosayebi-Samani M, Kuo MF, Nitsche MA (2020) Induction of longterm potentiation-like plasticity in the primary motor cortex with repeated anodal transcranial direct current stimulation - Better effects with intensified protocols?. *Brain Stimul* **13**:987-997 https://doi.org/10.1016/j.brs.2020.04.009 | PubMed

Allenby C, Falcone M, Bernardo L, Wileyto EP, Rostain A, Ramsay JR, Lerman C, Loughead J (2018) Transcranial direct current brain stimulation decreases impulsivity in ADHD. *Brain Stimul* **11**:974-981 https://doi.org/10.1016/j.brs.2018.04.016 | PubMed

American College Health Association (2022) Undergraduate student reference group executive summary. https://www.acha.org/wp-content/uploads/2024/07/NCHA-IIISPRING2022UNDERGRADREFERENCEGROUPEXECUTIVESUMMARY.pdf

Ariely D, Wertenbroch K (2002) Procrastination, deadlines, and performance: selfcontrol by precommitment. *Psychol Sci* **13**:219-224 https://doi.org/10.1111/1467-9280.00441 | PubMed

Au J, Karsten C, Buschkuehl M, Jaeggi SM (2017) Optimizing Transcranial Direct Current Stimulation Protocols to Promote Long-Term Learning. *Journal of Cognitive Enhancement* **1**:65-72 https://doi.org/10.1007/s41465-017-0007-6

Bikson M, Grossman P, Thomas C, Zannou AL, Jiang J, Adnan T, Mourdoukoutas AP, Kronberg G, Truong D, Boggio P, *et al.* (2016) Safety of Transcranial Direct Current Stimulation: Evidence Based Update 2016. *Brain Stimul* **9**:641-661 https://doi.org/10.1016/j.brs.2016.06.004 | PubMed

Blake A (2019) The Procrastination Equation: How to Stop Putting Things Off and Start Getting Stuff Done PiersSteel. *Performance Improvement* **58**:34-37 https://doi.org/10.1002/pfi.21314

Blunt AK, Pychyl TA (2000) Task aversiveness and procrastination: a multidimensional approach to task aversiveness across stages of personal projects. *Personality and Individual Differences* **28**:153-167 https://doi.org/10.1016/S0191-8869(99)00091-4

Brunoni AR, Boggio PS, Ferrucci R, Priori A, Fregni F (2013) Transcranial direct current stimulation: challenges, opportunities, and impact on psychiatry and neurorehabilitation. *Frontiers in Psychiatry* **4**:19 https://doi.org/10.3389/fpsyt.2013.00019 | PubMed

Brunoni AR, Nitsche MA, Bolognini N, Bikson M, Wagner T, Merabet L, Edwards DJ, Valero-Cabre A, Rotenberg A, Pascual-Leone A, *et al.* (2012) Clinical research with transcranial direct current stimulation (tDCS): challenges and future directions. *Brain Stimul* **5**:175-195 https://doi.org/10.1016/j.brs.2011.03.002 | PubMed

Chen F, He Q, Han Y, Zhang Y, Gao X (2018) Increased BOLD Signals in dlPFC Is Associated With Stronger Self-Control in Food-Related Decision-Making. *Frontiers in Psychiatry* **9**:689 https://doi.org/10.3389/fpsyt.2018.00689 | PubMed

Chen Z, Feng T (2022) Neural connectome features of procrastination: Current progress and future direction. *Brain and Cognition* **161**:105882 https://doi.org/10.1016/j.bandc.2022.105882 | PubMed

Chen Z, Liu P, Zhang C, Feng T (2020) Brain Morphological Dynamics of Procrastination: The Crucial Role of the Self-Control, Emotional, and Episodic Prospection Network. *Cerebral Cortex* **30**:2834-2853 https://doi.org/10.1093/cercor/bhz278 | PubMed

Chen Z, Liu P, Zhang C, Yu Z, Feng T (2021) Neural markers of procrastination in white matter microstructures and networks. *Psychophysiology* **58**:e13782 https://doi.org/10.1111/psyp.13782 | PubMed

Chen Z, Zhang R, Xie J, Liu P, Zhang C, Zhao J, Laplante JP, Feng T (2022) Hybrid brain model accurately predict human procrastination behavior. *Cognitive Neurodynamics* **16**:1107-1121 https://doi.org/10.1007/s11571-021-09765-z | PubMed

Chew T, Ho K.-A, Loo CK (2015) Inter- and Intra-individual Variability in Response to Transcranial Direct Current Stimulation (tDCS) at Varying Current Intensities. *Brain Stimul* **8**:1130-1137 https://doi.org/10.1016/j.brs.2015.07.031 | PubMed

Cho SS, Koshimori Y, Aminian K, Obeso I, Rusjan P, Lang AE, Daskalakis ZJ, Houle S, Strafella AP (2015) Investing in the future: stimulation of the medial prefrontal cortex reduces discounting of delayed rewards. *Neuropsychopharmacology* **40**:546-553 https://doi.org/10.1038/npp.2014.211 | PubMed

Chrysikou EG, Wing EK, van Dam WO (2022) Transcranial Direct Current Stimulation Over the Prefrontal Cortex in Depression Modulates Cortical Excitability in Emotion Regulation Regions as Measured by Concurrent Functional Magnetic Resonance Imaging: An Exploratory Study. *Biological psychiatry-Cognitive neuroscience and neuroimaging* **7**:85-94 https://doi.org/10.1016/j.bpsc.2019.12.004 | PubMed

Cirillo G, Di Pino G, Capone F, Ranieri F, Florio L, Todisco V, Tedeschi G, Funke K, Di Lazzaro V (2017) Neurobiological after-effects of non-invasive brain stimulation. *Brain Stimul* **10**:1-18 https://doi.org/10.1016/j.brs.2016.11.009 | PubMed

Cohen JR, Lieberman MD (2010) The common neural basis of exerting self-control in multiple domains. In: Hassin R, Ochsner K, Trope Y (Eds). *Self Control in Society, Mind, and Brain, Social Cognition and Social Neuroscience* New York: Oxford Academic. https://doi.org/10.1093/acprof:oso/9780195391381.003.0008

Eckert M, Ebert DD, Lehr D, Sieland B, Berking M (2016) Overcome procrastination: Enhancing emotion regulation skills reduce procrastination. *Learning and Individual Differences* **52**:10-18 https://doi.org/10.1016/j.lindif.2016.10.001

Ferrari JR (1994) Dysfunctional procrastination and its relationship with self-esteem, interpersonal dependency, and self-defeating behaviors. *Personality and Individual Differences* **17**:673-679 https://doi.org/10.1016/0191-8869(94)90140-6

Ferrari JR, O'Callaghan J, Newbegin I (2005) Prevalence of procrastination in the United States, United Kingdom, and Australia: arousal and avoidance delays among adults. *North American Journal of Psychology* **7**:1-6

Frost R, McNaughton N (2017) The neural basis of delay discounting: A review and preliminary model. *Neuroscience and Biobehavioral Reviews* **79**:48-65 https://doi.org/10.1016/j.neubiorev.2017.04.022 | PubMed

Green L, Myerson J (2004) A discounting framework for choice with delayed and probabilistic rewards. *Psychological Bulletin* **130**:769-792 https://doi.org/10.1037/0033-2909.130.5.769 | PubMed

Grunschel C, Schopenhauer L (2015) Why are students (not) motivated to change academic procrastination?: An investigation based on the transtheoretical model of change. *Journal of College Student Development* **56**:187-200 https://doi.org/10.1353/csd.2015.0012

Gustavson DE, du Pont A, Hatoum AS, Hyun Rhee S, Kremen WS, Hewitt JK, Friedman NP (2017) Genetic and Environmental Associations Between Procrastination and Internalizing/Externalizing Psychopathology. *Clinical psychological science* **5**:798-815 https://doi.org/10.1177/2167702617706084 | PubMed

Gustavson DE, Miyake A, Hewitt JK, Friedman NP (2014) Genetic relations among procrastination, impulsivity, and goal-management ability: implications for the evolutionary origin of procrastination. *Psychol Sci* **25**:1178-1188 https://doi.org/10.1177/0956797614526260 | PubMed

Gustavson DE, Miyake A, Hewitt JK, Friedman NP (2015) Understanding the cognitive and genetic underpinnings of procrastination: Evidence for shared genetic influences with goal management and executive function abilities. *Journal of experimental psychology* **144**:1063-1079 https://doi.org/10.1037/xge0000110 | PubMed

Hairston IS, Shpitalni R (2016) Procrastination is linked with insomnia symptoms: The moderating role of morningness-eveningness. *Personality and Individual Differences* **101**:50-56 https://doi.org/10.1016/j.paid.2016.05.031

Hare TA, Camerer CF, Rangel A (2009) (5927) Self-control in decision-making involves modulation of the vmPFC valuation system. *Science* **324**:646-648 https://doi.org/10.1126/science.1168450 | PubMed

