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

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eLife Assessment

This potentially **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. Support for the conclusions is **incomplete** owing to missing information about the analyses, the nature of the procrastination tasks, and the derived dependent measures.

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Abstract

Procrastination represents one of the most prevalent behavioral problems affecting individual health and societal productivity. Although it is often conceptualized as a form of self-control failure, its underlying neurocognitive mechanisms are 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 fully validated in real-world settings and not applied effectively to guide interventions. Here, we addressed this gap with a preregistered, 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), a key region for self-control, 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 retention (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 both decreased task aversiveness and increased perceived task outcome value, causal mediation analysis revealed a striking mechanism: the increase in task outcome value uniquely and sufficiently mediated the entire behavioral improvement. In conclusion, these findings provide causal evidence that enhancing DLPFC function mitigates procrastination by selectively amplifying the valuation of future rewards, not by simply reducing negative feelings about the task. This establishes a precise, value-driven neurocognitive pathway for self-control and offers a validated, theory-driven strategy for intervention.

Introduction

Procrastination is increasingly becoming one of the most prevalent behavior problems, which reflects the irrational voluntary postponement of scheduled tasks albeit being worse off for such delays (Blake, 2019 [↗](#); Steel, 2007 [↗](#)). In the cross-cultural investigations, more than 15% adults were identified as having chronic procrastination problems, and the situation for students was worse as 70-80% undergraduates engaged in procrastination (American College Health Association, 2022 [↗](#); Ferrari et al., 2005 [↗](#)). Moreover, the behavioral genetic evidence indicated 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 deleterious consequences of procrastination behavior should warrant more caution. There was cumulative evidence to show that procrastination behavior endangers working performance, financial status, interpersonal relationships, and subjective well-being (Ferrari, 1994 [↗](#); Pychyl & Sirois, 2016 [↗](#); Steel et al., 2021 [↗](#)). Further, many mental health problems have been found to be consequences of procrastination, such as sleep problems, depression, and anxiety (Hairston & Shpitalni, 2016 [↗](#); Johansson et al., 2023 [↗](#)). Even worse, chronic procrastination behavior has been consistently observed in downgrading one's life health as shown by the close association between procrastination and immune system disruption, gastrointestinal disturbance, as well as a high risk of hypertension and cardiovascular disease (Sirois, 2015 [↗](#); Sirois, 2016 [↗](#)). Thus, given these critical consequences, many efforts have been spent to delve into why we procrastinate irrationally.

To probe into 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 (Steel, 2007 [↗](#)). This theory suggests that individuals would procrastinate the task 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 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 decision, which highlights that procrastination would be 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 the 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.

It is worthwhile to note that this trade-off and resultant decisions to procrastinate were predominantly influenced by self-control capability, with higher self-control for less procrastination behavior (Blake, 2019 [↗](#); Ramzi & Saed, 2019 [↗](#); Zhao et al., 2021 [↗](#)). Thus far, there have been three promising pathways attempting to clarify how self-control works for reducing 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 in both behavioral and neural evidence, procrastinators consistently reported 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, the self-control was observed as powerful for downstream regulation of negative emotional stimuli (Paschke et al., 2016 [↗](#); Tice & Bratslavsky, 2000 [↗](#)). Therefore, the first possible pathway is to enhance self-control by facilitating emotional regulation towards 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 strengthening self-control (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 multifarious roles of self-control, it leads us to hypothesize the third pathway that both decreased task aversiveness and increased task-outcome value contribute to determining procrastination simultaneously through the exertion of self-control.

Supporting that, the left dorsolateral prefrontal cortex (DLPFC), responsible for self-control, has been frequently shown to be closely associated with procrastination. As the biomarker of self-control, the neuroanatomical changes of the left DLPFC were revealed to predict procrastination robustly (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 provided 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). Despite solid evidence indicating significant associations between self-control and the right DLPFC (Huang et al., 2025; Knoch & Fehr, 2007), Xu and colleagues demonstrated null effects of neuromodulating the right DLPFC had no effects on either value evaluation or emotional regulation in reporting procrastination willingness. Moreover, a substantial amount of neural evidence supported this conclusion that DLPFC is involved in long-term reward evaluation and value encoding via top-bottom self-control 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 causal 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-control (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 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 provide causal evidence clarifying the brain-behavior association of procrastination and revealing how self-control works to shape one's procrastination by manipulating left DLPFC activity in a multiple-session (dose) protocol.

Current Study

To clarify the causal cognitive mechanism of self-control on procrastination, we conducted a double-blind, randomized, multiple-session, placebo-controlled design, with a 2 (active HD-tDCS vs. sham control) $\times$ 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 mechanism of procrastination, we built upon the mixed-effects general linear model and Quasi-Bayesian causal mediation model to test whether the changes in task aversiveness and task outcome value caused by HD-tDCS could predict decreased procrastination. Finally, the follow-up investigation for actual procrastination was conducted after the experiment for 6 months to attempt to test the long-term retention of this neural stimulation effect.

Materials and methods

To promote the reproducibility and transparency, all the data processes and statistics were recorded and made available for tracking by using the “ProjectTemplate” package (see Supplemental Materials, SM). This study fully adhered to CONSORT reporting guidelines, and was preregistered in the OSF repository a priori (DOI 10.17605/OSF.IO/Y3EDT).

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, using 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 SM Methods). Seven participants were eventually excluded for analyses because they voluntarily dropped out before experimental completion. All the included participants were screened for depression and anxiety symptoms (see Tab. 1 [↗](#)).

A full randomized block design was used to assign both groups (active neuromodulation group, NM; sham-control group, SC) (see Fig. 2C [↗](#)). Statistical power was predetermined by G* Power and Power contour estimation at medium effect size ($1-\beta$ err prob = 0.80, $f = 0.25$), which indicated that total sample size at 18 can reach acceptable power (see SM Methods and Fig. S1 [↗](#)). All the 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 here 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 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 $\alpha = 0.890$). 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, $BF_{10} = 0.455$, error = 0.020 %).

To quantify one's daily emotions, the Positive and Negative Affect Schedule (PANAS) developed by Watson et al (1988) was adopted here. This scale consists of two subscales (i.e., positive affect, PA and negative affect, NA), each containing 10 terms, where higher scores indicate stronger affective states (Terracciano et al., 2003 [↗](#)). To control for potential confounding effects of daily emotions, both subscale scores were included as covariates of no interest in the statistical model. Additionally, this tool demonstrated good internal consistency for both sub-scales ($\alpha = 0.801$ for PA, $\alpha = 0.793$ for NA).

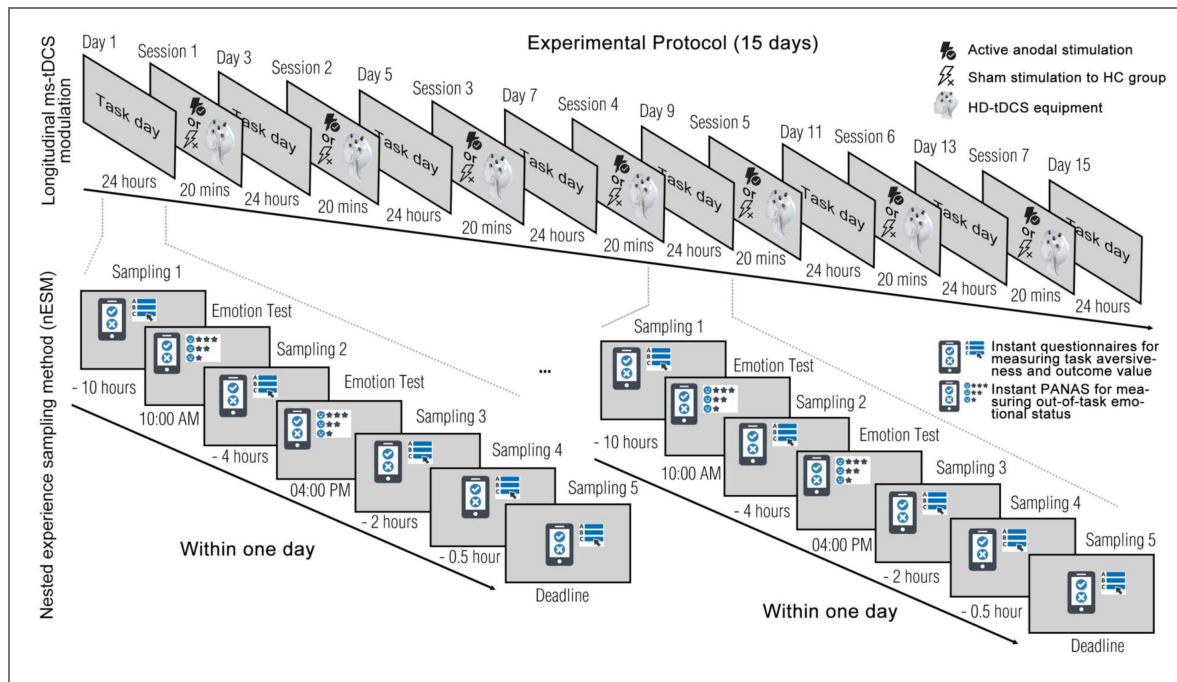


Figure 1. Experimental diagram of this study.

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 interests should be measured by experience sampling methods. The bottom sub-graph reflect 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” symbolize 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).

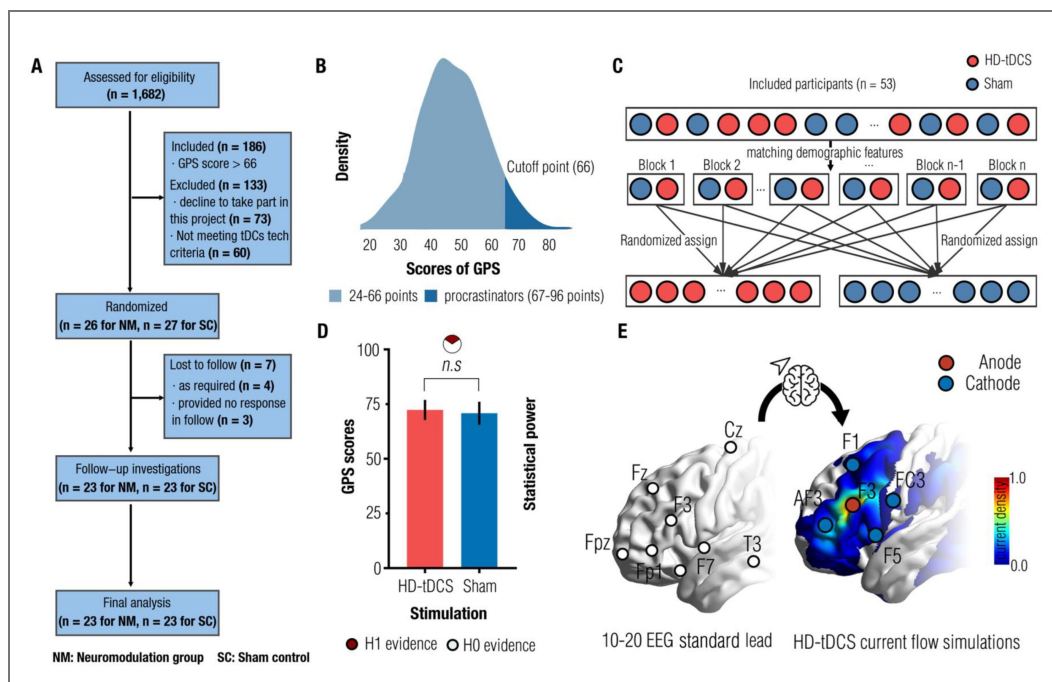


Table 1. Demographic information for included participants.

