Brain Research 1759 (2021) 147365

Contents lists available at ScienceDirect

Brain Research

journal homepage: www.elsevier.com/locate/brainres

Reduced risk-taking behavior during frontal oscillatory theta band neurostimulation

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ARTICLE INFO

Keywords:
tACS
Risk-taking behavior
Theta activity
Theta frequency
Decision neuroscience

ABSTRACT

Background: Most of our decisions involve a certain degree of risk regarding the outcomes of our choices. People vary in the way they make decisions, resulting in different levels of risk-taking behavior. These differences have been linked to prefrontal theta band activity. However, a direct functional relationship between prefrontal theta band activity and risk-taking has not yet been demonstrated.
Objective: We used noninvasive brain stimulation to test the functional relevance of prefrontal oscillatory theta activity for the regulatory control of risk-taking behavior.
Methods: In a within-subject experiment, 31 healthy participants received theta (6.5 Hertz [Hz]), gamma (40 Hz), and sham transcranial alternating current stimulation (tACS) over the left prefrontal cortex (lPFC). During stimulation, participants completed a task assessing their risk-taking behavior as well as response times and sensitivity to value and outcome probabilities. Electroencephalography (EEG) was recorded before and immediately after stimulation to investigate possible long-lasting stimulation effects.
Results: Theta band, but not gamma band or sham, tACS led to a significant reduction in risk-taking behavior, indicating a frequency-specific effect of prefrontal brain stimulation on the modulation of risk-taking behavior. Moreover, theta band stimulation led to increased response times and decreased sensitivity to reward values. EEG data analyses did not show an offline increase in power in the stimulated frequencies after the stimulation protocol.
Conclusion: These findings provide direct empirical evidence for the effects of prefrontal theta band stimulation on behavioral risk-taking regulation.

1. Introduction

Human decision-making often includes a certain degree of risk regarding its outcomes and outcome probabilities. Take, for example, financial investments, driving above the speed limit, or simply trying a new cuisine. In all of these situations, and many others, the outcomes of our decisions and actions cannot be predicted with absolute certainty. A decision-maker exhibits risk-taking behavior in these situations. Risk-taking is inevitable and may not only have (un)desired personal but also social and economic impacts (Trimpop, 1994). Therefore, the regulatory control of risk-taking behavior is of utmost importance for human decision-making.

During decision-making under risk, a complex mechanism is at work. This mechanism codes and flexibly evaluates the context, outcome probabilities, and previous information to define the optimal level of risk to be taken (Kuhnen and Knutson, 2005). Despite what is expected from pure rational models, risk-taking behavior is not consistent across different contexts, and the optimal decision is often rejected (Brand et al., 2007). These inconsistencies are likely a consequence of the complexity of the neural mechanisms involved in risk-taking behavior

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https://doi.org/10.1016/j.brainres.2021.147365
Received 26 November 2020; Received in revised form 4 February 2021; Accepted 4 February 2021
Available online 12 February 2021

and the control thereof, which have been extensively explored by previous studies (e.g., 3–6). Namely, risk-taking behavior is the result of a complex interplay between emotional responses to possibilities of reward (limbic activity) and the inhibition of such responses via the activation of frontal control regions (Floden et al., 2008). Among these, the ventromedial prefrontal cortex (VMPFC) and dorsolateral prefrontal cortex (DLPFC) are critical areas responsible for signaling the need for strategy adjustment and executive control, respectively (Galvan et al., 2006). However, the exact mechanism underlying such signaling processes remains unclear.

Electroencephalography (EEG) studies have shown that participants with a higher theta power (4–8 Hertz [Hz]) in the right prefrontal cortex (rPFC) compared to the left, i.e., a higher frontal theta band asymmetry, displayed more risk-taking behavior during gambling tasks (Gianotti et al., 2009; Studer et al., 2013a). Recent literature confirms the inverse relationship between risk-taking behavior and frontal theta power, where more risk aversion is observed in participants with higher frontal theta power and vice-versa (Schmidt et al., 2019b; 2018). Furthermore, other studies show a positive correlation between error detection, cognitive control, and increased right VMPFC theta power (Gallagher et al., 2009).

Moreover, theta oscillations are involved in neural network communication when cognitive control is required (Cavanagh and Frank, 2014). Prefrontal theta oscillations may therefore represent part of the signaling mechanism by which the VPMFC recruits the DLPFC in case recruitment of regulatory mechanisms is needed upon the detection of a risky context. However, although these EEG studies indicate that theta oscillations are related to risk-taking behavior, the functional behavioral relevance of this oscillatory pattern in the regulation of risk-taking has yet not been shown.

Noninvasive brain stimulation, such as transcranial alternating current stimulation (tACS), offers the possibility of inducing temporary oscillatory patterns in specific brain regions by applying changing electric currents on the scalp, transiently modulating brain activity. This allows the probing of the relationship between frequency patterns and behavioral responses (Reato et al., 2013a). To investigate the role of theta band frontal asymmetry in risk-taking behavior, Sela and colleagues (2012) applied theta band tACS over the right and left DLPFC while participants performed the Balloon Analog Risk Task. After left, but not right, DLPFC theta band stimulation, an increase in risk-taking behavior was found. This was not in line with prior EEG studies hypothesizing that right DLPFC stimulation increases risk-taking behavior by increasing frontal asymmetry, while left DLPFC tACS reduces risk-taking behavior due to an increase in theta band activity in the left hemisphere and consequent asymmetry reduction (Gianotti et al., 2009; Studer et al., 2013a). Sela and colleagues (2010) speculated that their findings may be due to a disruption of interhemispheric balance in participants' natural frontal asymmetry (Sela et al., 2012). The authors were not able to make conclusions about the frequency specificity of the stimulation as no control frequencies had been applied (Feurra et al., 2012). Moreover, they opted for using the Balloon Analog Task to estimate risk. This task mostly measures impulsivity and evaluates uncertainty rather than risk, which is a different economic construct (Lejuez et al., 2002) since the probabilities are not explicit to participants.

The present study aims at investigating this functional relationship between frontal theta band oscillations and risk-taking behavior. Although previous studies (Gianotti et al., 2009; Studer et al., 2013b) have shown a correlation between resting state frontal theta band asymmetry and risk-taking behavior, no direct causal relationship between theta band activity and risk-taking behavior regulation has thus far been shown. We therefore applied tACS to the left DLPFC in theta band (6.5 Hz) while participants performed a risk-taking task to affect frontal theta band activity and, as proposed by Sela and colleagues (2012), disrupt frontal theta band asymmetry. Moreover, we applied gamma band (40 Hz) tACS and sham stimulation as controls. We chose gamma band tACS as a control frequency as it has not been linked to risk-taking behavior thus far. We also implemented a new behaviorally controlled risk-taking protocol paired with financial incentives for more robust measures of risk-taking behavior.