Harriott J, Ferrari JR (1996) Prevalence of procrastination among samples of adults. *Psychol Rep* **78**:611-616 https://doi.org/10.2466/pr0.1996.78.2.611

Harris A, Hare T, Rangel A (2013) Temporally dissociable mechanisms of self-control: early attentional filtering versus late value modulation. *The Journal of neuroscience* **33**:18917-18931 https://doi.org/10.1523/jneurosci.5816-12.2013 | PubMed

Hu Y, Liu P, Guo Y, Feng T (2018) The neural substrates of procrastination: A voxelbased morphometry study. *Brain and Cognition* **121**:11-16 https://doi.org/10.1016/j.bandc.2018.01.001 | PubMed

Huang D, Chen S, Wang S, Shi J, Ye H, Luo J, Zheng H (2017) Activation of the DLPFC Reveals an Asymmetric Effect in Risky Decision Making: Evidence from a tDCS Study. *Frontiers in Psychology* **8**:38 https://doi.org/10.3389/fpsyg.2017.00038 | PubMed

Huang J, Chen L, Zhao H, Xu T, Xiong Z, Yang C, Feng T, Feng P (2025) The functional connectivity between the right rostral anterior cingulate cortex and the right dorsolateral prefrontal cortex underlies the association between future self-continuity and self-control. *Cereb Cortex* **35** https://doi.org/10.1093/cercor/bhaf092 | PubMed

Imai K, Keele L, Tingley D, Yamamoto T (2010) (2010) *Causal Mediation Analysis Using R. Advances in Social Science Research Using R* New York, NY.

Jannati A, Oberman LM, Rotenberg A, Pascual-Leone A (2023) Assessing the mechanisms of brain plasticity by transcranial magnetic stimulation. *Neuropsychopharmacology* **48**:191-208 https://doi.org/10.1038/s41386-022-01453-8 | PubMed

Jimura K, Chushak MS, Braver TS (2013) Impulsivity and self-control during intertemporal decision making linked to the neural dynamics of reward value representation. *The Journal of neuroscience* **33**:344-357 https://doi.org/10.1523/jneurosci.0919-12.2013 | PubMed

Johansson F, Rozental A, Edlund K, Côté P, Sundberg T, Onell C, Rudman A, Skillgate E (2023) Associations Between Procrastination and Subsequent Health Outcomes Among University Students in Sweden. *JAMA Network Open* **6**:e2249346 https://doi.org/10.1001/jamanetworkopen.2022.49346 | PubMed

Kelley NJ, Gallucci A, Riva P, Romero Lauro LJ, Schmeichel BJ (2018) Stimulating Self-Regulation: A Review of Non-invasive Brain Stimulation Studies of Goal-Directed Behavior. *Frontiers in Behavioral Neuroscience* **12**:337 https://doi.org/10.3389/fnbeh.2018.00337 | PubMed

Klein E, Beutel M, Müller K, Wölfling K, Brähler E, Zenger M (2019) Dimensionality and measurement invariance of the General Procrastination Scale-Screening (GPS-S) in a representative sample. *European Journal of Psychological Assessment* **35**:633-640 https://doi.org/10.1027/1015-5759/a000441

Knoch D, Fehr E (2007) Resisting the power of temptations: the right prefrontal cortex and self-control. *Annals of the New York Academy of Sciences* **1104**:123-134 https://doi.org/10.1196/annals.1390.004 | PubMed

Lay CH (1986) At last, my research article on procrastination. *Journal of Research in Personality* **20**:474-495 https://doi.org/10.1016/0092-6566(86)90127-3

Le Bouc R, Pessiglione M (2022) A neuro-computational account of procrastination behavior. *Nature Communications* **13**:5639 https://doi.org/10.1038/s41467-022-33119-w | PubMed

Lin Y, Feng T (2024) Lateralization of self-control over the dorsolateral prefrontal cortex in decision-making: a systematic review and meta-analytic evidence from noninvasive brain stimulation. *Cogn Affect Behav Neurosci* **24**:19-41 https://doi.org/10.3758/s13415-023-01148-7 | PubMed

Liu P, Feng T (2017) The overlapping brain region accounting for the relationship between procrastination and impulsivity: A voxel-based morphometry study. *Neuroscience* **360**:9-17 https://doi.org/10.1016/j.neuroscience.2017.07.042 | PubMed

Miao M, Chen Y, Zhou Z, Wen J, Zheng L (2024) Procrastination in the IntentionBehaviour gap: Exercise procrastination and the moderating role of emotion. *Psychol SportExerc* **74**:102672 https://doi.org/10.1016/j.psychsport.2024.102672 | PubMed

Minati L, Campanhã C, Critchley HD, Boggio PS (2012) Effects of transcranial direct-current stimulation (tDCS) of the dorsolateral prefrontal cortex (DLPFC) during a mixed-gambling risky decision-making task. *Cognitive Neuroscience* **3**:80-88 https://doi.org/10.1080/17588928.2011.628382 | PubMed

Myerson J, Green L, Warusawitharana M (2001) Area under the curve as a measure of discounting. *J Exp Anal Behav* **76**:235-243 https://doi.org/10.1901/jeab.2001.76-235 | PubMed

Paschke LM, Dörfel D, Steimke R, Trempler I, Magrabi A, Ludwig VU, Schubert T, Stelzel C, Walter H (2016) Individual differences in self-reported self-control predict successful emotion regulation. *Soc Cogn Affect Neurosci* **11**:1193-1204 https://doi.org/10.1093/scan/nsw036 | PubMed

Pychyl TA, Sirois FM (2016) Procrastination, Emotion Regulation, and Well-Being. In: Sirois FM, Pychyl TA (Eds). *Procrastination, Health, and Well-Being* Academic Press. pp. 163-188 https://doi.org/10.1016/B978-0-12-802862-9.00008-6

Ramzi F, Saed O (2019) The roles of self-regulation and self-control in procrastination. *Psychology and Behavioral Science International Journal* **13**:555863 https://doi.org/10.19080/PBSIJ.2019.13.555863

Rebetez MML, Barsics C, Rochat L, D'Argembeau A, Van der Linden M (2016) Procrastination, consideration of future consequences, and episodic future thinking. *Conscious Cogn* **42**:286-292 https://doi.org/10.1016/j.concog.2016.04.003 | PubMed

Rebetez MML, Rochat L, Barsics C, Van der Linden M (2018) Procrastination as a Self-Regulation Failure: The Role of Impulsivity and Intrusive Thoughts. *Psychol Rep* **121**:26-41 https://doi.org/10.1177/0033294117720695 | PubMed

Rozental A, Bennett S, Forsström D, Ebert DD, Shafran R, Andersson G, Carlbring P (2018) Targeting Procrastination Using Psychological Treatments: A Systematic Review and Meta-Analysis. *Frontiers in Psychology* **9**:1588 https://doi.org/10.3389/fpsyg.2018.01588

Rozental A, Carlbring P (2014) Understanding and treating procrastination: A review of a common self-regulatory failure. *Psychology* **5**:1488 https://doi.org/10.4236/psych.2014.513160

Sabé M, Hyde J, Cramer C, Eberhard A, Crippa A, Brunoni AR, Aleman A, Kaiser S, Baldwin DS, Garner M, *et al.* (2024) Transcranial Magnetic Stimulation and Transcranial Direct Current Stimulation Across Mental Disorders: A Systematic Review and DoseResponse Meta-Analysis. *JAMA Network Open* **7**:e2412616 https://doi.org/10.1001/jamanetworkopen.2024.12616 | PubMed

Schlüter C, Fraenz C, Pinnow M, Friedrich P, Güntürkün O, Genç E (2018) The Structural and Functional Signature of Action Control. *Psychol Sci* **29**:1620-1630 https://doi.org/10.1177/0956797618779380 | PubMed

Seibt O, Brunoni AR, Huang Y, Bikson M (2015) The Pursuit of DLPFC: Nonneuronavigated Methods to Target the Left Dorsolateral Pre-frontal Cortex With Symmetric Bicephalic Transcranial Direct Current Stimulation (tDCS). *Brain Stimul* **8**:590-602 https://doi.org/10.1016/j.brs.2015.01.401 | PubMed

Shin DW, Fan J, Luu E, Khalid W, Xia Y, Khadka N, Bikson M, Fu BM (2020) In Vivo Modulation of the Blood-Brain Barrier Permeability by Transcranial Direct Current Stimulation (tDCS). *Annals of Biomedical Engineering* **48**:1256-1270 https://doi.org/10.1007/s10439-020-02447-7 | PubMed

Siebner HR, Rothwell J (2003) Transcranial magnetic stimulation: new insights into representational cortical plasticity. *Experimental Brain Research* **148**:1-16 https://doi.org/10.1007/s00221-002-1234-2 | PubMed

Sirois F, Pychyl T (2013) Procrastination and the Priority of Short-Term Mood Regulation: Consequences for Future Self. *Social and Personality Psychology Compass* **7**:115-127 https://doi.org/10.1111/spc3.12011