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)

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 (see Fig. 1 [1](#)). To achieve the first goal, in the first-level longitudinal model, we delivered a 2 (active HD-tDCS neuromodulation group vs. sham-control group) $\times$ 2 (before first HD-tDCS neuromodulation vs. after last HD-tDCS neuromodulation) full factorial design. In detail, the 20-min anodal HD-tDCS neuromodulation targeting the left DLPFC was performed for HD-tDCS group at intervals of 2 days, while the sham-control 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 [1](#)). To examine the long-term effect of tDCS on reducing procrastination, all the participants were required to report task-execution willingness (TEW) (Zhang & Feng, 2020 [2](#); Zhang, Liu, et al., 2019 [3](#)), 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 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.

As for the second aim, in the second-level cross-sectional model, we developed a real-time interactive web-platform sampling system to implement experience sampling method (ESM) for tracking one's real-time evaluation of task aversiveness and task outcome value at five moments within one day needing to do task (see Fig. 1 [1](#), e.g., Day 1, 3, 5). On the basis of the temporal decision model (TDM) of procrastination, the emotional changes in task should be dynamic along with nearing the deadline, and might be hyperbolic discounting manner (Zhang & Feng, 2020 [2](#)). In this vein, the five recording moments of ESM model were determined by using a hyperbolic function toward the deadline, such as moments of 10:00, 16:00, 18:00, 19:30, 20:00 (deadline). Thus, recording moments of tasks were task-specific and participant-specific. 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 system. Before each session of HD-tDCS training, each participant were 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.

Quantification of covariates of interests

Outcome variables of this study are 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 [2](#)). 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). 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 are also interested in re-investigating their actual procrastination by using PR 6 months after the last neuromodulation to test the long-term retention of this neuromodulation effect.

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. The task aversiveness describes how disagreeable one perceives when performing this real-life task (Zhang & Feng, 2020 [2](#)). To quantify one's task aversiveness, participants are required to rate their feelings towards the task by using a 100-point 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”). Outcome value serves as the subjective

benefits for the task outcome brought about by completing the task before the deadline (Zhang & Feng, 2020). Likewise, participants are also required to rate their outcome value by using a 100-point 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”).

HD-tDCS protocol

The HD-tDCS suit (stimulator 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. 2F). Ramp-up and ramp-down duration have been 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 SM Method) (see Fig. 2F). 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/AgCl 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 $\sim 0.5 \text{ mA/cm}^2$ at the central electrode and of $\sim 0.125 \text{ mA/cm}^2$ 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 JASP software (<https://jasp-stats.org/>).

To clarify whether multiple-session HD-tDCS training can reduce actual procrastination, the general mixed-effect linear model (GLMM) was constructed using 2 (active vs. sham) $\times$ 2 (before first neuromodulation vs. after last neuromodulation) full factorial design for procrastination willingness and actual procrastination rates. 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). This analysis was conducted using the “lme4” package. Further, we capitalized on Markov Chain Monte Carlo Generalised linear mixed effects model (MCMCglmm) to overcome pitfalls of null-hypothesis significant test (NHST) that the above analysis might bring out (Hadfield 2009). Further, the post hoc analysis of the above statistics adopted Jeffreys-Zellner-Siow Bayesian examinations (see SM Methods)

(Wagenmakers et al., 2018 [↗](#)). Instead of continuous outcomes, we also conducted a between-group comparison for the number of tasks procrastination by using the χ^2 test with ϕ correction or Fisher exact test. Likewise, the Bayesian estimations were carried out as well. Regarding the 6-month follow-up investigation, this GLMM was also built to examine the long-term retention of neuromodulation on reducing procrastination. Also, these statistical models were validated using Bayesian factor analysis to provide more robust statistical evidence. In addition, we also capitalized on the stochastic gradient descent Cox model by using “BigSurvSGD” package to estimate the effect of neuromodulation on procrastination probability. To minimize the risk of a false-positive ratio, these statistical results have been validated by adjusting for the baseline effect from regressing pre-neuromodulation procrastination and other covariates to post-neuromodulation procrastination (see SI Results).

To reveal the potential mechanism of this effect, all statistics mentioned above were redone for the evaluation of task aversiveness and outcome values. On the basis of the temporal decision model of procrastination proposed by Zhang et al (2020) [↗](#), the task aversiveness evoked by executing a task was quite dynamic, and would increase dramatically once reaching the deadline, with the peak task aversiveness for the nearing deadline (and vice versa) (Zhang & Feng, 2020 [↗](#)). In this vein, to quantify the task aversiveness with consideration for its dynamics, the model-free area under the curve (AUC) was estimated. We assumed that the task aversiveness scores that derived from continuous time points (being far away from the deadline to the nearing deadline) formed a closed interval, and thus allowed for the estimation for AUC by using a definite integral:

$$\int_a^b f(x)dx, y = f(x) \in [a, b]$$

Here, by using ESM, task aversiveness was recruited at progressive time points close to the deadline, and thus formed an available function interval. Thus, the AUC represented the extent to which task aversiveness has changed in approaching the deadline, with a high AUC for low-level task aversiveness. As for the task outcome value, it was theoretically posited as a relatively stable evaluation of the task. In this vein, it was quantified by the self-reported 100-point scale after neuromodulation at least 12 hours later, to ensure no online effect.

Further, taking into account the longitudinal repeated measured data, the joint model of longitudinal and survival data (JM-LAD), in conjunction with a machine learning algorithm, was adopted, which has been reported to outperform mixed-effects models. As referred to in the existing study, we thus assumed that the procrastination probability (Pr(P)) could be determined by the joint statistic ($m_i(t)$) as follows:

$$h_i(t) = h_0(t)\exp\{\gamma^T w_i + \alpha m_i(t)\}, t > 0$$

where γ^T represented vector of regression coefficient, and α reported the fitting coefficient by longitudinal model. To determine the longitudinal component of this joint model, the vast majority of prevailing machine learning regression algorithms were initially placed into model, including decision tree regression, Boosting regression, random forest regression and support vector regression. The final model would be determined according to minimum normal mean square error (NMSE).

To ascertain the neurocognitive mechanism of tDCS in reducing procrastination, the Quasi-Bayesian causal 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 causal 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., averaged causal mediation effects, δ ; averaged direct effects, 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).

Results

Blinding

In both groups, almost all participants reported perceiving acceptable pain stemming from current stimulation and believed they were receiving treatment (91.30% (21/23) for active neuromodulation group (NM), 86.95% (20/23) for sham control group (SC), $\chi^2 = 0.224$, $p = .636$). In addition, statistical models excluded Session 1 and Session 4 because participants reported additional side effects in both groups (see SI Result and Tab. S1).

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

To identify whether ms-tDCS targeting the left DLPFC can alleviate procrastination willingness and actual procrastination, a linear mixed-effects model with Scatterthwaite algorithm was built, with task-execution willingness and actual procrastination rates (PR) as outcomes, respectively. Procrastination willingness changes for each session have been described in the Fig. 3A. Results showed a significant two-way interaction (NM v.s. SC $\times$ before neuromodulation vs. after neuromodulation) for procrastination willingness (Task-execution willingness, $F_{(1, 269)} = 43.86$, $p = 1.94 \times 10^{-8}$, $\eta_p^2 = .21$), and further demonstrated a significantly increased task-execution willingness (i.e., decreased procrastination willingness) after neuromodulation in the active neuromodulation group, but no such effects in the sham control group (Task-execution willingness, NM-before: 35.65 ± 30.20 , NM-after: 80.43 ± 19.92 , $F_{(1,44)} = 76.46$, $p < .001$; SC-before: 37.57 ± 26.46 , SC-after: 47.35 ± 30.49 , $F_{(1,44)} = 1.35$, $p = .251$) (Fig. 3B-C). A linear uptrend for task-execution willingness was observed across multiple sessions (Fig. 3D; $p < .01$, Mann-Kendall test). Meanwhile, changes to procrastination rates across all the sessions have been detailed in the Fig. 3E. A statistically significant interaction effect was identified ($F_{(1, 269)} = 35.54$, $p = 1.90 \times 10^{-10}$, $\eta_p^2 = .17$), and the post-hoc analysis showed decreased actual procrastination rates after ms-tDCS in the active neuromodulation group, but no such changes found in the sham-control group (actual PR, NM-before: 43.26 ± 39.09 , NM-after: 0.00 ± 0.00 , $F_{(1,44)} = 58.06$, $p < .001$; SC-before: 46.47 ± 40.75 , NM-after: 33.34 ± 37.82 , $F_{(1,44)} = 1.28$, $p = .26$) (Fig. 3F-G). Similarly, a significant downtrend for procrastination rates across all the sessions was identified (Fig. 3H; $p < .01$, Mann-Kendall test).

To obtain more solid statistical evidence, we further utilized a Bayesian mixed-effects model by using Markov Chain Monte Carlo (MCMC) sampling and revealed the same findings (see SI Results and Fig. S2–5). Furthermore, the χ^2 test of $R \times C$ contingency table was conducted for the frequency of procrastination, as this outcome is an ordinal variable. A significant reduction in group-averaged procrastination frequency was found in the active neuromodulation group after 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), $\chi^2 = 10.08$, $p < .001$). In addition, using a stochastic gradient descent algorithm in the Cox model, procrastination probability (risks) for procrastinators in the neuromodulation group was found to be reduced by 5.12 times compared to the sham-control group after ms-tDCS neuromodulation (OR (Odds Rates) = 5.12, 95% CI: 1.97 – 13.26, $p < .001$). In brief, these findings provided evidence to support the claim that ms-tDCS neuromodulation targeting the left DLPFC can be an effective way to reduce both procrastination willingness and actual procrastination through its long-term after-effects.