Considering that the EEG recording during tACS protocols is still a suboptimal option (Bland and Sale, 2019), we used EEG recordings before and immediately after the transcranial brain stimulation to monitor possible long-lasting power changes in the stimulated frequencies. These measurements aimed to investigate a possible functional relationship between electrophysiological effects and behavioral results.

We hypothesized that, compared to sham and gamma band stimulation, theta band stimulation to the left DLPFC decreases risk-taking behavior, confirming the central regulatory role of theta frequencies on the electrophysiological mechanism underlying the modulation of risk-taking behavior (Başar et al., 2001; Gianotti et al., 2009).

To test this hypothesis, we used a within-subjects design in which participants went through three different tACS protocols. In each session, participants received either theta (6.5 Hz), gamma (40 Hz), or sham stimulation. During the period of stimulation, participants were asked to play a computer gambling game in which they had to opt between two different bets with various payoffs and probabilities of winning, named the Maastricht Gambling Task (MGT). EEG recordings were performed before and after the stimulation period for three minutes in each block. More details of the experimental design and procedure can be found in Section 4.

2. Results

2.1. Behavioral results

In this section, we present the main behavioral results of our experiment. The detailed statistical methodology can be found in Section 4.6.

2.1.1. Main results: Risk

The estimated fixed effects analysis of the effects of the different protocols of stimulation on risk-taking behavior showed a significant reduction, of −0.301, on risk-taking behavior during theta band stimulation, t(66.69) = -2.04, p = .05, SE = 0.15, d = -0.50, indicating a medium negative effect of theta band stimulation on risk when compared to sham (Fig. 1). Moreover, gamma stimulation did not affect the participant's average risk-taking significantly compared to sham, t (69.992) = -1.22, p = .23, SE = 0.10, d = -0.29, confirming that the effects observed are frequency specific.

2.1.2. Probability scores

The linear mixed model analyses with probability as the dependent variable did not yield significant main effects for stimulation, F (2,47.29) = 0.76, p = .92. The estimated fixed effects analyses also did not yield significant effects of theta band stimulation, t(35.08) = 0.32, p = .75, SE = 0.01, d = 0.11, or gamma stimulation, t(67.70) = -0.80, p = .43, SE = 0.01, d = -0.19, when compared to sham, meaning that no significant differences in the probability scores were observed after the different stimulation protocols.

2.1.3. Value

These analyses of the effect of stimulation on the average values yielded a non-significant main effect of stimulation, F(2,91.89) = 2.43, p = .09. Further analyses of estimated fixed effects yielded significant effects of theta stimulation on value, with a reduction of −0.67 compared to sham, t (64.33) = -2.13, p = .04, SE = 0.32, d = -0.53, indicating a medium negative effect of theta band stimulation on the average value chosen by the participants when compared to sham. No significant effects were observed after gamma stimulation, t(71.19) = -1.27, p = .21, SE = 0.21, d = -0.30, compared to sham. This means that there was a significant reduction in the average value chosen by the

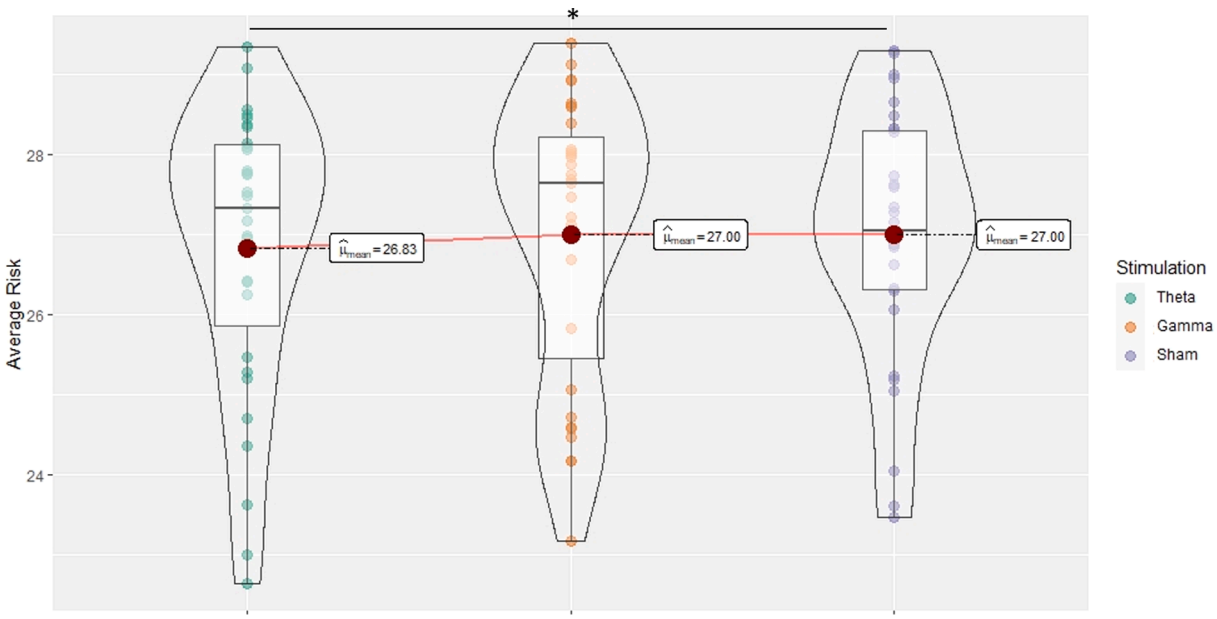

Fig. 1. Average Risk-taking Behavior (n = 31). Average risk-taking estimated by the average standard deviation of each participant's choice across stimulation conditions (theta [6.5 Hz] in green, gamma [40 Hz] in orange, and sham in purple). Risk can vary between 11.75 and 36.15. Dark red marks indicate the mean risk per condition.

participants due to the theta stimulation, confirming that this is a frequency-exclusive effect (Fig. 2). These findings reinforce the strong relationship between risk-taking behavior and valuation since both processes were affected by the same pattern of stimulation.