Sirois FM (2015) Is procrastination a vulnerability factor for hypertension and cardiovascular disease? Testing an extension of the procrastination-health model. *Journal of Behavioral Medicine* **38**:578-589 https://doi.org/10.1007/s10865-015-9629-2 | PubMed

Sirois FM (2016) Procrastination, Stress, and Chronic Health Conditions: A Temporal Perspective. In: Sirois FM, Pychyl TA (Eds). *Procrastination, Health, and WellBeing* Academic Press. pp. 67-92 https://doi.org/10.1016/B978-0-12-802862-9.00004-9

Smith BJ, Monterosso JR, Wakslak CJ, Bechara A, Read SJ (2018) A meta-analytical review of brain activity associated with intertemporal decisions: Evidence for an anterior-posterior tangibility axis. *Neuroscience and Biobehavioral Reviews* **86**:85-98 https://doi.org/10.1016/j.neubiorev.2018.01.005 | PubMed

Soleimani G, Kupliki R, Paulus M, Ekhtiari H (2023) Dose-response in modulating brain function with transcranial direct current stimulation: From local to network levels. *PLoS Computational Biology* **19**:e1011572 https://doi.org/10.1371/journal.pcbi.1011572 | PubMed

Steel P (2007) The nature of procrastination: a meta-analytic and theoretical review of quintessential self-regulatory failure. *Psychological Bulletin* **133**:65-94 https://doi.org/10.1037/0033-2909.133.1.65 | PubMed

Steel P, König CJ (2006) Integrating Theories of Motivation. *Academy of Management Review* **31**:889-913 https://doi.org/10.5465/amr.2006.22527462

Steel P, Taras D, Ponak A, Kammeyer-Mueller J (2021) Self-Regulation of Slippery Deadlines: The Role of Procrastination in Work Performance. *Frontiers in Psychology* **12**:783789 https://doi.org/10.3389/fpsyg.2021.783789 | PubMed

Taura AA, Abdullah MC, Roslan S, Omar Z (2015) Relationship between self-efficacy, task value, self-regulation strategies and active procrastination among pre-service teachers in colleges of education. *International Journal of Psychology and Counselling* **7**:11-17

Tedesco Triccas L, Burridge JH, Hughes AM, Pickering RM, Desikan M, Rothwell JC, Verheyden G (2016) Multiple sessions of transcranial direct current stimulation and upper extremity rehabilitation in stroke: A review and meta-analysis. *Clinical Neurophysiology* **127**:946-955 https://doi.org/10.1016/j.clinph.2015.04.067 | PubMed

Terracciano A, McCrae RR, Costa PT (2003) Factorial and construct validity of the Italian Positive and Negative Affect Schedule (PANAS). *European Journal of Psychological Assessment* **19**:131-141 https://doi.org/10.1027//1015-5759.19.2.131 | PubMed

Tice DM, Bratslavsky E (2000) Giving in to feel good: The place of emotion regulation in the context of general self-control. *Psychological inquiry* **11**:149-159 https://doi.org/10.1207/s15327965pli1103_03

Tsukuda B, Ikeda S, Minami S, Katsura K, Shimizu T, Kame T, Kojima Y, Nishida K, Yoshimura M, Kato M (2025) Comparison of the left DLPFC identified by the 5 cm rule, F3, and neuronavigation methods, and examination of the relationship between stimulation site and treatment effect of rTMS for depression. *Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation* **18**:536 https://doi.org/10.1016/j.brs.2024.12.937

Valle A, Roizenblatt S, Botte S, Zaghi S, Riberto M, Tufik S, Boggio PS, Fregni F (2009) Efficacy of anodal transcranial direct current stimulation (tDCS) for the treatment of fibromyalgia: results of a randomized, sham-controlled longitudinal clinical trial. *J Pain Manag* **2**:353-361 PubMed

van Eerde W, Klingsieck KB (2018) Overcoming procrastination? A meta-analysis of intervention studies. *Educational Research Review* **25**:73-85 https://doi.org/10.1016/j.edurev.2018.09.002

van Hooft EAJ, Born MP, Taris TW, van der Flier H, Blonk RWB (2005) Bridging the gap between intentions and behavior: Implementation intentions, action control, and procrastination. *Journal of Vocational Behavior* **66**:238-256 https://doi.org/10.1016/j.jvb.2004.10.003

Villamar MF, Volz MS, Bikson M, Datta A, Dasilva AF, Fregni F (2013) Technique and considerations in the use of 4x1 ring high-definition transcranial direct current stimulation (HD-tDCS). *Journal of Visualized Experiments* e50309 https://doi.org/10.3791/50309 | PubMed

Woodham RD, Selvaraj S, Lajmi N, Hobday H, Sheehan G, Ghazi-Noori A.-R, Lagerberg PJ, Rizvi M, Kwon SS, Orhii P, *et al.* (2025) Home-based transcranial direct current stimulation treatment for major depressive disorder: a fully remote phase 2 randomized sham-controlled trial. *Nature Medicine* **31**:87-95 https://doi.org/10.1038/s41591-024-03305-y | PubMed

Woods AJ, Antal A, Bikson M, Boggio PS, Brunoni AR, Celnik P, Cohen LG, Fregni F, Herrmann CS, Kappenman ES, *et al.* (2016) A technical guide to tDCS, and related non-invasive brain stimulation tools. *Clinical Neurophysiology* **127**:1031-1048 https://doi.org/10.1016/j.clinph.2015.11.012 | PubMed

Wu H, Gui D, Lin W, Gu R, Zhu X, Liu X (2016) The procrastinators want it now: Behavioral and event-related potential evidence of the procrastination of intertemporal choices. *Brain and Cognition* **107**:16-23 https://doi.org/10.1016/j.bandc.2016.06.005 | PubMed

Wu Y, Li L, Yuan B, Tian X (2016) Individual differences in resting-state functional connectivity predict procrastination. *Personality and Individual Differences* **95**:62-67 https://doi.org/10.1016/j.paid.2016.02.016

Wypych M, Michałowski JM, Droździel D, Borczykowska M, Szczepanik M, Marchewka A (2019) Attenuated brain activity during error processing and punishment anticipation in procrastination - a monetary Go/No-go fMRI study. *Scientific Reports* **9**:11492 https://doi.org/10.1038/s41598-019-48008-4 | PubMed

Xu T, Sirois FM, Zhang L, Yu Z, Feng T (2021) Neural basis responsible for selfcontrol association with procrastination: Right MFC and bilateral OFC functional connectivity with left dlPFC. *Journal of Research in Personality* **91**:104064 https://doi.org/10.1016/j.jrp.2021.104064

Xu T, Zhang S, Zhou F, Feng T (2023) Stimulation of left dorsolateral prefrontal cortex enhances willingness for task completion by amplifying task outcome value. *J Exp Psychol Gen* **152**:1122-1133 https://doi.org/10.1037/xge0001312 | PubMed

Yang Y, Chen Z, Chen Q, Feng T (2021) Neural basis responsible for episodic future thinking effects on procrastination: The interaction between the cognitive control pathway and emotional processing pathway. *Cortex* **145**:250-263 https://doi.org/10.1016/j.cortex.2021.09.013 | PubMed

Zha R, Bu J, Wei Z, Han L, Zhang P, Ren J, Li JA, Wang Y, Yang L, VollstädtKlein S, *et al.* (2019) Transforming brain signals related to value evaluation and self-control into behavioral choices. *Human Brain Mapping* **40**:1049-1061 https://doi.org/10.1002/hbm.24379 | PubMed

Zhang C, Ni Y, Feng T (2017) The effect of regulatory mode on procrastination: Bistable parahippocampus connectivity with dorsal anterior cingulate and anterior prefrontal cortex. *Behavioural Brain Research* **329**:51-57 https://doi.org/10.1016/j.bbr.2017.04.019 | PubMed

Zhang S, Becker B, Chen Q, Feng T (2019) Insufficient task-outcome association promotes task procrastination through a decrease of hippocampal-striatal interaction. *Human Brain Mapping* **40**:597-607 https://doi.org/10.1002/hbm.24397 | PubMed

Zhang S, Feng T (2020) Modeling procrastination: Asymmetric decisions to act between the present and the future. *Journal of experimental psychology-General* **149**:311-322 https://doi.org/10.1037/xge0000643 | PubMed

Zhang S, Liu P, Feng T (2019) To do it now or later: The cognitive mechanisms and neural substrates underlying procrastination. *Wiley Interdiscip Rev Cogn Sci* **10**:e1492 https://doi.org/10.1002/wcs.1492 | PubMed

Zhang S, Verguts T, Zhang C, Feng P, Chen Q, Feng T (2021) Outcome Value and Task Aversiveness Impact Task Procrastination through Separate Neural Pathways. *Cerebral Cortex* **31**:3846-3855 https://doi.org/10.1093/cercor/bhab053 | PubMed