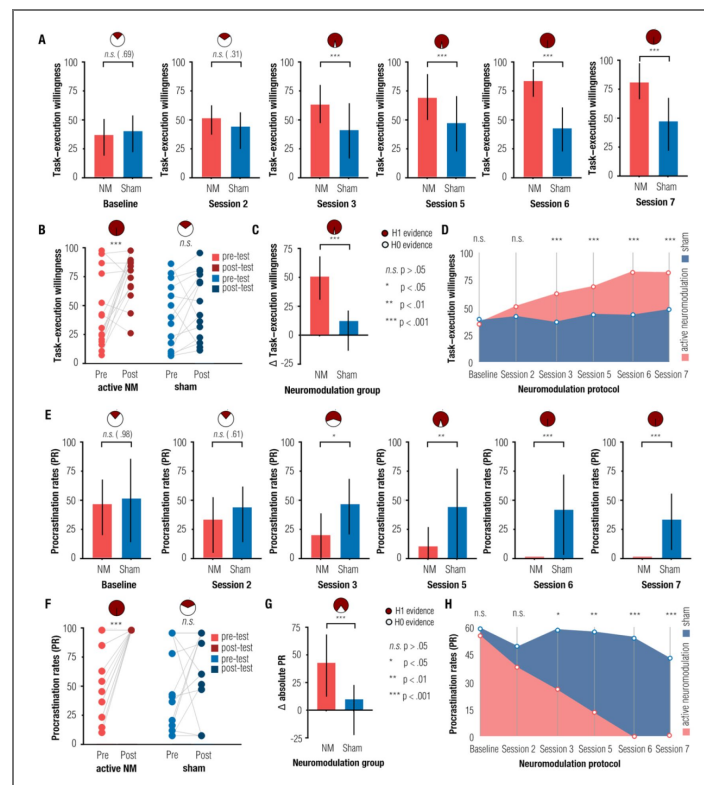


Figure 3. Results of neuromodulation effects to task-execution willingness and procrastination rates (PR).

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 group. C plots the changes of task-execution willingness for both group 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 group. G plots the absolute changes of PR for both group 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 represent 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).

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 2 (NM vs. SC) $\times$ 2 (before first neuromodulation vs. after last neuromodulation) mixed linear model to examine the effects of ms-tDCS on changes in task aversiveness and task outcome value. Task aversiveness changes across all the sessions have been shown in the Fig. 4A and 4C. Statistical analysis demonstrated significant decreases in task aversiveness and increases in task outcome value via ms-tDCS in the neuromodulation group but not in the sham-control group (Task aversiveness, $F_{(1, 269)} = 45.92$, $p = 7.73 \times 10^{-11}$, $\eta_p^2 = .13$, NM-before (AUC): 1.13 ± 0.53 , NM-after (AUC): 1.95 ± 0.85 , $F_{(1,44)} = 11.44$, $p < .005$; outcome value, $F_{(1, 269)} = 45.61$, $p = 8.87 \times 10^{-11}$, $\eta_p^2 = .19$, NM-before (AUC): 35.86 ± 27.82 , NM-after (AUC): 73.08 ± 23.33 , $F_{(1,44)} = 18.07$, $p < .001$) (see Fig. 4B and 4D; SI Results and Fig. S6–9). 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, it provides causal evidence clarifying that neuromodulation to self-control capacity reduces task aversiveness and enhances task-outcome value simultaneously.

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 propose that these changes, driven by enhanced self-control, account for how neuromodulation reduces real-world procrastination behavior. We utilized a generalized linear model to relate 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 ($\Delta_{\text{task-execution willingness}}$) ($\chi^2 = 15.95$, $p < .01$, $R^2 = .40$; $\Delta_{\text{Outcome value}}: \beta = 0.61$, S.E. = 0.12 , $p < .001$, 95% CI: $0.373 - 0.860$), 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, increased outcome value ($\Delta_{\text{Outcome value}}$) significantly predicted one's real-world procrastination behavior, whereas decreased task aversiveness showed no significant predictive effect (see Tab. 2). The Bayesian statistical evidence was further obtained to substantiate the above findings (Null model: $\text{BF}(M|\text{data}) = 7.44 \times 10^{-4}$, $\text{BF}_{10} = 1.00$; Model of including task outcome value: $\text{BF}(M|\text{data}) = 0.77$, $\text{BF}_{10} = 1.03 \times 10^{-4}$; Model of including task aversiveness: $\text{BF}(M|\text{data}) = 9.42 \times 10^{-5}$, $\text{BF}_{10} = 1.26$). In addition, by adopting the joint model of longitudinal and survival data in a machine learning scheme and linear probability model, we also revealed the predictive role of increased task outcome value to less procrastination when the longitudinal processes (sessions) were taken into account (see SI Results and Tab. S3–6). On balance, these findings provide evidence that reveals that the left DLPFC could strengthen one's self-control to increase task outcome value so as to decrease procrastination.

Increased task outcome value plays causal role to explain why ms-tDCS reduces procrastination

To clarify the causal neurocognitive mechanism of procrastination, the Quasi-Bayesian causal mediation analysis was undertaken by using White's heteroskedasticity-consistent estimator, with increased task outcome value as a mediated variable. Results showed the significant causal mediated role of increased task outcome value in increasing the task-execution willingness ($\delta = 21.73$, $p < .01$; $\zeta = 11.25$, $p = .07$, $p = 32.99$, $p < .01$, simulation = 1,000; see Fig. 5A) and real-world procrastination ($\delta = 30.75$, $p < .01$; $\zeta = 3.05$, $p = .52$, $p = 33.81$, $p < .01$, simulation = 1,000; see Fig. 5B), as caused by ms-tDCS neuromodulation. To ensure the robustness and specificity of these findings, the sensitivity analysis was implemented by changing sampling parameters and outcome variables. Results showed that the above findings were replicated when the bootstrap sampling

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). B plots the changes of AUC of task aversiveness for both group 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 group 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 represent 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).

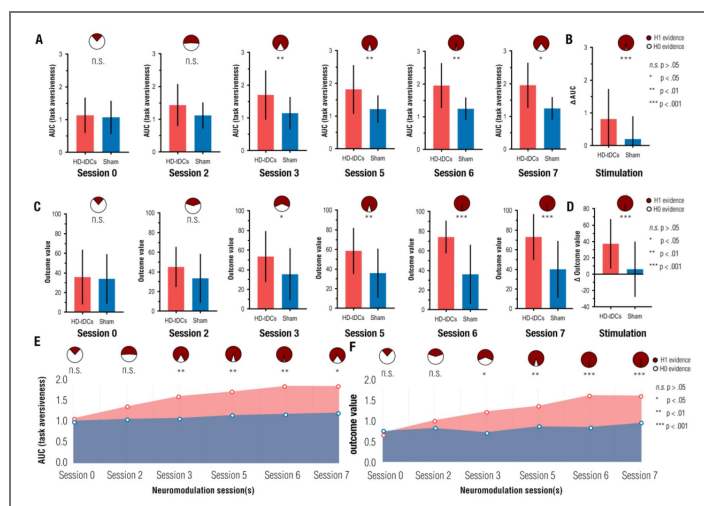


Table 2. Summary for general linear model in predicting changes of task aversiveness and outcome value to actual procrastination.

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	.29
Δ outcome value	0.85*	0.42	0.04	2.34	

algorithm was used to change parameters, and thus confirm the robustness of this model (see SI Results and Tab. S6–7). Likewise, the results of the control analysis further validated the specificity of these findings by showing a null causal mediated effect of this model to predict one's task aversiveness (see SI Results and Tab. S8). In summary, these findings revealed neurocognitive pathway underlying procrastination: the self-control governed by left DLPFC uniquely increases task-outcome value to reduce one's procrastination willingness and real-world procrastination behavior.

Long-term effects of ms-tDCS

We have also attempted to conduct a follow-up investigation to test the long-term retention 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 ($N_{\text{NM}} = 22$, $N_{\text{SC}} = 23$). Thus, the GLMM 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 ($\beta = 16.5$, $\text{SE} = 9.9$, $p = .049$). Simple-effect model demonstrated a decrease in actual procrastination rates in the active neuromodulation group after last stimulation for 6 months compared to baseline ($\beta = -22.05$, $\text{SE} = 10.0$, $p = .038$, Tukey correction; NM-before: 40.68 ± 37.96 , NM-after_{6-months}: 18.63 ± 29.80), and revealed null effects in the SC group ($\beta = 1.26$, $\text{SE} = 9.78$, $p = .99$, Tukey correction; SC-before: 46.47 ± 40.75 , SC-after_{6-months}: 47.73 ± 39.18) (see Fig. 6).

Likewise, the parallel Jeffreys-Zellner-Siow Bayesian factor model was constructed to provide solid statistical evidence. Results demonstrated the robustness of these findings by showing the acceptable Bayesian evidence strength to support the above statistical inferences (NM group: $\text{BF}_{10} = 2.88$, error = 0.76 %, $P_{(\text{inc}|\text{data})} = 0.74$; SC group: $\text{BF}_{10} = 0.29$, error = 0.98 %, $P_{(\text{inc}|\text{data})} = 0.22$). Furthermore, the difference of group-averaged procrastination frequency was found significant statistically between pre-neuromodulation and after-neuromodulation for 6 months by using the χ^2 test in the NM group ($\chi^2 = 3.30$, $p = .035$, NM-before: 68.19% (15/22), NM-after_{6-months}: 40.91% (9/22)), while observed no significant changes in the SC group ($\chi^2 = 0.11$, $p = .74$, SC-before: 69.56% (16/23), SC-after_{6-months}: 73.91% (17/23)). Taken together, this study revealed the long-term retention that was produced by ms-tDCS neuromodulation to reduce procrastination.

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 the decreased procrastination. On balance, our findings clarified the neurocognitive mechanism of procrastination by showing that self-control could increase task-outcome value so as to reduce procrastination. In addition, this study provided an effective way to reduce actual procrastination by using ms-tDCS neuromodulation.

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. Neurobiological substrates of procrastination have been investigated in recent years, and 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-control brain network that was constituted by the left DLPFC were found to link with more procrastination behaviors (Chen & Feng, 2022; Zhang et al., 2016). Notwithstanding that, it still remains unclear to claim their causal brain-behavior relationship - that is - no known evidence existed to clarify whether changes of the left DLPFC lead to procrastination or vice versa. The current study demonstrated the causal

Figure 5. Results of Quasi-Bayesian causal 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 = averaged direct effect, ACME = averaged causal mediated effect, CI = confident interval.