2.1.4. Response time

Estimated fixed effects analyses of the effects of stimulation on response time showed strong significant effects of stimulation, $F(2,50.24) = 35.80$, $p < .001$. Furthermore, these analyses yielded significant results for theta stimulation, $t(24.26) = 5.16$, $p < .001$, $SE = 0.07$, $d = 2.10$, indicating a large effect of theta band stimulation on response time and a nearly significant medium effect for gamma stimulation, $t(63.86) = 1.88$, $p = .07$, $SE = 0.03$, $d = 0.47$, when compared to sham. Theta stimulation led to an increase of 41.11% in response time (compared to sham). This implies that the theta stimulation led to an increase in the deliberation time, which cannot be attributed to the stimulation per se since this effect was only marginally significant in the gamma stimulation condition. Details can be observed in Fig. 3, where response time is plotted against contrast, or trial difficulty level, based on the cluster division previously explained, from easier decisions (which are clear, with big differences in EV between pink and blue) to difficult decisions in which the mental calculation to define the most advantageous option is more challenging.

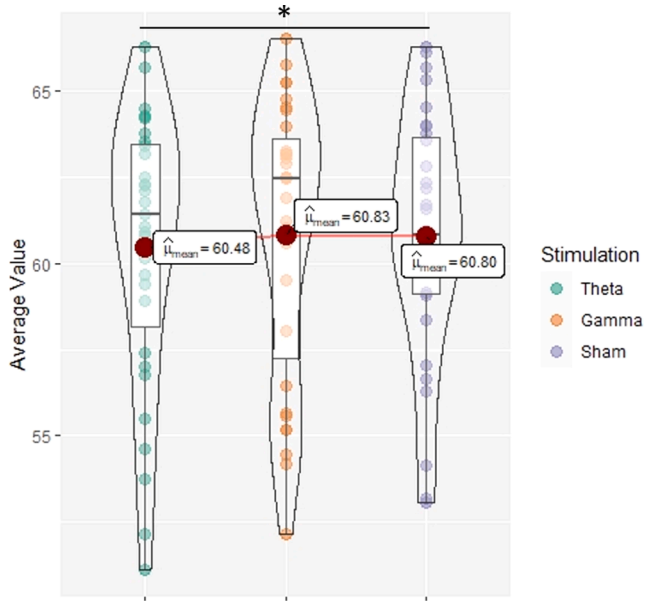

Fig. 2. Average Value (n = 31). Average value per condition (theta [6.5 Hz] in green, gamma [40 Hz] in orange, and sham in purple). Dark red marks indicate the mean value per condition.

2.2. EEG results

2.2.1. Theta band entrainment

To investigate the effects of theta band stimulation on EEG results, we ran a repeated measures ANOVA with theta power as the dependent variable, considering the entire interval of three minutes of data. The repeated measures ANOVA used a 3 (stimulation condition: theta, gamma, and sham) by 2 (time: before and after stimulation) by 6 (theta power averaged over 3 min on each electrode: F1, F5, F2, F6, P5, P6) within-subject design, with Bonferroni correction for multiple comparisons.

The results showed a significant main effect of time, with theta power increasing from an average of −0.135 to an average of −0.056 after stimulation, $F(1,6) = 3.38$, $p = .01$. There was no significant main effect of stimulation, $F(1,12) = 0.82$, $p = .63$, and no significant interaction effect between stimulation and time, $F(1,12) = 0.82$, $p = .63$ (for descriptives, please see the Table S2).

Further analyses included a 3 (stimulation condition) by 2 (time) repeated measures ANOVA using frontal asymmetry as the dependent variable. There was no significant effect of stimulation on frontal asymmetry, $F(1,2) = 1.19$, $p = .17$; time, $F(1,1) = 0.06$, $p = .81$, or of the interaction between time and stimulation, $F(1,2) = 0.81$, $p = .46$.

Most studies looking at tACS after-effects using EEG have not found electrophysiological effects lasting beyond the stimulation offset (Deans et al., 2007; Reato et al., 2013b; Strüber et al., 2015a). Therefore, we ran *post hoc* analyses to investigate whether the effects were visible only at the very beginning of the period after stimulation, fading during the full interval of three minutes. To do so, we ran a repeated measures ANOVA

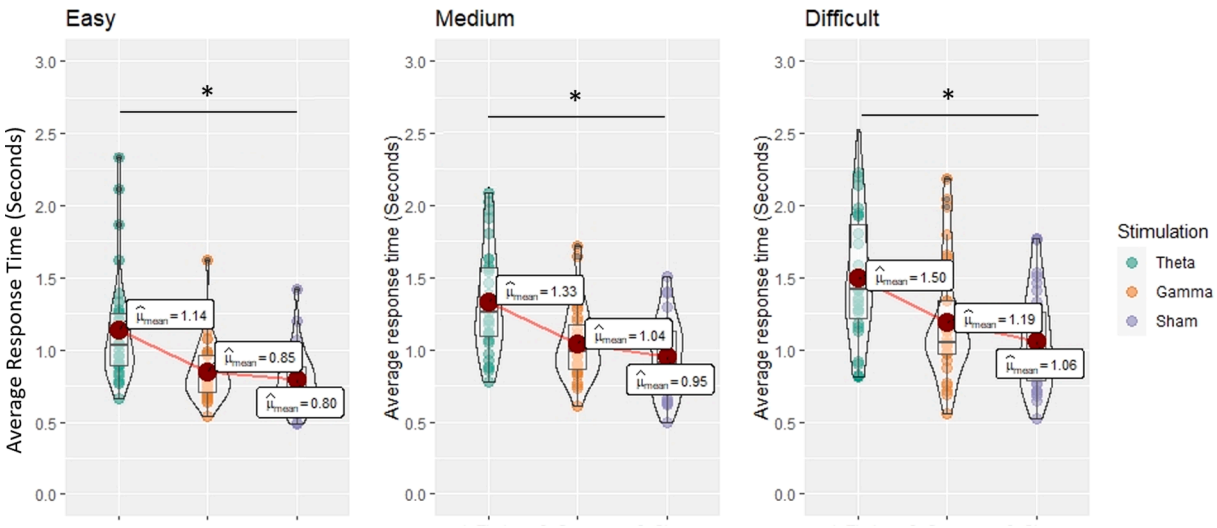

Fig. 3. Average Response Time by Task Difficulty Level (n = 31). Average response time per trial difficulty in seconds by stimulation protocol (theta [6.5 Hz] in green, gamma [40 Hz] in orange, and sham in purple). Dark red marks indicate the average response time per condition.

using as the dependent variable the difference in theta power between the first minute before stimulation and the first minute immediately after it. We used a 3 (stimulation conditions: theta, gamma, and sham) by 7 (theta power difference on each electrode: F1, F5, F2, F6, P5, P6 and change in frontal asymmetry) within-subject design, with Bonferroni correction for multiple comparisons. These analyses yielded a significant effect of stimulation, $F(1,12) = 4.44$, $p < .001$.