Zhang W, Wang X, Feng T (2016) Identifying the Neural Substrates of Procrastination: a Resting-State fMRI Study. *Scientific Reports* **6**:33203 https://doi.org/10.1038/srep33203 | PubMed

Zhao J, Meng G, Sun Y, Xu Y, Geng J, Han L (2021) The relationship between selfcontrol and procrastination based on the self-regulation theory perspective: the moderated mediation model. *Current Psychology* **40**:5076-5086 https://doi.org/10.1007/s12144-019-00442-3

1. **King AP,** Eckersley RJ (2019) Experimental Design and Sample Size Calculations (Statistics for Biomedical Engineers and Scientists).
2. **Faul F,** Erdfelder E, Lang AG, Buchner A (2007) G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods* **39**:175-191 https://doi.org/10.3758/bf03193146 | PubMed
3. **Baker DH,** et al. (2020) Power contours: Optimising sample size and precision in experimental psychology and human neuroscience. Psychological methods.
4. **Anderson,** et al. (2000) Null hypothesis testing: Problems, prevalence, and an alternative. *J Wildlife Manage* **2000 64**:912-923 https://doi.org/10.2307/3803199
5. **Wasserman L** (1997) Bayesian Model Selection and Model Averaging. *Journal of Mathematical Psychology* **44**:92-107 https://doi.org/10.1006/jmps.1999.1278 | PubMed
6. **Montagnini A,** Mamassian P, Perrinet L, Castet E, Masson GS (2007) Bayesian modeling of dynamic motion integration. *Journal of Physiology Paris* **101**:64-77 https://doi.org/10.1016/j.jphysparis.2007.10.013 | PubMed
7. **Benavoli A,** Corani G, Demsar J, Zaffalon M (2016) Time for a change: a tutorial for comparing multiple classifiers through Bayesian analysis. *Journal of Machine Learning Research* 18 https://doi.org/10.48550/arxiv.1606.04316
8. **Nathoo FS,** Masson M (2016) Bayesian alternatives to null-hypothesis significance testing for repeated-measures designs. *Journal of Mathematical Psychology* **72**:144-157 https://doi.org/10.1016/j.jmp.2015.03.003
9. **Wagenmakers E-J,** et al. (2018) Bayesian inference for psychology. Part II: Example applications with JASP. *Psychonomic Bulletin & Review* **25**:58-76 https://doi.org/10.3758/s13423-017-1323-7 | PubMed
10. **Ryan RM,** Bernstein JH, Brown KW (2010) Weekends, Work, and Well-Being: Psychological Need Satisfactions and Day of the Week Effects on Mood, Vitality, and Physical Symptoms. *Journal of Social & Clinical Psychology* **29**:95-122 https://doi.org/10.1521/jscp.2010.29.1.95
11. **Stone AA,** Schneider S, Harter JK (2012) Day-of-week mood patterns in the United States: On the existence of 'Blue Monday', 'Thank God it's Friday' and weekend effects. *Journal of Positive Psychology* **7**:306-314 https://doi.org/10.1080/17439760.2012.691980

Peer reviews

Reviewer #1 (Public review):

Summary:

The authors report the results of a tDCS brain stimulation study (verum vs sham stimulation of left DLPFC; between-subjects) in 46 participants, using an intense stimulation protocol over 2 weeks, combined with an experience-sampling approach, plus follow-up measures after 6 months.

Strengths:

The authors are studying a relevant and interesting research question using an intriguing design, following participants quite intensely over time and even at a follow-up time point. The use of an experience-sampling approach is another strength of the work.

Comments on revised version.

With the last round of revisions, the authors have now addressed my concerns.

https://doi.org/10.7554/eLife.108241.3.sa2

Reviewer #4 (Public review):

Summary:

The current study tested the effects of repeated sessions of tDCS targeting the DLPFC on procrastination behavior. The main outcome is that anodal versus sham DLPFC tDCS reduces procrastination behavior on both a short-term and a long-term scale up to six months after the stimulation sessions.

Strengths:

The current study tests competing models of procrastination with state-of-the-art high-definition transcranial electric stimulation. The study assesses stimulation effects on procrastination on both a short-term and a long-term scale, suggesting that repeated stimulation of the prefrontal cortex reduces procrastination on a time scale of up to six months.

Weaknesses:

The manuscript has already been reviewed and revised before, and it seems that the quality of the manuscript has substantially improved as a result of this revision process. I agree with the other reviewers that one must be cautious with drawing conclusions regarding the cognitive mechanisms underlying this effect, as many different cognitive functions are implemented by the DLPFC.

One aspect of the current results that puzzles me is the strength of the current stimulation effects. Meta-analyses suggest that tDCS shows only small-to-moderate effect sizes (with Cohen's d around 0.5). While the authors report no effect sizes for their statistical models, the small p values, in combination with the unusually small sample size of 18 participants per group, suggests that the effect size must be rather large. Can the authors provide an estimate of the effect size of their stimulation effects? If they are considerably larger than to be expected, could the authors give an explanation for why their stimulation setup is showing much stronger effects than comparable high-definition tDCS studies on cognition or decision making?

Regarding the strengths of the stimulation effects, I moreover found remarkable that the post-test procrastination rate was 100% in all (!) participants in the DLPFC group (figure 3F). I admit that it is hard to trust results that have no individual variation at all. This means that all participants are perfect responders to tDCS, which is again at variance what one typically expects for tDCS (where one usually has many non-responders). Do the authors have an explanation for this?

In any case, I am surprised by the rather small sample size. Due to the small effect sizes for tDCS, it is common to have a minimum of 30 subjects per group in between-subject designs. According to G*Power, a between-subject design with 17 subjects per group could detect only relatively large effect sizes of Cohen's d = 0.99 (alpha = 5%, power = 80%, independent-samples t-test). As explained above, this is far above the effect size that can be expected for

tDCS. In addition, small samples bear the risk that results strongly depend on outliers in the data, which might explain the strong effect size observed in the current study. The small sample size should be discussed as a major limitation of the current study and that the results need to be replicated by studies with larger sample sizes. Moreover, to rule out that the results are driven by outlier in the data, the authors should show individual data points in all plots showing empirical data.

Related to this, in the figure showing individual data points (3B/F), I count only around 10 data points per tDCS group for the 18 participants per group. I ask the authors to modify the plot that the data points from all participants can be seen (for example, by adding some noise on the x-axis for participants with the same value on the y axis).

Another surprising aspect of the data is that repeated sessions of tDCS change procrastination behavior up to six months after stimulation. Do the authors think that their tDCS setup leads to such long-lasting neuroplastic changes, and if yes, can they cite prior work where similar dosages of tDCS also showed such long-lasting effects? Or could the results be explained by learning effects, for example because participants in the DLPFC group learned during the repeated tDCS sessions that it feels internally rewarding to finish one's tasks instead of procrastinating them, and they still benefit from this kind of "learned industriousness" 6 months later? In any case, in my view it is important to be more specific about how seven sessions of tDCS can affect behavior half a year later.

Lastly, the link to the data repository works, but I could not inspect the data because I was asked to request access to the data, which I did not do in order to remain anonymous.

https://doi.org/10.7554/eLife.108241.3.sa1

Author response:

The following is the authors' response to the previous reviews

> ***Public Reviews:***
>
> ***Reviewer #1 (Public review):***
>
> *Summary:*
>
> *The authors report the results of a tDCS brain stimulation study (verum vs sham stimulation of left DLPFC; between-subjects) in 46 participants, using an intense stimulation protocol over 2 weeks, combined with an experience-sampling approach, plus follow-up measures after 6 months.*
>
> *Strengths:*
>
> *The authors are studying a relevant and interesting research question using an intriguing design, following participants quite intensely over time and even at a follow-up time point. The use of an experience-sampling approach is another strength of the work.*
>
> *Comments on revisions:*
>
> *Overall, I think the authors made many improvements to their manuscript. There are, however, still a number of concerns that first need to be addressed, since it is still not currently possible to fully evaluate the analyses, results, and conclusions presented in the paper. I list these points below:*
>
> *(1) The authors still use causal language where they must not use causal language. This is true for many places in the manuscript; I am highlighting here just a few places, but*

> *the authors nevertheless have to go carefully through the whole manuscript to change these instances.*

We sincerely thank the reviewer for this critical and well-taken point. We fully agree that our design (manipulating DLPFC excitability while measuring procrastination, task value, and aversiveness) does not directly measure or manipulate self-control, nor does it rule out alternative neurocognitive mechanisms. Accordingly, we have conducted a comprehensive, line-by-line revision of the entire manuscript to systematically replace causal claims with cautious, hypothesis-consistent language. In response, we have replaced all the wordings that may imply causal inferences, such as impair, cause, boost, by association-consistent phrasing. Furthermore, as you clearly raised below, we explicitly reframed self-control as a hypothesized theoretical construct rather than an empirically verified mediator throughout the whole re-revised manuscript. Please see specific revisions below:

Abstract Section (Page 2, Line 53-59)

"... a mediation analysis indicated a disassociable mechanism: the increase in task outcome value (but not task aversiveness) showed a statistical pattern consistent with accounting for the observed behavioral improvement. In conclusion, these findings are consistent with the hypothesis that enhancing DLPFC function may reduce procrastination by selectively amplifying the valuation of future rewards, not by simply reducing negative feelings about the task."