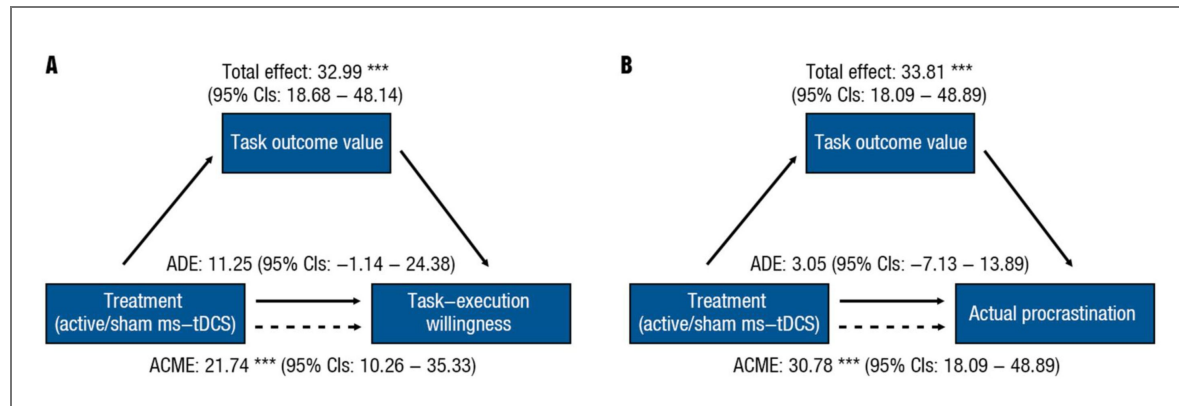
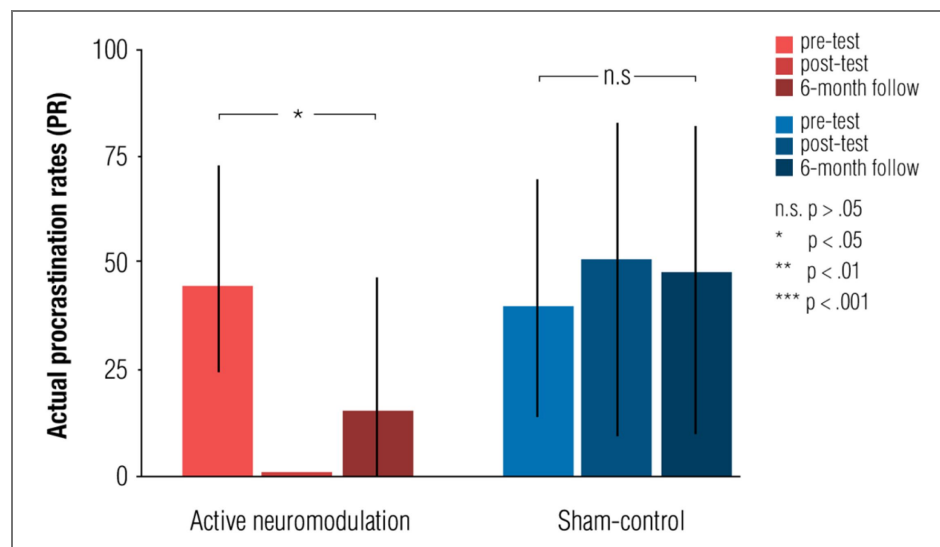


Figure 6. Changes of actual procrastination rates among pre-test, post-test and 6-month follow-up for active neuromodulation group and sham-control group.

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 means to re-investigate the actual procrastination rates after last neuromodulation for 6 months.



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 associated with many aspects of self-control communities, 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-control which modulates value signals (Hare et al., 2009 [↗](#); Harris et al., 2013 [↗](#)). Meanwhile, procrastination has been argued to be the consequence of self-control failures and self-regulation failure for a long time (Ariely & Wertenbroch, 2002 [↗](#); Rebetz 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 self-control network were interpreted as the phenotype of reduced self-control, making individuals more prone to procrastinate tasks, as reported in existing literature (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 was 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 [↗](#)). On the other hand, 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, Liu, et al., 2019 [↗](#)). In this vein, this study advanced our understanding of the neurocognitive mechanism of procrastination by showing that the cortical excitability of the DLPFC produced by active neuromodulation could boost self-control to increase task outcome value so as to reduce procrastination behavior.

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 neuromodulation the current study performed has a remarkable advantage in reducing procrastination - that is, the intention-action gap was attenuated so as to overcome procrastination behavior effectively. 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 multiple-session tDCS stimulation could boost cumulative effects of consolidation for activity-dependent 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, it led us to presume that the 6-

month long-term retention of the ms-tDCS effect on reducing procrastination might be attributed to long-term neuroplastic changes in the dlPFC. 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 limited size of the sample and the underrepresented demographic characteristics (e.g., over 80% for females), it warrants caution in generalizing these findings elsewhere. 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 domain-specific for procrastination. Moreover, this study did not collect data on participants' trait procrastination, obscuring the conclusion for whether effects of this neuromodulation were limited in those tasks or not. Finally, given the absence of cathodal HD-tDCS stimulation as a contrast condition in causal inference, it warrants caution that the increased DLPFC excitability may not be the exclusively neural mechanism for procrastination.

In conclusion, this study 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 found that the ms-tDCS neuromodulation could decrease task aversiveness and increase task outcome value while, and further demonstrated that the increased task outcome value could predict decreased procrastination through enhancing self-control. In this vein, the current study moves forward our understanding of neurocognitive mechanism of procrastination by showing the prominent role of increased task outcome value in reducing procrastination. Also, it provides an effective method for intervening in human procrastination.

Data and Code 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 for reproducing these findings are available at Open Science Framework (OSF) repository (10.17605/OSF.IO/Y3EDT). 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 Prisma.

Supplementary information

SI Methods

To reach a higher reproducibility and transparency, “ProjectTemplate” package of R Studio (<http://projecttemplate.net/index.html> [↗](#)) was drawn upon for recording and tracking all of processes this study has done in a standardized document. This final record would be released once all the processes and statistics have been done. This study fully adhered to CONSORT reporting guideline for providing comprehensive reports for these findings. The CONSORT Checklist and CONSORT Diagram have been included below.

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 (King et al., 2019 [link](#)). 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 (a err prob = 0.05, $1 - \beta$ err prob = 0.80, $f = 0.25$) by building a repeated measures, within-between, and interaction effect ANOVA model (Faul et al., 2007 [link](#)). In this vein, the medium effect size can be reached once total sample size $n = 18$, noncentrality $\lambda = 15.75$, 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 (Baker et al., 2020 [link](#)). 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 [link](#)). More detail for how to estimate and plot this map can be found elsewhere (Baker et al., 2020 [link](#)).

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/> [link](#)), 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-adjusted 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 pronounced 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 (A) 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.

Jeffreys-Zellner-Siow Bayesian examinations

Also, to make the claims for the null inferences more robust and reliable, the equivalence test (ET) and Bayesian estimations were jointly adopted. ET needs to be an estimator of the smallest effect size of interest (SESOI), describing whether the null effect falls within this interval, with overlapping for significant null effects. Thus, “TOSTER” package of R was used to test the null hypothesis by pre-determining the interval as ± 0.5 (default). Further, to confirm the null effect, Bayesian estimations were implemented in the BEST (Bayesian estimation supersedes the t-test) package of R. Building upon MCMC (Markov Chain Monte Carlo), subsequent distributions were defined as having normality with maximum variance, and determined whether the 90 % High Density Interval (HDI) overlapped in the region of practical equivalence (ROPE) for inferring the null effect. Likewise, the “BestCMCM” function was used for this analysis, adopting a default interval of ± 0.40 .

There are no denying this fact that the conventional Null-hypothesis Significant Test (NHST) has ineluctable imperfections, particularly in the concerns on the misuse of p-value (Anderson et al., 2000). A growing number of studies have promoted this idea that the Bayesian model was the most attractive alternative for the statistical analysis (Wasserman, 1997; Montagnini et al., 2007). Compared to the NHST method, the Bayesian model can be profit from the apriori null hypothesis, thus balancing the fits between the apriori hypothesis and aposteriori cumulative evidence (Wasserman, 1997). In addition, it is of also attractive that the Bayesian model has the strength for the liberal precondition and sensitive detection power (Benavoli et al., 2016; Nathoo and Masson, 2016). In brief, we predefined the apriori distribution for the null hypothesis using the revised Cauchy profile (Wagenmakers et al., 2018); then, the aposteriori evidence was obtained that relying on this Cauchy distribution; finally, canonical Bayes algorithm was utilized to estimate the Bayes factor (BF) for providing the power of evidence on the null-hypothesis or alternative-hypothesis:

$$p(D|\theta) = \prod_{n=1}^N p(x_n|\theta) \quad p(\theta|D) = \frac{p(D|\theta)p(\theta)}{\int p(D|\theta)p(\theta)d\theta}$$

As these functions showed, the left one is the algorithm for the apriori information and the right one for the estimation of the aposteriori evidence. Thus, all the neural markers derived from these significant clusters and topological dynamics of brain network were progressively putted into this model to validate our findings. These processes were implemented in the JASP package (x64 v0.9.0.1; <https://jasp-stats.org/>; JASP team 2017).

SI Results

Side effects

In the Session 1, a significant unexpected side 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 (Ryan et al., 2010, Stone et al., 2012). 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 additional effects occur here. The χ^2 test was used to examine whether the additional side effect reports posed significantly differences between groups across all the sessions (see Tab. S1).

	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
χ^2	-	.023	-	.200	.026	-	-	.500

Table S1. The number of reporting additional side 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 additional side effects occur. 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, whilst 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.

Sensitivity analysis for interaction effects

To examine sensitivity of main findings regarding Group*Experiment interaction, we reanalyzed interaction effect by removing regressors of PANAS scores in the mixed-effect linear model. This replicated these findings by showing statistically significant interaction effect between Group (NM vs control) and training manipulation (active stimulation vs sham stimulation) on task-execution willingness changes ($\beta = -25.8$, $SD = 14.7$, $p < .05$) and procrastination rate changes ($\beta = -25.8$, $SD = 14.7$, $p < .05$).

Bayesian estimations for statistics upon the neuromodulation effects

Here, the Bayesian inference was used to reveal whether neuromodulation can alleviate one's procrastination problem by “MCMCglmm” package. Likewise, the significant increments of task-execution willingness and actual PR were found in NM group after neuromodulation but it was found null in the SC group (task-execution willingness, post mean distribution = -21.56 , 95 % CI: $-27.89 - -15.54$, $pMCMC < .001$; actual PR, post mean distribution = 24.82 95 % CI: $16.38 - 33.23$,

pMCMC < .001). Also, the Jeffreys-Zellner-Siow Bayesian factors for the χ^2 test was calculated to provide evidences for the changes of procrastination frequency before and after neuromodulation. Results demonstrated the significant effect to reduce procrastination by showing the significant decreasing procrastination frequency after neuromodulation in NM group but not SC group (BF_{10} hypergeometric = 76.84; > 10 means a strong evidence to support alternative hypothesis).

Bayesian estimations for statistics upon the neuromodulation effects towards task aversiveness and outcome value

Likewise, the Bayesian inference was used to validate the neuromodulation effects towards the changes of task aversiveness and outcome value. Results showed the significant reduction of task aversiveness and increased outcome value in NM group after neuromodulation but null findings in the SC group (task aversiveness, post mean distribution = - 0.48, 95 % CI: -0.62 – -0.34, pMCMC < .001; outcome value, post mean distribution = - 20.89, 95 % CI : -26.98 – - 14.52, pMCMC < .001).