Further contrasts showed significant effects of both theta and gamma band stimulation. There was a large and significant effect of theta band stimulation (and not gamma) on asymmetry change (pre-post) when compared to sham $t(2) = 2.53$, $p = .01$, $d = 3.58$. However, this effect was mainly driven by a decrease in asymmetry in the sham condition, indicating that the decrease is due to the task execution and not to the stimulation.

Following these steps, we ran a time frequency analysis considering all times recorded before and after stimulation. These analyses yielded no significant difference between the experimental conditions (theta and gamma band stimulation) compared to sham. The frequency spectrum contrasting pre- vs. post-power spectrum for each condition can be seen in the supporting information (S3 Fig. 1).

We also conducted a partial correlation analysis between the frontal theta asymmetry, theta power in F1, F3, F5, F6, P2, and P6, and the behavioral responses (probabilities chosen, average value chosen, risk, and response time). The level of asymmetry before or after the stimulation did not significantly correlate with either of the behavioral measures. Theta power in F1 and F2 was significantly correlated to the probabilities chosen ($r = 0.11$ and $p = .02$ and $r = 0.01$ and $p = .01$, respectively) although there were no significant effects of stimulation on the probabilities chosen by the participants. The results also indicate trends regarding the correlations between theta power in F1 and F2 and the average values chosen ($r = 0.0$ and $p = .10$ and $r = 0.08$ and $p = .09$, respectively) and between theta power in these same electrodes and risk ($r = 0.08$ and $p = .08$ and $r = 0.08$ and $p = .07$, respectively).

The inclusion of asymmetry in theta power in any of the electrodes in the regression models used to analyze the behavioral results did not improve the fit of these models and therefore was discarded.

2.2.2. *Gamma band entrainment*

The effects of gamma band stimulation were investigated using a 3 (stimulation condition: theta, gamma, and sham) by 2 (time: before and after stimulation) by 6 (theta power averaged over 3 min on each electrode: F1, F5, F2, F6, P5, P6) within-subject repeated measures ANOVA, with Bonferroni correction for multiple comparisons. No significant effect of stimulation condition, $F(1,12) = 0.85$, $p = .60$, nor of time, $F(1,6) = 1.05$, $p = .42$, or the interaction between stimulation and time, $F(1,12) = 1.04$, $p = .47$, was observed. Therefore, there was no significant gamma entrainment, or its effects were not visible in the behavioral or EEG results.

3. Discussion

The present study aimed at investigating the functional relationship between frontal theta band oscillations and risk-taking behavior. Although previous studies (Gianotti et al., 2009; Studer et al., 2013b) have shown a correlation between resting state frontal theta band asymmetry and risk-taking behavior, no direct causal relationship has thus far been shown. We hypothesized that theta oscillations underlie the neuronal communication for recruiting the DLPFC when the decision-making process includes risk, being fundamental for the modulation of risk-taking behavior (Cavanagh and Frank, 2014). We therefore expected theta band stimulation to cause a reduction in risk-taking behavior and that this effect is frequency specific.

As predicted, we were able to effectively reduce risk-taking behavior in healthy participants using theta band tACS over the left DLPFC compared to sham and gamma band stimulation. These findings confirm the functional relationship between theta band frequencies and risk-taking behavior regulation, being a fundamental part of the electrophysiological mechanism responsible for this modulation. Theta band tACS leads to a significant decrease, of 1.12%, in risk-taking behavior compared to sham. This was not the case during gamma stimulation.

To our knowledge, our study is the first to show the frequency specificity of this effect. Moreover, we observed a significant reduction in value sensitivity due to theta band (and not gamma) stimulation, meaning that participants opted for lower values after theta band stimulation compared to the results obtained in the sham or gamma conditions. These results are in line with previous studies, where participants became more risk-averse after noninvasive brain stimulation with reduced sensitivity to value (Boggio et al., 2010; Fecteau et al., 2007a; Gilmore et al., 2018; Levasseur-Moreau and Fecteau, 2012). However, our study was able to show that this effect is also frequency specific. Therefore, it is expected that theta frequencies would play a fundamental role in the reduction in value sensitivity, meaning the recruitment of the DLPFC as the executive control to modulate the VMPFC response to the value (Hare et al., 2011).

The stimulation did not affect the probabilities chosen by the participants, indicating that the choice of probabilities might be regulated

by a different electrophysiological mechanism. Even though our results indicate that probabilities and value are evaluated independently in our brain, behaviorally and in terms of neurological activity, these processes are at least strongly correlated (Knutson et al., 2001; Kuhnen and Knutson, 2005; Tobler et al., 2007). This means that both inputs (bet value and its probabilities) are considered to inform the decision process, which justifies the use of standard deviation as an estimation of risk. Our approach considers the option's expected value (meaning the bet's probabilities and value) to estimate risk, which is in our perspective a more naturalistic evaluation of risk. Our findings indicate that participants' reductions in risk-taking behavior were mainly driven by a reduction in the average value sensitivity.

Although we did not have a specific hypothesis regarding the response time, it is interesting to notice that theta stimulation increased response time compared to sham and gamma stimulation. It may be speculated that the increased response time reflects a longer deliberation process (Rubinstein, 2013).

It is important to note that our results contradict the study by Sela and colleagues (2012). Their results indicated an increase in risk-taking behavior after theta band stimulation, which might be explained by their choice of the Balloon Analog Task as the experimental paradigm. Since this task has a strong factor of impulsivity, the effect observed should reflect an increase in impulsivity and not in risk-taking behavior (Lejuez et al., 2002; Schonberg et al., 2011). Moreover, they considered the tolerability to losses (measured as sequential explosions) as an indicator of riskier choices (Sela et al., 2012), which means that their results might also indicate a reduction in loss-aversion. Since our experimental paradigm (MGT) avoids loss-aversion and impulsivity, we may have more directly assessed risk-taking behavior. Finally, we must consider the impact of the use of real monetary incentives in economic decision-making (Xu et al., 2016). Since our task was monetarized, the observed results have a higher reliability.

It is also interesting to highlight that our results showed considerable robustness despite the use of random trial selection for payment. This compensation method, despite being widely used in economics experiments, might have led to a decrease in risk-taking behavior and electrophysiological responses to monetary feedback (Schmidt et al., 2019a; Schmidt and Hewig, 2015). However, since we used the same method of compensation across sessions and treatment conditions, it should not influence a specific treatment effect.

In addition to assessing the behavioral effects of our oscillatory brain state neuromodulation on risk-taking modulation, we also used EEG to measure oscillatory activity before and after tACS. It is important to highlight, however, that up to this point, to our knowledge, there is no evidence of long-lasting effects of theta or gamma band tACS on frequency modulation (Heise et al., 2019; Reato et al., 2013c; Strüber et al., 2015a). This means that significant effects on EEG data after stimulation would also depend on long-lasting effects of our stimulation protocol, considering the technical limitations of online recording already discussed (Bland and Sale, 2019).