Introduction Section (Page 3, Line 83-86)

"... Even worse, chronic procrastination has been consistently associated with poor general health conditions, such as immune system disruption, gastrointestinal disturbance, hypertension and cardiovascular disease (Sirois, 2015; Sirois, 2016)."

Introduction Section (Page 4, Line 143-145)

"Consistent with this framework, the left dorsolateral prefrontal cortex (DLPFC)—a region frequently implicated in value-based decision-making and top-down regulation—has been associated with procrastination. ..."

Introduction Section (Page 4, Line 135-139)

"... Also, given the integrative nature of prefrontal regulatory functions, we hypothesize a third pathway whereby both decreased task aversiveness and increased task-outcome value may jointly contribute to reduced procrastination, potentially reflecting coordinated downstream effects on valuation and affective processing."

Introduction Section (Page 5, Line 183-186)

"... Thus, this study aims to clarify the brain-behavior association between DLPFC neuromodulation and procrastination, and to test whether observed changes in task valuation and aversiveness are consistent with theoretical models of top-down regulation."

Results Section (Page 11, Line 534-536)

"... Thus, these findings are consistent with the view that neuromodulation of the left DLPFC is associated with reduced task aversiveness and increased task-outcome value."

Results Section (Page 11, Line 579-584)

"... In summary, these findings identified a statistical pathway consistent with the theoretical model: neuromodulation of the left DLPFC was associated with increased task-outcome value, which in turn was associated with reduced procrastination."

Discussion Section (Page 12, Line 617-620)

“On balance, our findings provide evidence consistent with the hypothesis that neuromodulation of the left DLPFC is associated with reduced procrastination, primarily through increasing task-outcome value rather than merely reducing task aversiveness. ...”

Discussion Section (Page 13, Line 664-669)

“... Building on this foundation, among several theoretical interpretations and cognitive pathways, our study showed the one plausible neurocognitive mechanism of procrastination: the cortical excitability of the DLPFC produced by active neuromodulation may engage prefrontal regulatory networks to increase task outcome value, which in turn is associated with reduced procrastination behavior, statistically supporting the theoretical accounts of temporal decision model (TDM, Zhang et al., 2019).”

Discussion Section (Page 14, Line 755-762)

“... Moreover, this study did not collect data for assessing participants' self-control at either baseline or post-neuromodulation. Accordingly, we explicitly note that self-control was not directly measured or manipulated in this study; the observed associations between DLPFC neuromodulation, task-outcome value, and procrastination are consistent with theoretical models positing a role for top-down regulatory processes, but do not constitute direct evidence that self-control mechanisms were engaged. This limitation precludes definitive conclusions about the unique contribution of self-control-related pathways versus alternative neurocognitive mechanisms.”

> *Some examples:*
>
> *(a) In response to my comment (1) in the previous round, where the authors adjusted their text, the authors still use causal language in their last sentence "... procrastination behavior has been observed to impair general health..." Unless the cited study truly allowed causal conclusions, the causal language should be removed here as well.*

Thank you for pointing out this inappropriate phrasing. As you kindly suggested, we have reworded it as “Even worse, chronic procrastination has been consistently associated with poor general health conditions, such as immune system disruption, gastrointestinal disturbance, hypertension and cardiovascular disease” (Introduction Section, Page 3, Line 83-86).

> *(b) The authors still make (causal) claims about the involvement of self-control in their observed results. To reiterate from the previous round of revisions: The authors cannot make any strong claims about the role of self-control processes because they do not directly measure self-control nor do they directly manipulate self-control or have a design that would rule out alternative mechanisms other than self-control. Therefore, their claims about self-control have to be toned down. It is laudable that the authors have added a statement towards the end of their discussion about not being able to make strong conclusions about the role of self-control. But the authors need to use similar careful wording not just at the end of the discussion but throughout the manuscript.*

We appreciate you reiterating this concern. In the re-revised manuscript, we have thoroughly removed or rewritten all the statements implying causal inferences, and have substantially toned-down claims for the roles of self-control in procrastination reduction from this neuromodulation. Please see instances for what we have replied to the Comment #1.

> *(i) In the abstract, the authors use the formulation "...conceptualized roles of self-control on procrastination..." -- this wording is still too strong, suggesting that you actually*

> *studied self-control.*

Thank you for providing this specific instance. This inappropriate sentence has been removed.

> *(ii) In the introduction (page 4, lines162-169), the way the authors formulate these sentences suggests that they directly measured self-control. Again, the authors need to make it explicit that they are not directly measuring self-control but its hypothesized down-stream consequences on valuations/behavior.*

Many thanks. This statement has been removed, and we have reworded it as "... Thus, this study aims to clarify the brain-behavior association between DLPFC neuromodulation and procrastination, and to test whether observed changes in task valuation and aversiveness are consistent with theoretical models of top-down regulation." (Introduction Section, Page 5, Line 183-186).

> *(iii) In the discussion, for example, on page 11, lines 555 and following, the authors write: "One major contribution this study has made is to disentangle the neurocognitive mechanism of procrastination by demonstrating that self-control could increase task-outcome value so as to reduce procrastination."*

As you kindly instructed, we have rewritten this statement as "One contribution of this study is to provide empirical evidence partially consistent with the temporal decision model (TDM), showing that increased task-outcome value—rather than decreased task aversiveness—was statistically associated with reduced procrastination following DLPFC neuromodulation." (Introduction Section, Page 12, Line 625-628), which no longer implies any conclusions for the role of self-control in this study.

> *Again, please be aware that you are NOT demonstrating that self-control does anything, since you only measure procrastination rates, outcome values, and task aversiveness. It is possible that mechanisms other than self-control might be relevant for this. Perhaps neuromodulation directly increases outcome values, without involvement of self-control processes. You simply cannot know that and therefore you cannot make those claims in the form that you are making them. You can write that the observed results are consistent with the idea that neuromodulation might have had an effect on self-control and this in turn might have affected outcome values. But you also need to make it explicit that, to substantiate these claims, you would need more direct evidence that indeed self-control was involved. These more careful formulations would not at all reduce the value of your work, but indeed they would rather demonstrate your carefulness in interpreting the results you obtained.*

We sincerely thank the reviewer for this exceptionally clear and constructive guidance. We fully agree that our study design does not measure or manipulate self-control, and therefore we cannot demonstrate that self-control processes are causally involved in the observed effects. As you correctly note, it is entirely possible that neuromodulation directly modulates outcome valuation or engages alternative neurocognitive pathways (e.g., attentional allocation, feedback learning, or affective processing) without invoking self-control mechanisms.

In direct response, as we replied above, we have completely rewritten the whole revised manuscript to remove any assertions that we "identified" a role of self-control. The revised text now explicitly states as follow: (1) our findings merely are consistent with the theoretical hypothesis that DLPFC neuromodulation might engage prefrontal self-regulatory functions, which in turn influence outcome valuation; (2) we explicitly acknowledge that substantiating this specific pathway would require more direct evidence. Rather than single sentence, we have applied this careful, hypothesis-consistent framing systematically across the Abstract,

Introduction, Results, and Discussion. As you suggested, these revisions more accurately reflect the interpretative boundaries of our data and demonstrate our commitment to rigorous, transparent scientific reporting. Please see specific cases for this revision above.

> *(2) I am still puzzled by the power analysis. In the text, you write that a sample size of 18 participants (i.e., 9 per group) would be sufficient to achieve 80% power. I still feel this seems far too optimistic and hard to believe, but that is not my point here. While in the text, you write that you need 18 participants, the G*power output seems to suggest a sample size of 34, not 18. Why this contradiction? Or is it not contradictory? If it is not, then please explain it more fully.*

We appreciate you pointing out this critical typo. In the last round of revision, we mean that 18 participants per group are required to achieve at least 80% statistical power, rather than a total sample size, as shown by the G*Power software. *We are sorry for this critical typo to confuse you. As you correctly pointed out, the G*Power indicated that the minimum sample size to reach 80% power is 34 (i.e., 17 per group). Thus, we selected 36 (i.e., 18 per group) participants as minimum sample size in case of potential drop-out. We have thoroughly corrected this typo, and double-checked no such numeric issues:

Methods Section (Page 5, Line 234-237)

"... statistical power was predetermined by G*Power at a relatively medium effect size (1-β err prob = 0.80, f = 0.25), indicating the total sample size at 34 (17 per group) to reach acceptable power. To account for potential attrition, we determined to recruit 36 participants, at least.".