Results of joint model of longitudinal and survival data

taking into account for the longitudinal repeated measured data, the joint model of longitudinal and survival data (JM-LAD), in conjunction of machine learning algorithm, was adopted. Compared to standard JM, this one was found to outperform for analysis panel data by reaping huge fruits from machine learning (ML) algorithms in longitudinal sub-model (Wen, 2015). Thus, the normal mean standard error (NMSE) was used to determined which ML algorithm is best for this sub-model with lower NMSE value for better model fitting performance:

$$NMSE = \frac{\sum_{j=1}^j \sum_{k=1}^k [f(j, k) - \hat{j}(j, k)]^2}{\sum_{j=1}^j \sum_{k=1}^k [f(j, k)]^2}$$

Results showed that K-Nearest Neighbor (KNN) was the best longitudinal sub-model as the minimum NMSE (see Tab. S3 [↗](#)).

	Classification Tree	Bagging	Random Forest	KNN	SVM
NMSE	3.260	0.853	0.788	0.374	0.602

Table S3. Summary of NMSE for all the machine learning algorithms. KNN = K-nearest neighbor; SVM = support machine learning

By using KNN, the Outcome value of task was found to predict whether one would procrastinate significantly, whilst it showed null effects by using task aversiveness (see Tab. S4 [↗](#)–[5](#) [↗](#)).

	Longitudinal Process			Event Process		
	Intercept	Time	Time * Group	Group	Associ	AIC/BIC
Value	1.012	0.192	-0.142	-1.643	0.773	378.54/
P-value	< .001	< .001	< .001	0.0012	0.0416	405.96

Table S4. Summary of JM for longitudinal process and event process by using outcome value as independent variable.

	Longitudinal Process			Event Process		
	Intercept	Time	Time * Group	Group	Associ	AIC/BIC
Value	30.59	8.11	-7.68	-2.16	-0.01	2016.66/
P-value	< .001	< .001	< .001	0.0007	0.88	2044.09

Table S5. Summary of JM for longitudinal process and event process by using task aversiveness as independent variable.

Given the longitudinal effects in multiple neuromodulation sessions, we capitalized on the joint model of longitudinal machine learning and survival data for the fits of both task aversiveness and outcome value to actual procrastination behavior across all the sessions. Outcome value of task was found to predict whether one would procrastinate significantly, whilst it showed null effects by using task aversiveness (Outcome value, $\beta = 0.77$, OR = 2.15, $p < .05$, method: pseudo-adaptive Gauss-Hermite; Task aversiveness, $\beta = -0.001$, OR = 0.99, $p = .88$, method: pseudo-adaptive Gauss-Hermite) (see Tab. S3 [4](#)–[5](#)).

Results of linear probability panel model

Given the panel data pattern (7 longitudinal sessions $\times$ 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, $p < .05$; overcome 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 ($\chi^2 = 22.75$, $p < .0001$). Also, the Hausman test was done for determining what types of models would be accepted. Results indicated that the random effect model is unstability ($\chi^2 = 12.92$, $p < .0015$). Thus, the fixed effect model controlling both time and individual variants was adopted as final one.

Results of sensitivity analysis

To examine the robustness and specificity of this causal medication 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 Tab. S6 [4](#)–[7](#)).

	β	S.E	P value	T value	adj R ²
Task aversiveness	0.39	0.11	0.404	1.123	.13
Outcome value	3.86 ***	3.44	0.0003	3.721	

Table S6. Summary for linear probability panel model in predicting task-execution willingness by using task aversiveness and outcome value. S.E. means standard error.

	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 causal mediation model in predicting task-execution willingness by using treatment (active ms-tDCS vs sham) from mediated effect of increased task outcome.

Further, we inputted the age and gender as outcome variables into this model for testing whether this causal 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 Tab. S8 [↗](#)).

	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 causal mediation model in predicting actual procrastination by using treatment (active ms-tDCS vs sham) from mediated effect of increased task outcome.

Results of GLMM adjusted for baseline

To minimize false-positive risks, rather than 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 pre-neuromodulation 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 GLMM.

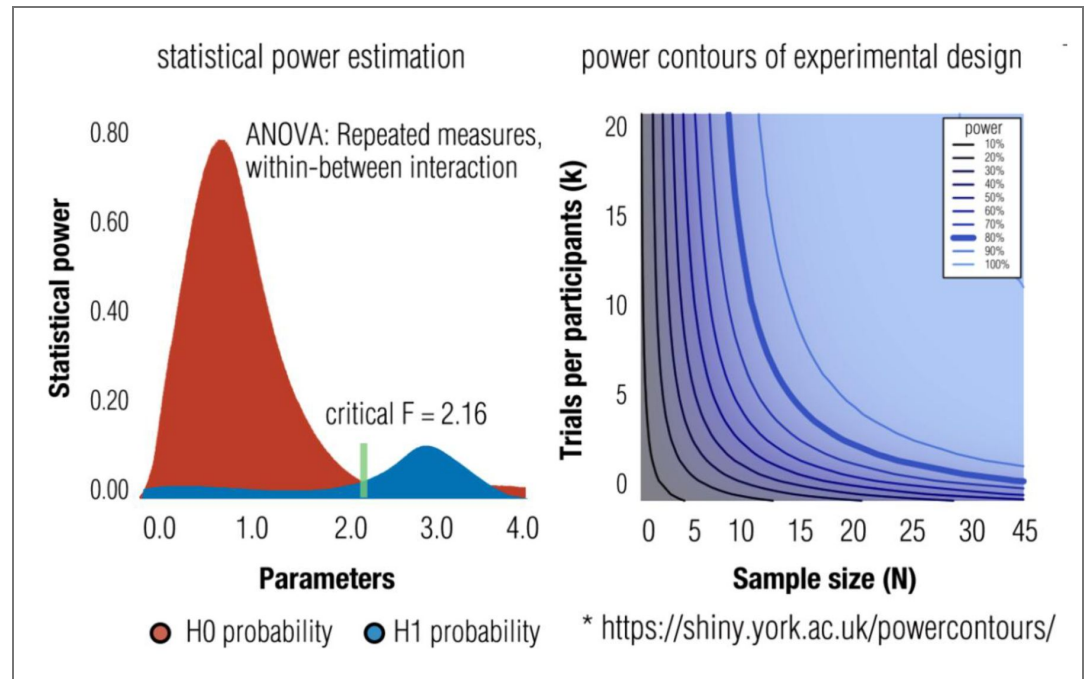


Figure S1. Statistical power estimation by using ANOVA model and power contours according to experimental design

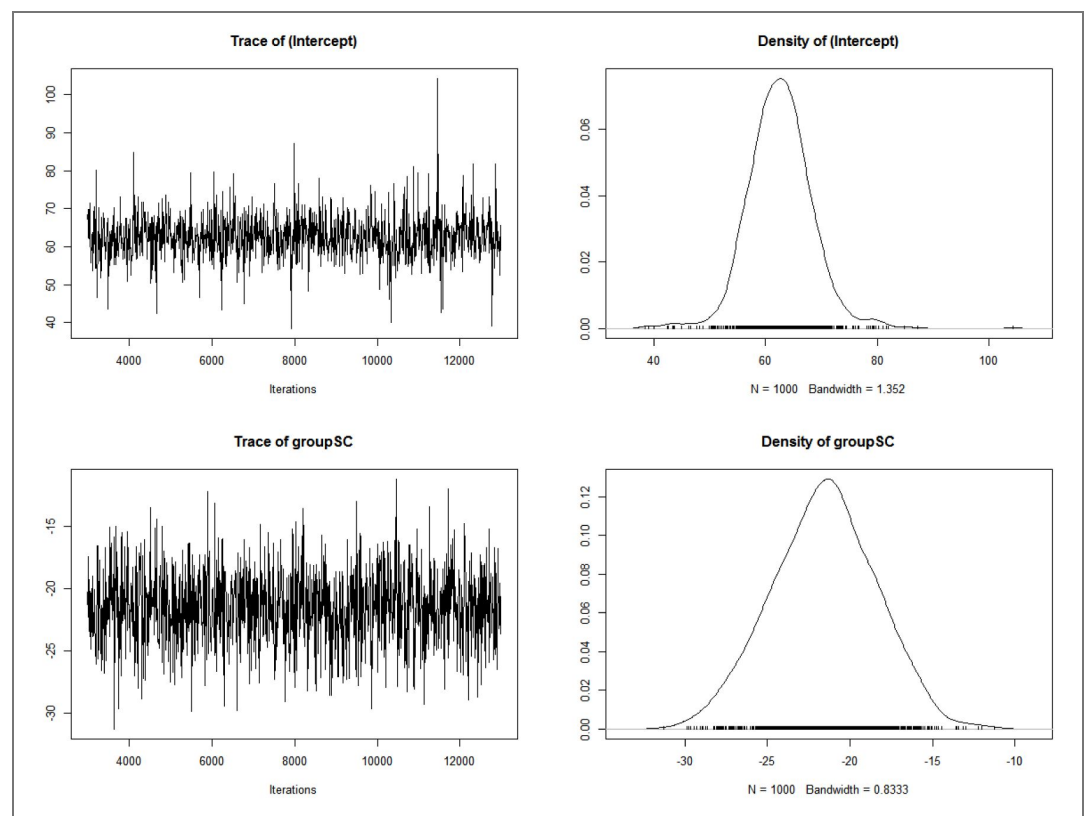


Figure S2. Density of intercept and group-between effect from MCMC sampling for the task-execution willingness.

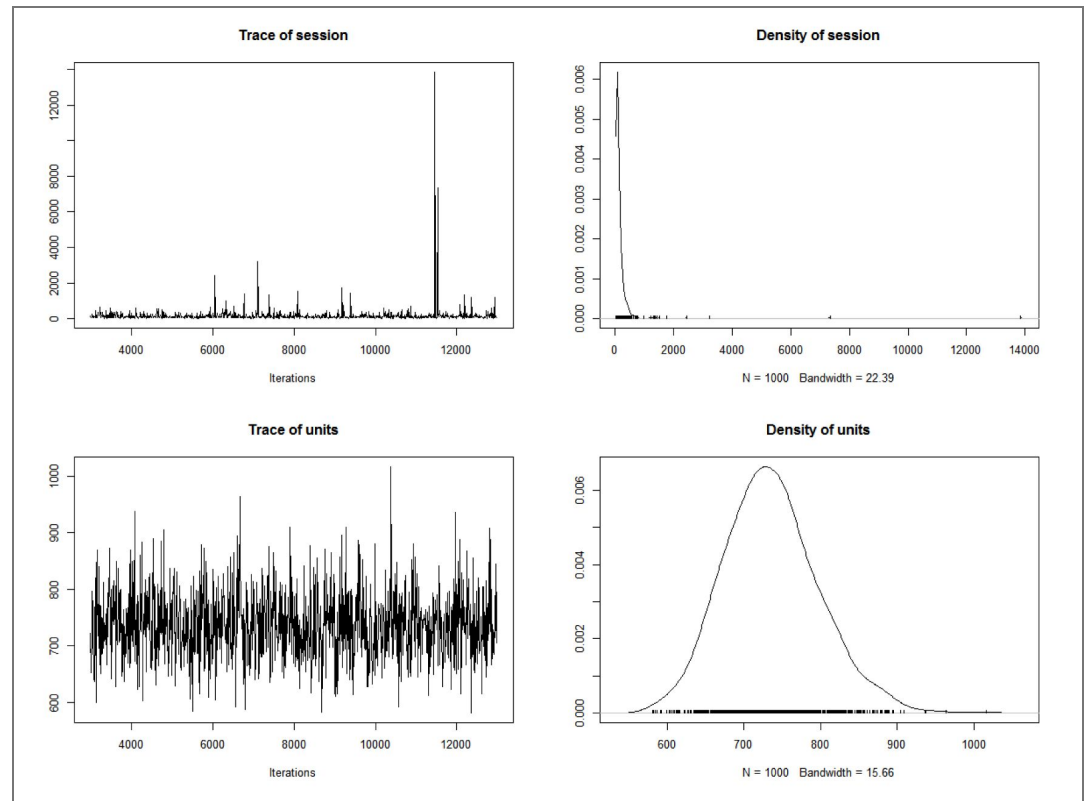


Figure S3. Density of intercept and individual-within effect from MCMC sampling for the task-execution willingness.