When comparing theta power before and after theta tACS, no significant changes were found, nor did we reveal significant changes in hemispheric theta band asymmetry after theta band stimulation. This may seem surprising and in contrast to our behavioral effects being attributed to and interpreted as being caused by tACS-induced increase in left theta power. However, it is important to note here that while behavioral effects were assessed during tACS being applied simultaneously with task execution, the EEG measurements, due to tACS artifacts, were restricted to assessing the oscillatory activity after both the behavioral performance and tACS had ended. Especially the latter may be a straightforward explanation for the absence of significant EEG effects in a pre-post tACS design as such effects rely on a significant longer-lasting neurophysiological effect of tACS beyond the period of stimulation itself.

However, the question as to whether tACS-induced entrainment is longer lasting is far from being settled (Strüber et al., 2015a). Offline effects of tACS are rarely reported, and various previous studies have also reported difficulties in establishing longer-lasting effects of tACS on excitability or neural plasticity (Bland and Sale, 2019; Reato et al., 2013a; Schutter, 2016; Strüber et al., 2015b). Considering our results, we may therefore speculate that the EEG effects were only present during the task and stimulation and faded away immediately after tACS had ended. Our *post hoc* analyses focusing only on the first minute of post-EEG measurements after TACS and contrasting these effects against the entire post-EEG period indicate time-sensitive changes in theta band asymmetry in line with this speculation. Yet, our study was not designed to conclusively test any other related hypotheses regarding the difference between the immediate versus lasting effects of tACS on neural oscillatory activity. Follow-up studies with online measurements using algorithms to remove stimulation artifacts could be used to investigate such possibility although, currently, this methodology is still under debate (Bland and Sale, 2019).

In addition, we also revealed that the task execution itself had lasting effects on theta band asymmetry, as indicated by *post hoc* analyses of the EEG measures immediately after the task execution in the sham condition. In other words, unrelated to tACS, the mere behavioral performance in risk-taking modulation tasks considerably affected theta band asymmetry after task execution had been completed. At the same time, our behavioral results showed no significant correlation with resting-state frontal theta band asymmetry at baseline, indicating that these effects cannot be explained only by the resting-state frontal asymmetry or by changes in asymmetry due to the stimulation.

The stimulation frequency specificity of our significant behavioral findings, however, confirming our *a priori* hypothesis that specifically theta, not gamma or sham, neurostimulation should affect risk-taking behavior, clearly represents supporting evidence for the functional relationship between theta band stimulation and risk-taking regulation driven by a reduction in sensibility to reward. This work also contributes to the understanding of the frontal areas' interaction in the regulation of risk-taking behavior as much as the role of theta band oscillations in this process. Moreover, it gives insights into the causes of individual differences in risk-taking, granting the analysis of frontal resting state brain activity a potential role in inferring differences in individual risk-proneness. This can be used in the construction of more accurate economic models of risk-taking.

Moreover, these findings can potentially contribute to the development of diagnosis and intervention techniques for patients with abnormal risk-taking behavior since this is characteristic of a range of psychiatric and neurological disorders (Rao et al., 2008). For example, the use of theta band stimulation might be an interesting tool to compensate increases in risk-taking behavior due to the use of L-dopa in patients with Parkinson's disease (Cools et al., 2003; 2002) or help patients with attention deficit/hyperactivity disorder (ADHD), which is known to be associated with abnormal risk-taking behavior (Pollak et al., 2019). Nevertheless, these suggestions should be explored in future studies.

3.1. Conclusion

Although it is widely accepted that the DLPFC has an important role in risk-taking regulation, it is not clear how the recruitment of this area occurs in the presence of risk. Theta oscillations are potentially responsible for neuron communication when cognitive control is needed (Cavanagh and Frank, 2014). In our study, we provided empirical evidence for the direct functional relationship between prefrontal theta band activity and risk-taking regulation using high definition theta band tACS with gamma band entrainment and sham as control. A significant reduction in risk-taking behavior was observed after theta band, but not gamma band or sham tACS over the left DLPFC, confirming the specific role of theta frequencies in risk-taking behavior regulation. Such findings indicate that prefrontal theta band oscillations are potentially the basis for communication between frontal areas during risk-taking

regulation.

4. Experimental procedure

4.1. Participants

Thirty-two healthy, right-handed students (16 female, mean age 23.8 years, range 18–31 years, SD = 3.45) participated in this study. All participants had normal or corrected-to-normal vision and gave written informed consent after being introduced to the experiment. They were screened for tACS safety, following the recommended procedures of Antal and colleagues (2017) (Antal et al., 2017), screening for, e.g., skin diseases, implants, neurological disorders, pregnancy, and medication.

The study was approved by the Ethics Review Committee Psychology and Neuroscience (ERCPN) of Maastricht University, The Netherlands (ERCPN 188_07_02_2018). Participants were compensated, in the form of vouchers with monetary value, based on the choices they made and luck in the risk-taking task and for participating in the experiment. The stimulation was well tolerated by 31 out of 32 participants. One participant reported skin redness in the area of the stimulation after participating in session 1 and therefore decided to stop participation in the experiment. The results of this participant were excluded from the analyses.

4.2. Procedure

Each participant received theta band (6.5 Hz), gamma band (40 Hz), and sham tACS in three separate sessions. The sessions were separated by an average of seven days (+/-1) to avoid carry-over effects. Fig. 4 provides an overview of our procedure and experimental design. The order of stimulation conditions (interventions) was randomized across participants.

Participants were invited to the laboratory, where they reviewed the participant information and signed the safety pre-experimental check and the written consent form. In each session, participants were informed about the experimental procedures and task and positioned at the workstation where the tACS and EEG electrodes were placed. EEG was measured before and after the stimulation. During tACS, participants had to perform the computerized MGT.

Participants were informed at the beginning of the session that by the end of it, one random trial of the task would be selected for payment. They were asked to use an online random number generator to determine the number of the trial that would be paid out. This was done in each of the three sessions. During the task, experimental currency was expressed as points. Every point earned in the selected trial was converted to € 0.10. All participants also received a participation fee of € 7.5 per hour (1.5 h per session). The payments varied between € 33.75 and € 63.75 and were made only after the third session. All task details and payment rules were explained before the task (Fig. 4).