> *(3) I have several comments about the mixed-effects analysis.*
>
> *First of all, I want to thank the authors for adding more details, things have become much clearer now. However, I still have a few questions and comments related to these analyses:*
>
> *(a) The variable Emotions was within-subjects, as far as I understood. Accordingly, Emotions should most likely be modelled with random slopes varying over participants (in addition to being modelled as a fixed effect).*

We thank you raising this reasonable concern on the mixed-effect linear modeling. Yes, the Emotions reflect daily baseline affect, which is modeled as covariates of no interests to adjust for daily emotional fluctuation (if any). In this vein, this baseline emotion score is included for each participant across all the sessions. Therefore, it should be modeled with random slopes as you assumed indeed.

In response, we have remodeled this mixed-effects analysis by including the daily baseline emotion as random slopes varying over participants. After centering the variables, we estimated the revised models as "Procrastination Rate ~ Group * Treatment day + Age + Gender + SES + Emotions + (1 + Treatment day + Emotions || SubjectID)" and "Task execution willingness ~ Group * Treatment day + Age + Gender + SES + Emotions + (1 + Treatment day + Emotions || SubjectID)". Notably, as you correctly assumed, fitting this complicated random-effect structure is likely to result in convergence failure, given the limited sample size in the present study. Therefore, we hypothesized the independence among random effects for model simplification. Consistent with this assumption, model comparisons indicated that the simplified models fit better than original ones (Procrastination Rate model, ΔAIC = -1.0, ΔBIC = -11.8, LRT, $\chi^2(3) = 5.04$, $p = .17$; Task-execution willingness model, ΔAIC = -5.5, ΔBIC = -16.4, LRT, $\chi^2(3) = 0.51$, $p = .91$).

Taken together, as you kindly suggested, we have rebuilt the mixed-effect models by adding daily baseline emotion as random slopes varying over participants, and have demonstrated

the consistent findings with the original one:

Methods Section (Page 8-9, Line 412-419)

"... Given the risks of convergence failure with the two correlated random-effects structure (i.e., treatment days and self-reported emotions), we hypothesized that the random effects are independent, leading to model simplification. Consistent with this assumption, model comparisons favored the simplified independent structure over the full correlated structure for both outcomes. For the actual procrastination model, the simplified model showed lower AIC (Δ = -1.0) and BIC (Δ = -11.8), with a non-significant likelihood ratio test (χ^2 (3) = 5.04, p = .17). For the task-execution willingness model, the simplified model was also preferred (ΔAIC = -5.5, ΔBIC = -16.4; LRT: χ^2 (3) = 0.51, p = .91)."

Results Section (Page 9-10, Line 469-489)

"For procrastination willingness, results showed a statistically significant interaction effect between multi-session neuromodulations and groups (β = -7.84, SE = 1.80, t = -4.36, DF = 45.6, p < .001; Fig. 3A). In the post-hoc simple effect analysis, it demonstrated a significantly increased task-execution willingness (i.e., decreased procrastination willingness) after neuromodulation in the active neuromodulation group (NM-before: 35.65 ± 30.21, NM-after: 80.43 ± 19.92, Mean Diff = 41.79, SE = 7.58, DF = 103.4, t.ratio = 5.51, p < .0001, Tukey correction), but no such effects were identified in the sham control group (SC-before: 37.57 ± 26.46, SC-after: 47.35 ± 30.49, Mean Diff = 2.58, SE = 7.56, DF = 96.8, t.ratio = 0.34, p = .73, Tukey correction) (Fig. 3B-C). A linear uptrend for task-execution willingness was further observed across multiple sessions in the active NM group, indicating gradually increasing neuromodulation effects (Fig. 3D; p < .01, Mann-Kendall test). For actual procrastination behavior, changes to actual procrastination rates across all the sessions have been detailed in the Fig. 3E. Similarly, a statistically significant interaction effect was identified here (β = -7.37, SE = 2.40, t = -3.02, DF = 46.6, p = .004), and the simple effect analysis further revealed decreased actual procrastination rates after ms-tDCS in the active neuromodulation group (NM-before: 56.74 ± 39.10, NM-after: 0.00 ± 0.00, Mean Diff = 44.40, SE = 9.36, DF = 110.0, t.ratio = 4.74, p < .0001, Tukey correction), but no such prominent changes found in the sham control group (SC-before: 46.47 ± 40.76, SC-after: 33.35 ± 37.82, Mean Diff = 7.53, SE = 9.28, DF = 102.0, t.ratio = 0.81, p = .42, Tukey correction) (Fig. 3F-G)."

> *(b) The analyses still cannot fully be evaluated as I cannot access the scripts and data. The authors mention that the scripts and data should be available via a link they provide (https://doi.org/10.57760/sciencedb.35140). However, when I try to access these materials via this link, no page opens; it seems the link is dead?*

Thank you very much for bringing this case to us. We checked this link and found it to be still active.

To ensure accessibility for your evaluation, we have uploaded scripts and data into this online submission system. Please do let us know if you are still unable to access them. We are glad to send them to you by other available pathways. This link is a private access to you, and the repository would be openly available for other users upon the final publication.

> *(c) What are the results and conclusions if you do not include the covariates of no interest? I.e., please re-run your main models without age, gender, SES, Emotions.*

Thank you for raising this question. As you clearly instructed, we have rerun main models without all those covariates. As shown in the table below, the results for the key predictors of interest (Group, Treatment day, and their interaction) remained largely unchanged in terms of effect size, direction, and statistical significance:

DV	Effect	Covariate	β	SE	t	p	95% CI
Task-execution Willingness	Group	Included	-22.18	5.66	-3.92	<.001	[-33.50, -10.86]
	Group	Excluded	-21.57	5.50	-3.92	<.001	[-32.57, -10.57]
	Treatment day	Included	9.53	1.28	7.47	<.001	[6.97, 12.09]
	Treatment day	Excluded	9.18	1.31	6.99	<.001	[6.56, 11.80]
Actual procrastination rate	Group×Day interaction	Included	-7.84	1.80	-4.36	<.001	[-11.44, -4.24]
	Group×Day interaction	Excluded	-7.48	1.86	-4.03	<.001	[-11.20, -3.76]
	Group	Included	-25.96	6.49	-4.00	<.001	[-38.94, -12.98]
	Group	Excluded	-24.77	6.19	-4.00	<.001	[-37.15, -12.39]
	Treatment day	Included	8.92	1.73	5.15	<.001	[5.46, 12.38]
	Treatment day	Excluded	8.99	1.77	5.09	<.001	[5.45, 12.53]
	Group×Day interaction	Included	-7.37	2.44	-3.02	.004	[-12.25, -2.49]
	Group×Day interaction	Excluded	-7.44	2.50	-2.98	.005	[-12.44, -2.44]

Author response table 1. Comparison to statistics derived from model with covariates (i. e., age, gender, SES, Emotions) and without covariates

> *(d) The authors mention that they use GLMMs, which would suggest generalized mixed-effects models, but they do not describe what family/distribution they used. Since they mention lmerTest and seem to report F-tests, my guess is that they used Gaussian models. However, both their DVs (procrastination rates and their ratings) are bounded variables and at least procrastination rates hit the lower boundary. That can mean that their analyses suffer from inflated Type 1 and/or Type 2 rates. Therefore, please repeat the analyses with an appropriate generalized mixed-effects model (perhaps a beta regression type of model?).*

We are very grateful to you for raising this crucial statistical point. As you correctly pointed out, we used the Gaussian distribution in estimating this model. We are sorry to confuse you due to the absence of reporting family/distribution we used. In the original manuscript, we meant “general” linear mixed-effect model, rather than “generalized” one. As you clearly and correctly raised, procrastination rates and willingness are technically bounded, and that

procrastination rates frequently reached the lower boundary (0%) in the present study, which are in high risks to be inflated for Type 1 and/or Type 2 error.

Thus, as you kindly suggested, a beta family distribution with logit function is used to reanalyze those main effects of interest. Results are tabulated in Author response table 2.

DV	Effect	*b*	SE	*z*	*p*	Consistency with Gaussian model
Actual procrastination rate	Group	-0.760	0.202	-3.77	< .001	Yes (p < .001)
	Treatment Day	0.225	0.064	3.50	< .001	Yes (p < .001)
	Group×Day interaction	-0.182	0.091	-2.01	.045	Yes (p = .004)
Task-execution Willingness	Group	-1.081	0.282	-3.83	< .001	Yes (p < .001)
	Treatment Day	0.446	0.064	7.00	< .001	Yes (p < .001)
	Group×Day interaction	-0.375	0.086	-4.35	< .001	Yes (p < .001)

Author response table 2.

These convergent results confirm that the critical main effect (i.e., Group and Treatment day) and their interaction remain statistically significant across distributional specifications, and that our primary conclusions are not artifacts of the Gaussian assumption. Taken them together, as you kindly suggested, we have repeated the analyses with beta regression family distribution, which replicated our main findings, potentially supporting their statistical robustness.