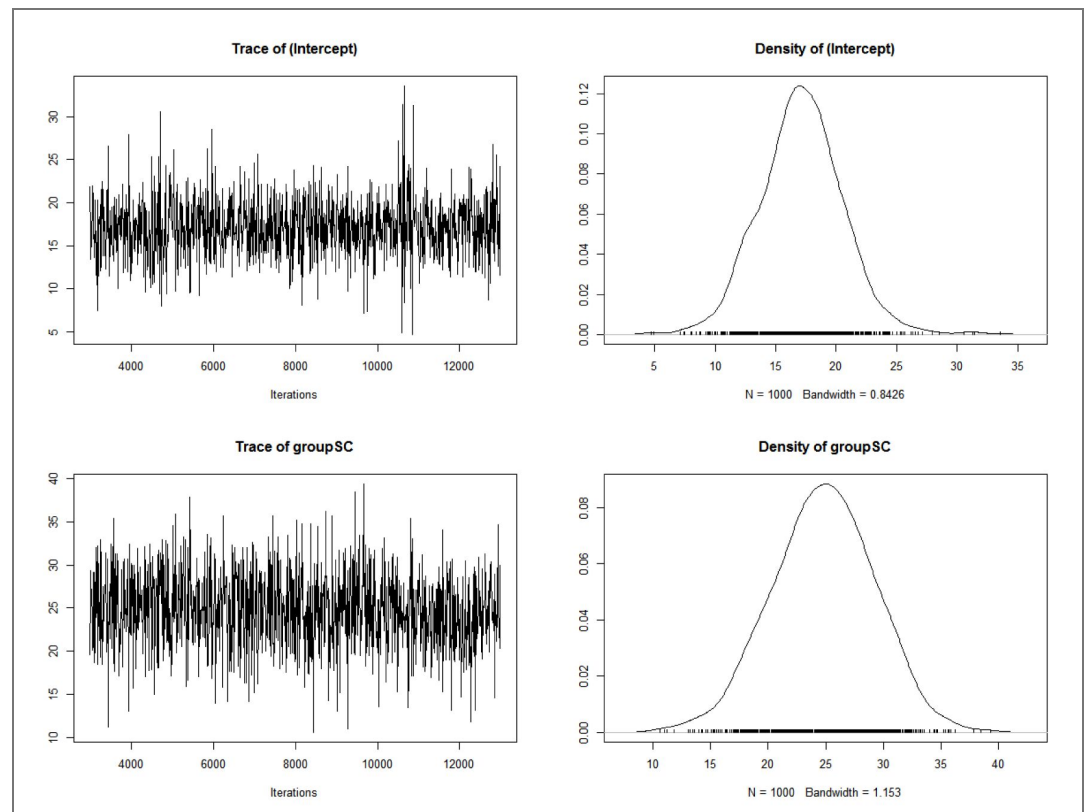


Figure S4. Density of intercept and group-between effect from MCMC sampling for the actual PR.

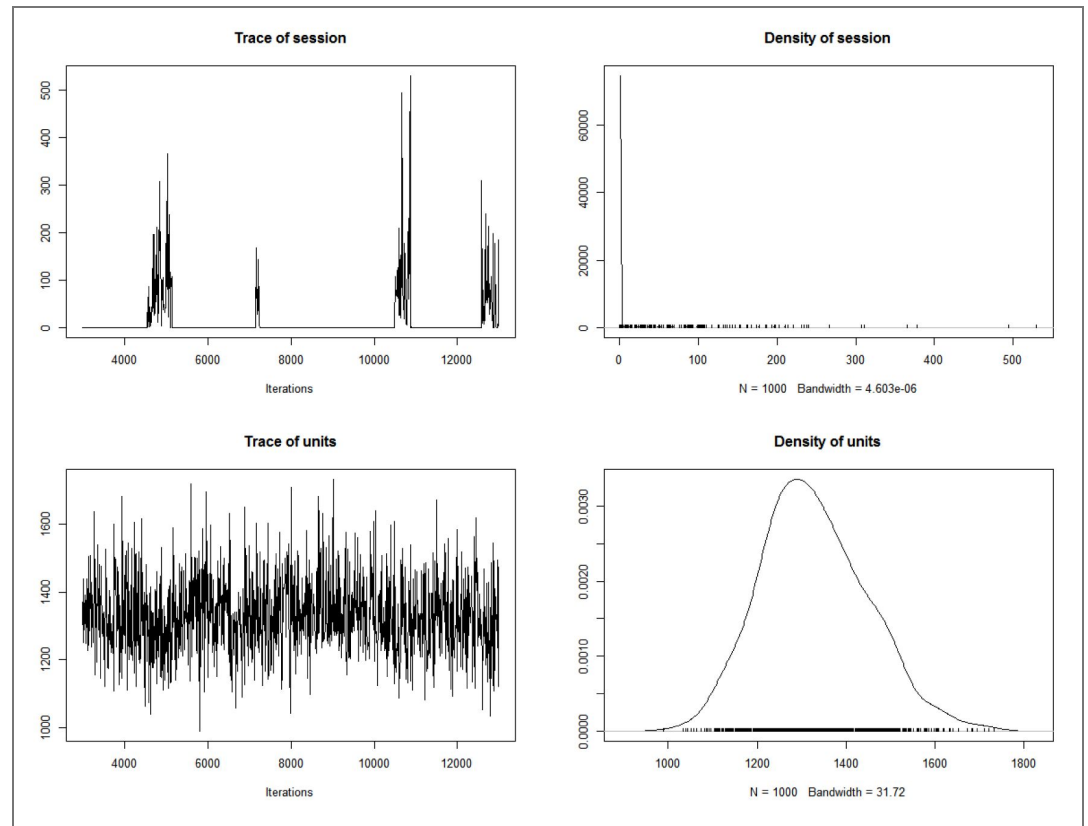


Figure S5. Density of intercept and individual-within effect from MCMC sampling for the actual PR.

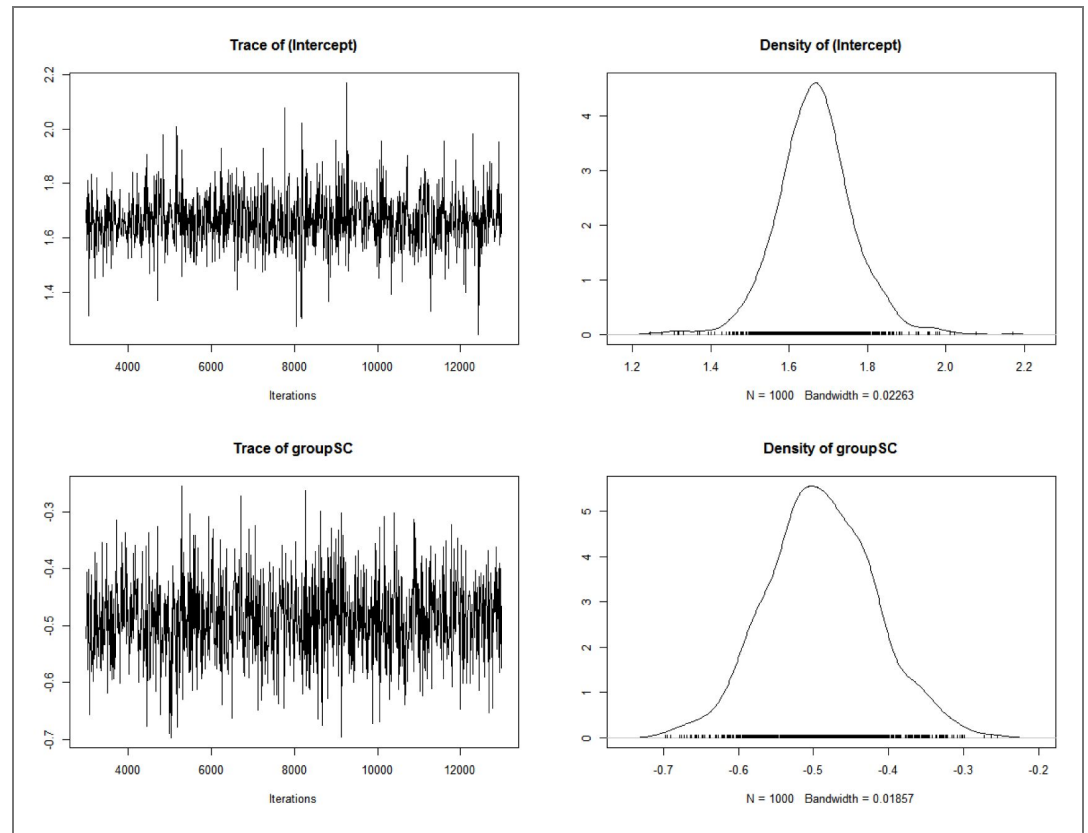


Figure S6. Density of intercept and individual-within effect from MCMC sampling for the task aversiveness.