4.3. Maastricht Gambling task (MGT)

A customized experimental protocol to elicit and assess risk-taking behavior was developed based on the widely used "risk task" (Rogers et al., 1999), also known as the Cambridge Gambling Task (CGT). The CGT is a valid measurement of risk-taking behavior (Deakin et al., 2004; Yazdi et al., 2019), controlling for impulsivity, and has been used in multiple studies using noninvasive brain stimulation (Boggio et al., 2010; Fecteau et al., 2014; Knoch et al., 2006; Valasek et al., 2010). However, the CGT does not control for memory and wealth effects because the trials are not independent, meaning that participants carry gains and losses from the previous trials; moreover, it is confounded by loss aversion as participants can lose points during the task.

Therefore, we developed a revised protocol, the MGT. This computerized task presents six boxes (see Fig. 5 for an example screen) to the participant, which can be colored either pink or blue. The number of pink boxes is randomized and ranges from 1 to 5, with the remaining boxes being blue. One of the colored boxes hides a token (represented by a yellow X), and the participant has to guess the color of the box that hides the token by choosing left (pink) or right (blue).

Each color has a different bet value representing the potential earnings if the chosen color is correct (hit). A wrong guess results in zero payoff. For example, in Fig. 5, the trial offers a chance of 3/6 (50%) of earning 50 points if pink is chosen and 3/6 (50%) of earning 100 points if blue is chosen. The bet values were selected randomly among five different values (5, 25, 50, 75, or 100) for each color in each trial independently. The participant's goal was to obtain the maximum of points in each trial. To remove the impact of loss aversion, the MGT does not allow for losses. Trials have no inter-dependency in the MGT. Payoffs are calculated for each trial independently and are not cumulated over

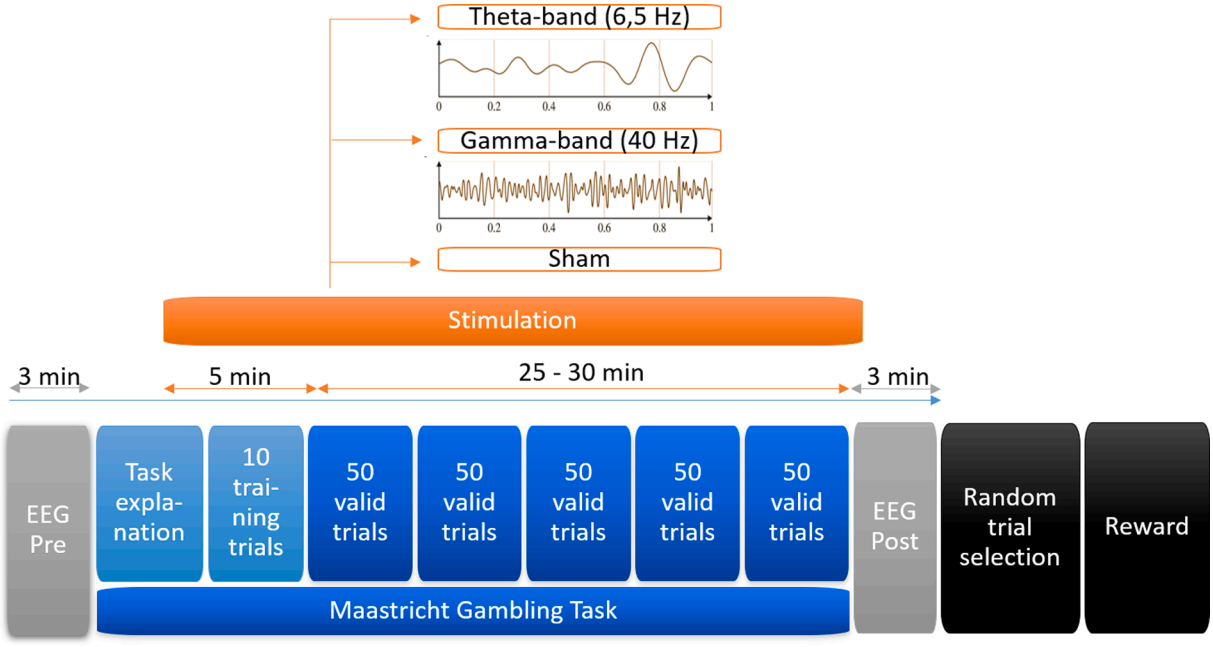

Fig 4. Experimental Design. General experimental procedures showing timing, EEG recordings, task presentation, and experimental conditions.

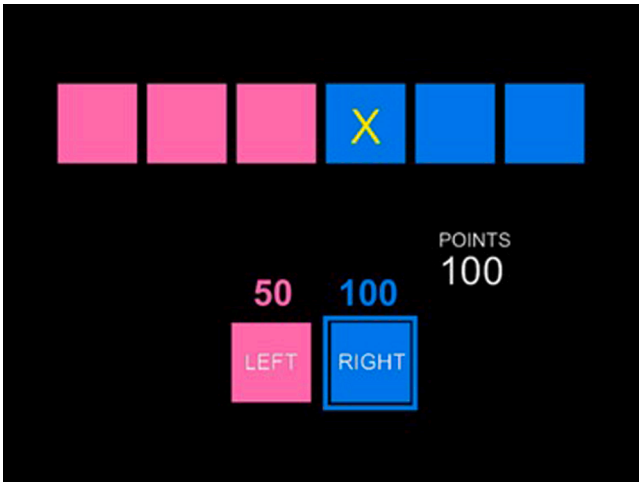

Fig. 5. Maastricht Gambling Task (MGT). Example screen presenting a trial where choosing blue offers a payout of 100 points with a probability of 3/6 (EV = 50) and pink offers a payout of 50 points and a probability of 3/6 (EV = 25). In this example, the participant chose the highlighted option (blue), and the token (yellow x) was revealed to be hidden behind one of the blue boxes. In this example, the participant gained 100 points, presented in white.

trials. This avoids memory and wealth effects.

Finally, participants see all the possible combinations of the five different bet values with the different probabilities, resulting in 125 unique trials; therefore, participants can perceive that there is no deception and that all possibilities are randomly assigned. Each trial is displayed twice, which yields a total of 250 trials in random order to guarantee consistent results. Participants had an average of consistency of 100% in the probabilities chosen and 93% (standard deviation 4%, median 93%) in terms of risk-taking and average value chosen across the repeated trials. No participant made significantly inconsistent decisions comparing the two repetitions of each trial in a session.

The tokens' location, color distribution, and bet values are determined independently and randomly across trials. With this, we guaranteed that there was no deception and full randomization. This also minimizes the chance of any specific strategy development. All prticipants were informed explicitly that there was no winning strategy since all results were random.

When a participant chooses a color, the choice is highlighted, and the position of the token is revealed (Fig. 5). Therefore, in this same example, if the participant had chosen blue, and the tokens were hidden behind a blue box, she/he would receive 100 points (as indicated in the white text on the right).