Following your suggestion, we have added those results derived from such sensitivity analyses into the revised manuscript:

Methods Section (Page 9, Line 430--436)

"... To examine whether our findings were sensitive to the distributional assumptions of the dependent variables, we re-analyzed the main models using an alternative distributional specification. Given that both procrastination rates (ranging from 0% to 100%) and task-execution willingness (measured on a 0-100 visual analog scale) are bounded continuous outcomes, and that procrastination rates frequently reached the lower boundary (0%) in the present study, a Beta regression model with a logit link function was employed for a sensitivity analysis."

Results Section (Page 10, Line 506-512)

"... Furthermore, as a sensitivity analysis, we reran the main LMMs using Beta regression distribution with a logit link function, which is appropriate for the both bounded outcomes mentioned above (i.e., procrastination rate and procrastination willingness). The main effects (i.e., Group and Treatment day) and their interaction remained significant for both procrastination rate and willingness (see SI Results and Tab. S5), confirming that our findings are robust to alternative distributional assumptions."

> *(e) When reporting the results of the mixed-effects models, the authors report the regression coefficient, standard error, DFs and p value, but not the actual test statistic.*

> *Please add the information about the test statistic and report all degrees of freedom (in case of F tests that would be the degrees of freedom of the test and the residual degrees of freedom).*

We truly thank you for this nuanced reminder. As you suggested, we have added actual test statistics, including t-values and all degrees of freedom (DF). Please see specific instances below:

Results Section (Page 9-10, Line 469-489)

"For procrastination willingness, results showed a statistically significant interaction effect between multi-session neuromodulations and groups (β = -7.84, SE = 1.80, t = -4.36, DF = 45.6, p < .001; Fig. 3A). In the post-hoc simple effect analysis, it demonstrated a significantly increased task-execution willingness (i.e., decreased procrastination willingness) after neuromodulation in the active neuromodulation group (NM-before: 35.65 ± 30.21, NM-after: 80.43 ± 19.92, Mean Diff = 41.79, SE = 7.58, DF = 103.4, t.ratio = 5.51, p < .0001, Tukey correction), but no such effects were identified in the sham control group (SC-before: 37.57 ± 26.46, SC-after: 47.35 ± 30.49, Mean Diff = 2.58, SE = 7.56, DF = 96.8, t.ratio = 0.34, p = .73, Tukey correction) (Fig. 3B-C). A linear uptrend for task-execution willingness was further observed across multiple sessions in the active NM group, indicating gradually increasing neuromodulation effects (Fig. 3D; p < .01, Mann-Kendall test). For actual procrastination behavior, changes to actual procrastination rates across all the sessions have been detailed in the Fig. 3E. Similarly, a statistically significant interaction effect was identified here (β = -7.37, SE = 2.40, t = -3.02, DF = 46.6, p = .004), and the simple effect analysis further revealed decreased actual procrastination rates after ms-tDCS in the active neuromodulation group (NM-before: 56.74 ± 39.10, NM-after: 0.00 ± 0.00, Mean Diff = 44.40, SE = 9.36, DF = 110.0, t.ratio = 4.74, p < .0001, Tukey correction), but no such prominent changes found in the sham control group (SC-before: 46.47 ± 40.76, SC-after: 33.35 ± 37.82, Mean Diff = 7.53, SE = 9.28, DF = 102.0, t.ratio = 0.81, p = .42, Tukey correction) (Fig. 3F-G)."

> *(f) Thank you for adding the analysis where you remove the last two sessions. But currently you present them in the manuscript without explaining/motivating why you do this. Please add this motivation, as otherwise it will be puzzling for the reader why you conduct these analyses.*

Thank you for this very practical requirement to clarify the motivation of reanalyzing main models from removing the last two sessions. Please see specific explanation as follow:

Results Section (Page, Line 499-506)

"... To systematically test whether such effects are biased by extreme data points or patterns, we reran the main LMMs by iteratively removing data from the last two sessions, which showed extraordinarily high effectiveness from neuromodulation (e.g., all the participants in the neuromodulation group had no actual procrastination behavior in session #6 and #7). Results showed the significant group*neuromodulation sessions interaction effects across all those nested models (removing session #6, #7 or both, all p < .05; see SI Results and Tab. S3-4), potentially indicating a statistical robustness from the data pattern."

> *(4) Mediation analysis*
>
> *In your manuscript, you present some mediation analyses. Please be aware that such mediation analyses cannot establish causality and they suffer from extremely high Type 1 error rates (see, e.g., https://datacolada.org/103). My suggestion would be to completely remove all mediation analyses. However, if you want to keep them, then you need to be extremely careful in how you present the results. You need to explicitly mention that you cannot derive any causal conclusions from them and that simulation*

> *studies have shown that such mediation analyses suffer from extremely high Type 1 errors.*

We sincerely thank you for this exceptionally important methodological guidance. We fully agree that mediation analyses, especially those based on observational measures rather than experimentally manipulated mediators, cannot establish causal pathways and are susceptible to inflated Type 1 error rates, as rigorously demonstrated in recent simulation studies (*https://datacolada.org/103*).

As you kindly suggested, please allow us to retain those mediation analyses upon explicitly highlighting that this mediation statistical model cannot generate any causal conclusions. In response, we have systematically replaced all instances of “causal mediation” with “statistical mediation” or “exploratory mediation analysis”, and removed causal verbs (e.g., “depends on”, "drives", "explains") in favor of association-consistent phrasing (e.g., “is statistically mediated”, “aligns with the hypothesis that”) throughout the abstract, introduction, methods, results and discussion sections. Furthermore, in the Discussion section, we explicitly reiterated the limitations of extending this mediation associations to causal conclusions. Please see specific modifications underneath:

Abstract Section (Page 2, Line 52-56)

“... While the intervention is significantly associated with both decreased task aversiveness and increased perceived task outcome value, a mediation analysis indicated a disassociable mechanism: the increase in task outcome value (but not task aversiveness) showed a statistical pattern consistent with accounting for the observed behavioral improvement.”

Methods Section (Page 9, Line 448-453)

“... As these mediation analyses are based on observational measures rather than experimentally manipulated mediators, they do not establish causal pathways. Simulation studies have shown that such analyses can suffer from inflated Type 1 error rates. Results should therefore be interpreted as hypothesis-generating and statistically consistent with the proposed theoretical model, rather than as confirmatory evidence of causal mechanisms.”

Methods Section (Page 9, Line 438-440)

“... the Quasi-Bayesian mediation analysis was used to model the association between the effects of tDCS, task aversiveness/outcome and decreased procrastination.”

Results Section (Page 11, Line 568-573)

“As an exploratory analysis, results indicated that increased task outcome value was associated with changes in the task-execution willingness ($\delta = 21.73$, $p < .01$; $\zeta = 11.25$, $p = .07$, $\rho = 32.99$, $p < .01$, simulation = 1,000; see Fig. 5A) and real-world procrastination ($\delta = 30.75$, $p < .01$; $\zeta = 3.05$, $p = .52$, $\rho = 33.81$, $p < .01$, simulation = 1,000; see Fig. 5B), in the context of ms-tDCS neuromodulation. ...”

Results Section (Page 11, Line 582-584)

“... Nevertheless, all mediation findings are now explicitly labeled as “exploratory” and framed as quantifying statistical associations consistent with the TDM pathway, not causal mediation.”

Discussion Section (Page 14, Line 747-755)

“Notably, we explicitly acknowledge that exploratory Quasi-Bayesian mediation analyses, based on observational measures rather than experimentally manipulated mediators, cannot establish causal pathways and are susceptible to inflated Type 1 error rates as demonstrated

in recent simulation studies. These findings should be interpreted strictly as hypothesis-generating and statistically consistent with the proposed theoretical model, rather than as confirmatory evidence of causal mechanisms. Substantiating the precise neurocognitive pathway will require future studies employing stronger causal designs, such as experimental manipulation of task valuation or longitudinal cross-lagged modeling. ...”

> *As an example (but the mediation results are mentioned in several places, for example, also in the abstract): On page 10, lines 501-503: What you can causally conclude is that neuromodulation affects your measured variables (outcome values, procrastination rates, task aversiveness), but you cannot conclude that the effect of neuromodulation on procrastination rates causally operates via outcome values. Thus, please adjust the formulation accordingly. The same applies to the mediation section that follows right afterwards (page 10, lines 505-522).*

Thank you for offering those specific instances. As we replied above, they have been revised accordingly:

Results Section (Page 11, Line 557-559)

“... Collectively, these findings provide statistical evidence consistent with the hypothesis that the outcome-value pathway may contribute to procrastination reduction.”