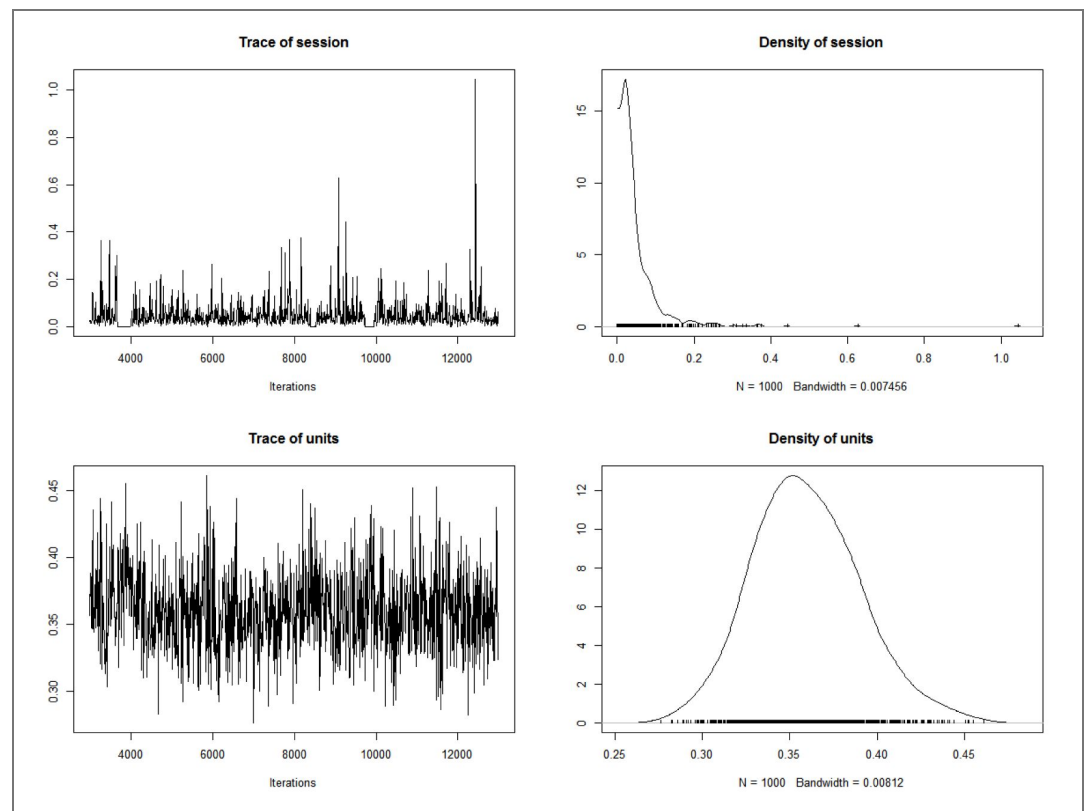


Figure S7. Density of intercept and group-between effect from MCMC sampling for the task aversiveness.

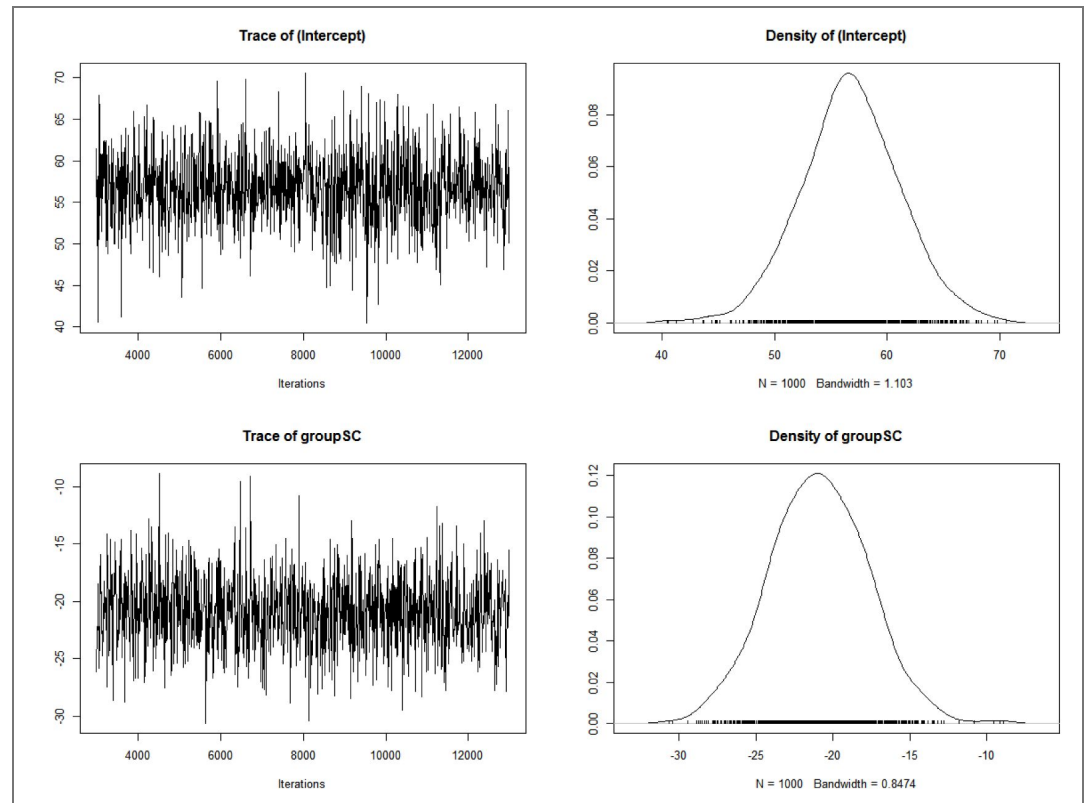


Figure S8. Density of intercept and group-between effect from MCMC sampling for the outcome value.

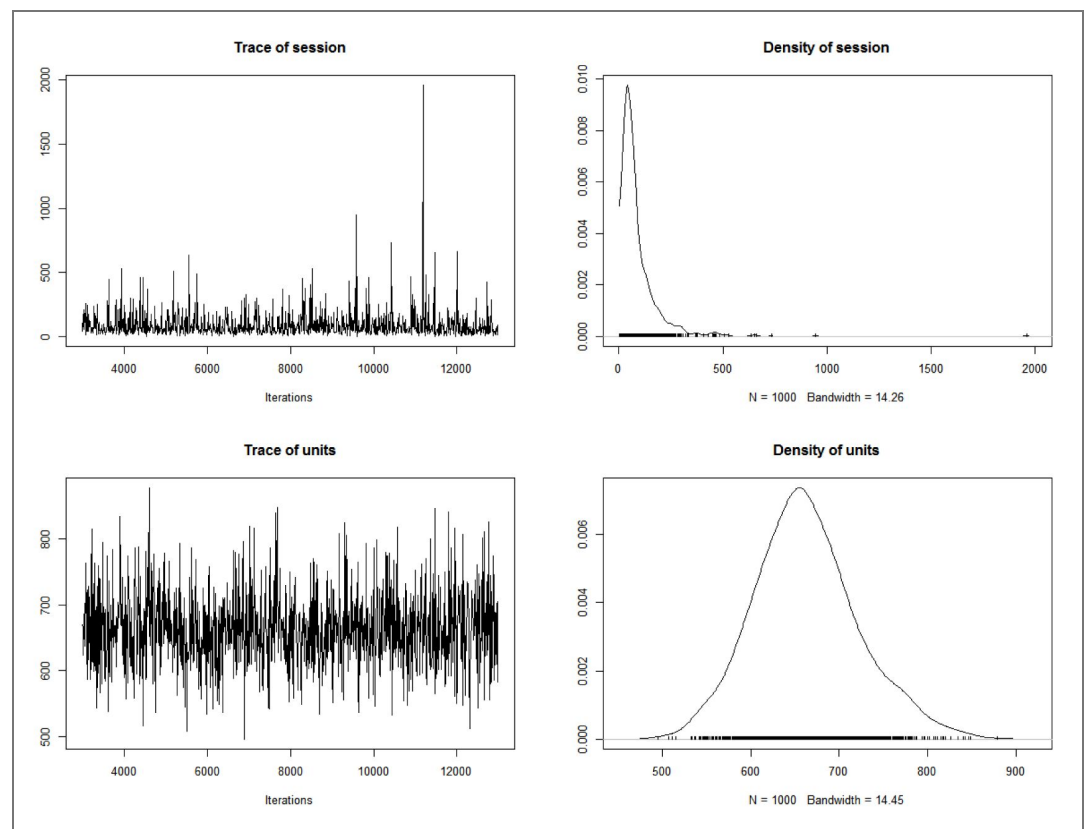


Figure S9. Density of intercept and individual-within effect from MCMC sampling for the outcome value.

	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

Table S9. Summary for causal mediation model in predicting task aversiveness by using treatment (active ms-tDCS vs sham) from mediated effect of increased task outcome.

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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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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.

Weaknesses:

There are quite a few weaknesses, some related to the actual study and some more strongly related to the reporting about the study in the manuscript. The concerns are listed roughly in the order in which they appear in the manuscript.

- (1) In the introduction, the authors present procrastination nearly as if it were *the* most relevant and problematic issue there is in psychology. Surely, procrastination is a relevant and study-worthy topic, but that is also true if it is presented in more modest (and appropriate) terms. The manuscript mentions that procrastination is a main cause of psychopathology and bodily disease. These claims could possibly be described as 'sensationalized'. Also, the studies to support these claims seem to report associations, not causal mechanisms, as is implied in the manuscript.
- (2) It is laudable that the study was pre-registered; however, the cited OSF repository cannot be accessed and therefore, the OSF materials cannot be used to (a) check the preregistration or to (b) fill in the gaps and uncertainties about the exact analyses the authors conducted (this is important because the description of the analyses is insufficiently detailed and it is often unclear how they analyzed the data).
- (3) Related to the previous point: I find it impossible to check the analyses with respect to their appropriateness because too little detail and/or explanation is given. Therefore, I find it impossible to evaluate whether the conclusions are valid and warranted.
- (4) Why is a medium effect size chosen for the a priori power analysis? Is it reasonable to assume a medium effect size? This should be discussed/motivated. Related: 18 participants for a medium effect size in a between-subjects design strikes me as implausibly low; even for a within-subjects design, it would appear low (but perhaps I am just not fully understanding the details of the power analysis).
- (5) It remains somewhat ambiguous whether the sham group had the same number of stimulation sessions as the verum stimulation group; please clarify: Did both groups come in

the same number of times into the lab? I.e., were all procedures identical except whether the stimulation was verum or sham?

(6) The TDM analysis and hyperbolic discounting approach were unclear to me; this needs to be described in more detail, otherwise it cannot be evaluated.

(7) Coming back to the point about the statistical analyses not being described in enough detail: One important example of this is the inclusion of random slopes in their mixed-effects model which is unclear. This is highly relevant as omission of random slopes has been repeatedly shown that it can lead to extremely inflated Type 1 errors (e.g., inflating Type 1 errors by a factor of then, e.g., a significant p value of .05 might be obtained when the true p value is .5). Thus, if indeed random slopes have been omitted, then it is possible that significant effects are significant only due to inflated Type 1 error. Without more information about the models, this cannot be ruled out.

(8) Related to the previous point: The authors report, for example, on the first results page, line 420, an F-test as $F(1, 269)$. This means the test has 269 residual degrees of freedom despite a sample size of about 50 participants. This likely suggests that relevant random slopes for this test were omitted, meaning that this statistical test likely suffers from inflated Type 1 error, and the reported p-value $< .001$ might be severely inflated. If that is the case, each observation was treated as independent instead of accounting for the nestedness of data within participants. The authors should check this carefully for this and all other statistical tests using mixed-effects models.

(9) Many of the statistical procedures seem quite complex and hard to follow. If the results are indeed so robust as they are presented to be, would it make sense to use simpler analysis approaches (perhaps in addition to the complex ones) that are easier for the average reader to understand and comprehend?

(10) As was noted by an earlier reviewer, the paper reports nearly exclusively about the role of the left DLPFC, while there is also work that demonstrates the role of the right DLPFC in self-control. A more balanced presentation of the relevant scientific literature would be desirable.