To gain more insight into the different types of trials, we divided them into three clusters according to the differences (or contrasts) in expected values offered by the two options (pink and blue), which could capture the difficulty of making a choice in the trial. The lower this difference, the more difficult it is for a subject to make a choice. This led to the division of trials into the following clusters: low, medium, and high contrast. In our analysis, we excluded trials with no difference in expected value since this group of options includes fewer trials than the remaining clusters and would not allow balanced analyses. Trials with one strictly dominant option, meaning (for simplicity) trials where the options have differences in expected value $> |65|$, were excluded. This exclusion was made since these were considered non-informative because these choices are considered obvious and would hardly be affected by any environmental or intrinsic factor. In total, 204 out of 250 trials were analyzed per session. The cluster division can be seen in detail in the S1 Table.

4.4. Transcranial alternating current stimulation (tACS)

We aimed at stimulating the left DLPFC. A small circular (diameter: 2.1 cm, thickness: 2 mm) electrode and a large (outer diameter: 11 cm; inner diameter: 9 cm, thickness: 2 mm) rubber ring tACS electrode (neuroConn, Ilmenau, Germany) were placed using conductive gel (Ten20 conductive Neurodiagnostic electrode paste, WEAVER and company, Aurora, CO, USA) onto the left DLPFC, with the small electrode positioned over F3 (based on the international 10–20 EEG system) and the large electrode around it. Electrode positioning and tACS simulation were modeled with SimNIBS (Thielscher et al., 2015), as shown in Fig. 6.

This ring electrode montage enables a higher spatial focality compared to standard rectangular electrodes (Kuo et al., 2013). Alternating current was applied using a neuroConn DC-stimulator with remote triggering (neuroConn, Ilmenau, Germany) and DataStreamer software (ten Oever et al., 2016), for which we created stimulus protocols on Matlab2018b (The Mathworks Inc., Massachusetts, USA) for each condition. Stimulation frequency and intensity were set to 6.5 Hz (theta-range stimulation) and 40 Hz (gamma-range stimulation), and a stimulation intensity of 1.5 mA peak to peak, phase offset set to 0 and 100 cycles were used for ramping up. Intensities and frequencies were defined based on settings used previously in similar experiments (Santarnecchi et al., 2019; Sela et al., 2012). For the sham tACS, the current was ramped up at a 6.5 Hz frequency for 30 s and ramped down immediately after. The impedance of the tACS electrodes was kept below 15 kΩ during stimulation. The average stimulation time lasted 30 min. Participants were blind to the stimulation protocol and the experimental hypotheses. Questionnaires applied after the experimental session confirmed that participants were unaware of the stimulation protocol.

4.5. Electroencephalography (EEG)

EEG electrodes were positioned according to the 10–20 international EEG system around the stimulation site (F1 and F5), contralateral to the stimulation site (F2 and F6) and on the parietal cortex (P5 and P6), with Cz being used as reference and the left mastoid used as ground. EEG measurements were done immediately before and after the tACS, each lasting three minutes, to measure resting-state theta band activity (measurement before the stimulation) and the effects of the entrainment (after stimulation). Participants were asked to stay with their eyes closed, relaxed and to avoid any movement.

Data were recorded (DC-200 Hz, sampling rate 500 Hz) with a BrainAmp Standard EEG amplifier and the BrainVision Recorder software (BrainProducts GmbH, Munich, Germany). Impedance levels were kept below 15 kΩ. Offline preprocessing was conducted using the Fieldtrip toolbox (Oostenveld et al., 2011) and custom Matlab scripts. EEG recordings were low pass-filtered in the analog domain (cutoff frequency: 250 Hz) and then digitized (sampling rate: 1000 Hz). Offline preprocessing was performed with a notch-filter (50 Hz) to remove electrical noise and demean the data over the full dataset. After that, it was segmented into 90 trials of two seconds each. Trials with high variance and excessive noise were excluded by visual inspection and variance analyses.

4.6. Statistical methodology

To assure transparency and facilitate the reproducibility of our study, all data collected and codes used to analyze them are available at https://doi.org/10.17632/vtz4vt9z5w.1. We analyzed the four following different behavioral dependent variables: 1) Risk, 2) Probability scores 3) Value, and 4) Response time.

4.6.1. Risk

The measure of risk-taking behavior should be dependent on both

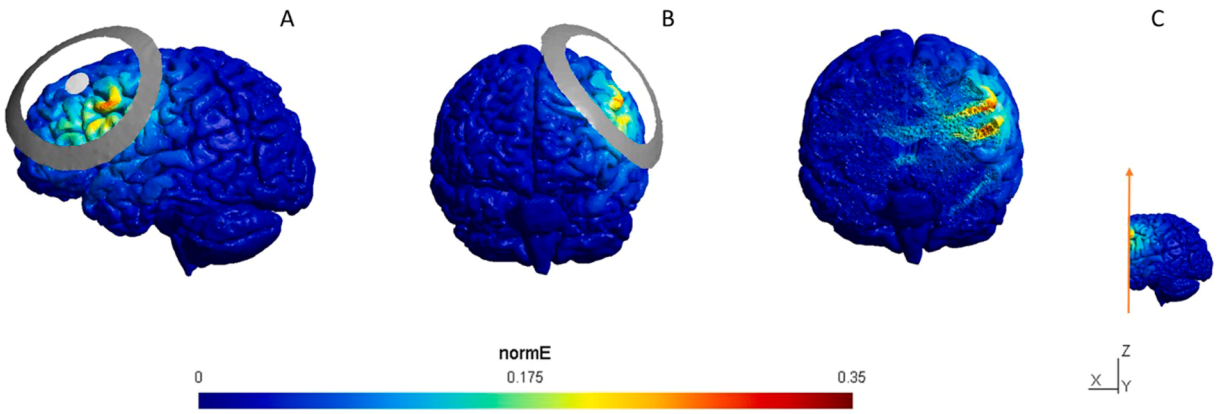

Fig. 6. SimNIBS tACS Simulation. Left lateral (A) and frontal (B) view of the stimulation and coronal cut at F3 to show the potential subcortical reach of the stimulation (C). Colors stand for the normalized electric field (0–0.35), meaning that the red areas are the areas where the electric stimulation has a higher incidence.

the probabilities of outcomes and the value associated with each outcome. In our experiment, betting on color X (X = blue or pink) in a trial with probability p and a payoff of x would have an expected payoff of xp. For instance, when choosing pink, the probability of being correct (a hit) and getting the reward is equal to the proportion of pink boxes during that trial, and the probability of being incorrect and getting no reward is equal to the proportion of blue boxes. Therefore, the expected payoff from choosing color X in a trial is given by the following:

$$E(X) = xp \tag{1}$$

For example, in a trial with one blue box with a bet value of 100 and five pink boxes with a bet value of five, the expected payoff for blue and pink are, respectively, 16.67 and 4.17. This makes blue more attractive for a risk-neutral participant. Therefore, an option is strictly dominant for a risk-neutral participant if it has a higher expected payoff.