Results Section (Page 11, Line 563-582)

“Increased task outcome value is specifically associated with reduced procrastination in the context of neuromodulation

To explore the potential neurocognitive pathways of procrastination, the Quasi-Bayesian mediation analysis was undertaken, with increased task outcome value as a statistically mediated variable. As an exploratory analysis, results indicated that increased task outcome value was associated with changes in the task-execution willingness ($\delta = 21.73$, $p < .01$; $\zeta = 11.25$, $p = .07$, $\rho = 32.99$, $p < .01$, simulation = 1,000; see Fig. 5A) and real-world procrastination ($\delta = 30.75$, $p < .01$; $\zeta = 3.05$, $p = .52$, $\rho = 33.81$, $p < .01$, simulation = 1,000; see Fig. 5B), in the context of ms-tDCS neuromodulation. To ensure the statistical robustness and specificity of these findings, the sensitivity analysis was implemented by changing sampling parameters and outcome variables. By doing so, those findings were validated statistically robust, as shown by replicated observations across bootstrapping sampling subsets (see SI Results and Tab. S6-7). Moreover, the results of the control analysis further validated the specificity of these findings by showing a null statistically mediated effect of this model to predict one’s task aversiveness (see SI Results and Tab. S8). In summary, these findings identified a statistical pathway consistent with the theoretical model: neuromodulation of the left DLPFC was associated with increased task-outcome value, which in turn was associated with reduced procrastination.”

> *(5) In the introduction, the authors introduce several theoretical procrastination frameworks (TMT, mood repair, TDM). Do the results of the current paper help to decide which framework might be the most appropriate, at least for the authors data set? It might be of interest to address this explicitly.*

We do thank the reviewer for this insightful theoretical question. We agree that explicitly positioning our findings within the broader theoretical landscape strengthens the conceptual contribution of our work. Upon careful consideration, we believe that our results provide the strongest empirical support for the TDM over alternative frameworks (TMT, mood repair). TDM uniquely posits procrastination as contingent on the dynamic trade-off between task aversiveness and task-outcome value. In the present study, neuromodulation was identified to be associated with both pathways but only increased outcome value statistically predicted reduced procrastination. This aligns precisely with TDM’s hypothesis that value-based

processes may dominate aversiveness-avoidance processes in driving behavioral change. Neither TMT (which emphasizes temporal discounting of utility per se) nor the mood repair perspective (which prioritizes short-term affect regulation) explicitly predicts this dissociable pattern. As you kindly suggested, we have extended the Discussion section to explicitly contextualize our findings into this theoretical landscape (Discussion Section, Page 13, Line 669-687).

> *(6) The language is sometimes hard to understand and seems in quite some places grammatically incorrect. Thus, I think the paper would profit very much from thorough English proofreading.*

We sincerely thank you for this practical suggestion. We fully agree that the original manuscript contained grammatical inaccuracies and awkward phrasing that could hinder readability. In response, we have engaged a professional academic editing service to thoroughly proofread and polish the entire manuscript. All sentences have been revised for grammatical correctness, syntactic clarity, and academic tone, while strictly preserving the original scientific meaning and technical terminology. We believe this language improvements have substantially enhanced the readability and overall quality of the paper.

> ***Reviewer #2 (Public review):***
>
> *Summary:*
>
> *Chen and colleagues conducted a cross-sectional longitudinal study, administering high-definition transcranial direct stimulation (HD-tDCS) targeting the left DLPFC to examine the effect of HD-tDCS on real-world procrastination behavior. They find that seven sessions of active neuromodulation to the left DLPFC elicited greater modulation of procrastination measures (e.g., task-execution willingness, procrastination rates, task aversiveness, outcome value) relative to sham. They show that HD-tDCS reduces task aversiveness and increases task-execution willingness on real-world tasks as quantified by intensive experience sampling methods, providing causal evidence for the role of DLPFC in modulating contextual features to delaying or completing one's goals.*
>
> *Strengths:*
>
> - *This is a well-designed protocol with rigorous administration of high-definition transcranial direct current stimulation across multiple sessions. The intensive experience sampling approach which probes and assesses self-relevant task goals is innovative and aims to address an important question regarding the specific role of DLPFC in modulating specific features of chronic procrastination behavior (e.g., task-execution willingness, task aversiveness).*
> - *The quantification of task aversiveness through AUC metrics is a clever approach to account for the temporal dynamics of task aversiveness, which is notoriously difficult to quantify.*
>
> *Weaknesses:*
>
> - *While the findings that neurostimulation reduces procrastination behavior is compelling, there remain several alternative interpretations for these effects. For example, it could be that the task-execution willingness isn't increased per se, but rather that the goal completion becomes more valuable as participants learn from feedback or become more aware of their successful attainment of or failure to complete task goals. It is unclear whether the effects could be driven by improved working memory or attention to the reported tasks (and this limitation is addressed by the authors). In short, it is also difficult to examine the temporal dynamics of how these goals are selected across time.*

We sincerely thank you for raising these thoughtful and methodologically important points. We fully agree that the observed reductions in procrastination could reflect multiple neurocognitive pathways beyond the value-based mechanism emphasized in our primary analysis.

In response, we have thoroughly removed claims on the "unique mechanism" of value-based pathways to procrastination reduction, and fully substituted language implying exclusive mediation by "value amplification" with more cautious phrasing (e.g., "statistically consistent with a value-based pathway"; "one plausible mechanism among several processes"). In the revised manuscript, we reiterated that the pattern of results, showing increased outcome value predicting reduced procrastination while decreased aversiveness did not, aligned with the Temporal Decision Model, yet does not rule out concurrent contributions from attention, learning, or executive processes. To clearly bring this interpretative boundary of our primary findings for audiences, we have explicitly warranted such cautions in the Discussion Section. Please see specific modifications underneath:

Discussion Section (Page 13, Line 664-669)

"... Building on this foundation, among several theoretical interpretations and cognitive pathways, our study showed the one plausible neurocognitive mechanism of procrastination: the cortical excitability of the DLPFC produced by active neuromodulation may engage prefrontal regulatory networks to increase task outcome value, which in turn is associated with reduced procrastination behavior, statistically supporting the theoretical accounts of temporal decision model (TDM, Zhang et al., 2019). "

Discussion Section (Page 13, Line 676-687)

"... Despite statistically supporting the TDM, we acknowledge that alternative neurocognitive mechanisms could contribute to the observed reductions in procrastination. For instance, repeated exposure to the experience-sampling protocol may have enhanced participants' awareness of task progress or facilitated feedback-based learning, thereby increasing the subjective value of goal completion independent of DLPFC neuromodulation. Similarly, improvements in working memory for task maintenance, attentional allocation to reported goals, or strategic shifts in goal selection across sessions could plausibly mediate the intervention effects. While our double-blind, sham-controlled design and inclusion of daily emotional covariates help mitigate some non-specific confounds, the present study did not incorporate direct measures of these alternative processes. Consequently, we cannot definitively isolate the value-based pathway posited by the TDM from concurrent contributions of attention, learning, or executive functions."

> - *It is unclear whether the current evidence support long-retention of this neurostimulation intervention. The study includes one 6-month timepoint after the study to examine the long-term retention of the neural stimulation effect. Future studies that evaluate the long-term effects across multiple time points would strengthen the evidence for the robustness of this intervention.*

We genuinely appreciate you for this insightful and methodologically reasonable point. We fully agree that a single 6-month follow-up assessment, while valuable, provides only preliminary evidence for long-term retention, and that multiple follow-up timepoints would substantially strengthen claims about the durability of neuromodulation effects. To carefully address this point, we have rephrased the "long-term retention" as "long-term after-effects" throughout the whole revised manuscript, and overall toned down the claims on the retention effects. Moreover, this limitation has been explicitly elaborated in the Discussion section:

Abstract Section (Page 2, Line 49-50)

"... we assessed the effect of anodal HD-tDCS on real-world procrastination behavior at offline after-effect (2-day interval) and long-term after-effect (6-month follow-up)."

Results Section (Page 12, Line 606-608)

"... Therefore, beyond short-term effects, the benefits of ms-tDCS neuromodulation on reducing procrastination were still detectable at a 6-month follow-up, providing preliminary evidence consistent with long-term after-effects."

Discussion Section (Page 14, Line 721-725)

"... Thus, the detectable effects at 6 months are consistent with the hypothesis that repeated neuromodulation may induce neuroplastic changes in the DLPFC that support sustained behavioral change. However, we explicitly note that a single follow-up timepoint cannot establish the stability or trajectory of these effects; future studies with multiple longitudinal assessments are required to substantiate claims about long-term retention."

Discussion Section (Page 15, Line 771-775)

"... Finally, while our 6-month follow-up provides preliminary evidence for sustained effects, the use of a single follow-up timepoint limits our ability to characterize the temporal trajectory of retention. Future studies incorporating multiple follow-up assessments (e.g., 1-month, 3-month, 6-month, 12-month) would strengthen evidence for the robustness and durability of this intervention."

> ***Recommendations for the authors:***
>
> ***Reviewer #1 (Recommendations for the authors):***
>
> *Please see my detailed comments above (6 points; several of them with subpoints a, b, c, etc).*

Thank you for listing those specific and helpful recommendations above. Please see our detailed response posed above, point-by-point.

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