(11) Active stimulation reduced procrastination, reduced task aversiveness, and increased the outcome value. If I am not mistaken, the authors claim based on these results that the brain stimulation effect operates via self-control, but - unless I missed it - the authors do not have any direct evidence (such as measures or specific task measures) that actually capture self-control. Thus, that self-control is involved seems speculation, but there is no empirical evidence for this; or am I mistaken about this? If that is indeed correct, I think it needs to be made explicit that it is an untested assumption (which might be very plausible, but it is still in the current study not empirically tested) that self-control plays any role in the reported results.

(12) Figures 3F and 3H show that procrastination rates in the active modulation group go to 0 in all participants by sessions 6 and 7. This seems surprising and, to be honest, rather unlikely that there is absolutely no individual variation in this group anymore. In any case, this is quite extraordinary and should be explicitly discussed, if this is indeed correct: What might be the reasons that this is such an extreme pattern? Just a random fluctuation? Are the results robust if these extreme cells are ignored? The authors remove other cells in their design due to unusual patterns, so perhaps the same should be done here, at least as a robustness check.

(13) The supplemental materials, unfortunately, do not give more information, which would be needed to understand the analyses the authors actually conducted. I had hoped I would find the missing information there, but it's not there.

In sum, the reported/cited/discussed literature gives the impression of being incomplete/selectively reported; the analyses are not reported sufficiently transparently/fully to evaluate whether they are appropriate and thus whether the results are trustworthy or not. At least some of the patterns in the results seem highly unlikely (0 procrastination in the verum group in the last 2 observation periods), and the sample size seems very small for a between-subjects design.

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Reviewer #2 (Public review):

Summary:

Chen and colleagues conducted a cross-sectional longitudinal study, administering high-definition transcranial direct stimulation 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 report that tDCS effects on task-execution willingness and procrastination are mediated by task outcome value and claim that this neuromodulatory intervention reduces procrastination rates quantified by their task. Although the study addresses an interesting question regarding the role of DLPFC on procrastination, concerns about the validity of the procrastination moderate enthusiasm for the study and limit the interpretability of the mechanism underlying the reported findings.

Strengths:

- (1) This is a well-designed protocol with rigorous administration of high-definition transcranial direct current stimulation across multiple sessions. The approach is solid and aims to address an important question regarding the putative role of DLPFC in modulating chronic procrastination behavior.
- (2) 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:

- (1) The lack of specificity surrounding the "real-world measures" of procrastination is problematic and undermines the strength of the evidence surrounding the DLPFC effects on procrastination behavior. It would be helpful to detail what "real-world tasks" individuals reported, which would inform the efficacy of the intervention on procrastination performance across the diversity of tasks. It is also unclear when and how tasks were reported using the ESM procedure. Providing greater detail of these measures overall would enhance the paper's impact.
- (2) Additionally, it is unclear whether the reported effects could be due to differential reporting of tasks (e.g., it could be that participants learned across sessions to report more achievable or less aversive task goals, rather than stimulation of DLPFC reducing procrastination per se). It would be helpful to demonstrate whether these self-reported tasks are consistent across sessions and similar in difficulty within each participant, which would strengthen the claims regarding the intervention.
- (3) It would be helpful to show evidence that the procrastination measures are valid and consistent, and detail how each of these measures was quantified and differed across sessions and by intervention. For instance, while the AUC metric is an innovative way to

quantify the temporal dynamics of task-aversiveness, it was unclear how the timepoints were collected relative to the task deadline. It would be helpful to include greater detail on how these self-reported tasks and deadlines were determined and collected, which would clarify how these procrastination measures were quantified and varied across time.

(4) There are strong claims about the multi-session neuromodulation alleviating chronic procrastination, which should be moderated, given the concerns regarding how procrastination was quantified. It would also be helpful to clarify whether DLPFC stimulation modulates subjective measures of procrastination, or alternatively, whether these effects could be driven by improved working memory or attention to the reported tasks. In general, more work is needed to clarify whether the targeted mechanisms are specific to procrastination and/or to rule out alternative explanations.

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Reviewer #3 (Public review):

This manuscript explores whether high-definition transcranial direct current stimulation (HD-tDCS) of the left DLPFC can reduce real-world procrastination, as predicted by the Temporal Decision Model (TDM). The research question is interesting, and the topic - neuromodulation of self-regulatory behavior - is timely.

However, the study also suffers from a limited sample size, and sometimes it was difficult to follow the statistics.

The preregistration and ecological design (ESM) are commendable, but I was not able to find the preregistration, as reported in the paper.

Overall, the paper requires substantial clarification and tightening.

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Author response:

Reviewer #1:

(1) We fully thank you to point out the risks of sensationalizing ramification of procrastination on psychopathology, and would rewrite the Introduction section by adding balanced evidence and overall toning down such inappropriate claims meanwhile.

(2) Thank you to raise this crucial question. We are sorry for this fundamental technical issue to preregistration. This occurs from a seriously technical hurdle. The OSF has banned my OSF account, as it claimed to detect “suspicious user’s activities” in my account. This causes no accesses to all materials that already deposited in this OSF account, including this preregistration. We have contacted OSF team, but received no valid technical solution. We reckon that this may be mistaken by my affiliation changes to Third Military Medical University of People’s Liberation Army (PLA). To tackle with this technical issue, we shall upload preregistration in a new repository soon.

(3) This is a back-to-back study to conceptually probe into whether strengthening left DLPFC can mitigate procrastination via reducing task aversiveness or weighting outcome value. Thus, the current study selected a medium effect size in aprior by following the previous one (Xu et al., 2023). This effect size is calculated by the new tool called “Power Contours” (Baker et al., 2021), which weights statistical power by increasing within-subject repeated measures. As you kindly pointed out, we shall clarify effect size calculation in the revised manuscript.

- (4) Yes, both groups come in the same number of times into the lab for tDCS stimulation, except to the type (active vs sham).
- (5) We shall add full details for clarifying TDM and hyperbolic discounting modeling.
- (6) Thank you to raise this very crucial statistical question. We shall double-check whether multiple sessions are modeled as random slopes, and would like to reanalysis it in case which those random slopes are omitted.
- (7) Thank you. We have no intentions of confusing you by adding those complicated statistics, but indeed enrich understanding of how we can interpret those findings.
- (8) Yes, as mentioned above, we shall add balanced evidence to clarify both left and right DLPFC may function to self-control capability in the Introduction section.
- (9) Yes, this is a conceptual hypothesis --- actively stimulating left DLPFC could improve self-control functions. Thank you for this very nuanced but crucial insight, and we could explicitly clarify the nature of our conclusions.
- (10) Yes, we ensure that all the participants successfully completed their tasks before deadline at session 6 and 7, and the procrastination rates have been all decreased to 0. Personally speaking, this is somewhat surprise to us as well, but we affirmed this case. For a portion of participants included in the active group, we have received written letters of thanks from them. Thus, this is surprise but exciting finding. Furthermore, thank you for this helpful suggestion, and we would like to do this robustness check by iteratively removing each session, to obviate the statistical biases from an extreme pattern.
- (11) Yep, we fully agree with you to add full details in the main text rather in Supplemental materials, and would like to do so in the first round of revision.

Reviewer #2:

- (1) Thank you for this very crucial suggestion. We are sorry for this case that much details are omitted to comply with editorial requirement at Nature Human Behaviour (last submission). We do apologize to confuse you as those ambiguous descriptions, and would like to clearly clarify how we measure participants' procrastination in the real-world tasks. In brief, we asked participant to report a real task that would really happen in the tomorrow and its deadline is also no more than tomorrow. When tomorrow comes, we used ESM to require participant reporting real task completion rate (0-100%) at five time points before the deadline. The five time points are determined by a hyperbolic discounting model (see how and why we set those five time points in the full author's response letter later). When participant reports the real task completion rate (0-100%) at a given time point, she/he is required to provide a photo to prove its authenticity. The dependent variable --- real-world procrastination rates --- is thus calculated as 100% subtracts the task completion rate (0-100%) when the deadline meets. That is to say, if participant reports task has been fully completed before or when deadline meets, his/her real-world procrastination rate is $100\% - 100\% = 0\%$; if reporting task has been completed 60% when deadline meets, the real-world procrastination rate is determined as $100\% - 60\% = 40\%$. Do not worry for spurious reporting, we asked all the participants to provide photo verifying the real task completion rate. This is merely a short instance. We shall show the full details in the formal author response letter later.
- (2) This is a very meaningful point. We agree with you for this case that participants may learn how to complete this experiment task swiftly rather benefit from neuromodulation. This speculation makes sense, but is compromised by experimental control and empirical observations. Firstly, we do not say "You must complete this task" or "The task completion is

associated with bonus/rewards you may get” for participants, which indicates no motivations to do so. Then, the measures to task completion rate are not yet fully based on self-reporting, and we mandate them to provide photos for verification. Thus, this controls the marked risks of spurious reporting. Lastly, all the participants, including ones in either active or sham group, received all the same treatments, excepting “real simulation” and “sham simulation” protocol. Results demonstrated the significant amelioration in the active group rather than sham one, indicating no significant “placebo” or “task learning” side effect.

(3) Thank you. As you kindly suggested, we would like to add huge details for those measures in the revised manuscript. While this is a great idea, we did not collect procrastination scores from scales after neuromodulation, and would like to warrant this point into the Limitation section.

(4) Yep, this is a conceptual hypothesis --- actively stimulating left DLPFC could improve self-control functions. We cannot rule out possibilities of amplifying working memory, attention or other cognitive components from this neuromodulation protocol. We fully agree with you for this helpful recommendation --- we would like to tone down those claims regarding the roles of DLPFC on self-control, and explicitly warrant that this mechanism may be specialized to the procrastination.

Reviewer #3:

(1) Thank you for taking valuable time to review our manuscript. Yep, limited sample size should warrant cautions to draw a solid conclusion. We would like to claim it into the limitation section. Also, we have streamlined and tightened statistic section by removing complicated and redundancy statistical models.

(2) As mentioned above, we are sorry for this fundamental technical issue to preregistration. This occurs from a seriously technical hurdle. The OSF has banned my OSF account, as it claimed to detect “suspicious user’s activities” in my account. This causes no accesses to all materials that already deposited in this OSF account, including this preregistration. We have contacted OSF team, but received no valid technical solution. We reckon that this may be mistaken by my affiliation changes to Third Military Medical University of People’s Liberation Army (PLA). To tackle with this technical issue, we shall upload preregistration in a new repository soon.

(3) Yep, thank you for this very helpful suggestion. As you kindly indicated, we would like to clarify measures, analyses, methods, and protocols, as well as tighten the whole manuscript.

References

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Again, we wholeheartedly appreciate all of those very helpful and insightful comments, with each one to contribute substantially for the quality of this manuscript. Notably, those response we presented above are merely provisional and initial. We shall revise our manuscript following those suggestions, one-by-one, along with a full-length response letter.

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