The measure of risk takes into account the level of variation (Tobler et al., 2007). The variance of payoffs from choosing color $X$is given by the following:

$$Var(X_i) = \sum p(x - E(X))^2 \tag{2}$$

For example, in a trial with one blue box with a bet value of 25, and five pink boxes with a bet value of five, the expected payoffs of both options are the same, 4.17. However, the variance of blue (86.81) is much higher than that of pink (3.47). Therefore, the option blue is considered riskier than pink. Therefore, for two bets with the same *expected value*, the one with a larger variance is considered riskier. From variance, we calculated standard deviation (SD) as our measure of risk-taking behavior (e.g., Myerson, 2005), which is our main dependent variable, from now on referred to as "Risk."

$$Risk = SD = \sqrt{Var(X)} \tag{3}$$

4.6.2. Probability scores

Previous studies have considered only the choice of specific outcome probabilities as an indicator of risk (Boggio et al., 2010; Fecteau et al., 2007b; Knoch et al., 2006), meaning that in these studies, a choice is typically considered risky if the probability is below 50% and safe if its probability is above 50%. To allow a more refined analysis of participant's preferences of probabilities, they were transformed into a scale ranging from −2 to 2. The choice of a higher probability was classified with a negative score and that of a lower probabilities received a positive score. In simple terms, these scores indicate that options with a higher level of uncertainty have positive scores, while safer options have negative scores. These probability scores can be seen in Table 1.

4.6.3. Value and response time

To analyze the average value chosen by a participant in each session, their choices of bet values independent of the trial result (being correct or incorrect) were averaged. That variable is named Value. Furthermore, response times (RT) were also recorded for every decision.

Table 1
Probability Scores. Higher scores indicate that participants chose the trials with lower probabilities (risk prone), while lower scores indicate that participants chose higher probabilities (risk averse). For example, if a participant chooses blue in a trial where the distribution of blue boxes is 1/6 (and pink boxes 5/6), the participant would receive a score of 2, indicating that the participant chose the lowest probability possible. If in this same trial the participant chooses pink, the score would be −2, indicating that this participant chose the highest possible probability.

Pink	Blue	Choice	Probability
5	1	Blue	2
1	5	Pink	2
4	2	Blue	1
2	4	Pink	1
3	3	Pink	0
3	3	Blue	0
4	2	Pink	−1
2	4	Blue	−1
5	1	Pink	−2
1	5	Blue	−2

4.6.4. Behavioral data analyses

The behavioral data were preprocessed using custom Matlab (The Mathworks Inc., Massachusetts, USA). We performed a series of linear mixed model analyses to estimate the effects of stimulation (sham, theta, and gamma) on risk-taking behavior. Our final models were fixed effects models, with participant-specific random effects. All the analyses presented normally distributed residuals and showed no heteroscedasticity, and no observations were removed as outliers.

Overall, we constructed linear mixed models where each observation is a unique subject-cell pair. Each cell is a unique combination of session and contrast. That is, three sessions by three levels clusters (LC [low contrast], MC [medium contrast], and HC [high contrast]), resulting in nine unique observations (cells) per subject. The resulting models can be represented as follows:

$$Y_{ij} = (\gamma_0 + u_{0i}) + \gamma_1 Stim_{ij} + \gamma_2 Cluster_{ij} + \varepsilon_{ij}$$

Y_{ij} stands for each of the behavioral outcome variables; i stands for the i-th participant, and j represents the j-th cell; γ_0 stands for fixed effect intercept; u_{0i} stands for the subject-specific random effect; *Stim* stands for Stimulation condition (sham, theta, gamma); and *Cluster* stands for the three different levels of contrast of the trials (low, medium, high contrast). *Stim* and Cluster are subject-cell specific, hence the subscript ij.

To analyze the effects of stimulation on risk-taking behavior, measured as the average standard deviation of the chosen option (as described above), we fitted a linear mixed model, estimated using maximum likelihood (ML) and compound symmetry heterogeneous (CSH) covariance structure to predict Risk, with Stimulation and Cluster as factors (formula = Risk ~ Stimulation + Cluster + Stimulation * Cluster).

The analyses of the effects of stimulation on the probability scores used reduced maximum likelihood (REML) and heterogeneous Toeplitz (TPH) covariance structure to predict Probability scores with Stimulation and Cluster as factors (formula = Probability score ~ Stimulation + Cluster + Stimulation * Cluster).

To estimate the effect of stimulation on the average values, we fitted a linear mixed model estimated using REML and CSH as covariance structure to predict Value with Stimulation and Cluster as factors (formula = Value ~ Stimulation + Cluster + Stimulation * Cluster).

Finally, to analyze the participant's RT, we used a linear mixed model with RT as the dependent variable, estimated using ML and TPH as covariance structure, with Stimulation and Cluster as factors (formula = RT ~ Stimulation + Cluster + Stimulation * Cluster).

We checked the correlation among the behavioral dependent variables and checked the robustness of our results for each behavioral dependent variable when using appropriate controls of other behavioral outcomes. These controls did not affect the main results, which were confirmed with additional repeated measures ANOVA analyses, omitted here for conciseness.

4.7. EEG analyses

We preprocessed the data separately for low (1–20 Hz) and high (20–90 Hz) frequencies. For low frequencies, a fast Fourier transformation was performed with hanning tapers and output frequencies between 1 and 20 Hz. For high frequencies, a fast Fourier transformation was performed with discrete prolate spheroidal sequences (DPSS) tapers, a smoothing factor of 5 Hz, and output frequencies between 20 and 90 Hz. Then, the data were log normalized to control for discrepancies driven by individual variability (Smulders et al., 2018).

To look for differences in theta and gamma power before and after the stimulation protocols, the power spectra were averaged for the pre- and post-stimulation measurements. Theta band was defined between 5 and 8 Hz, with 1.5 Hz above and 1.5 Hz below the stimulation frequency (6.5 Hz). Gamma band was defined between 35 and 45 Hz, with 5 Hz above and 5 Hz below the stimulation frequency (40 Hz). Since gamma frequencies include a greater frequency range, we opted for a greater range (5 Hz instead of 1.5 Hz) around the stimulation frequency.

Theta and gamma power were analyzed for all channels pre- and post-stimulation, with focus on the frontal left channels (F1 and F5) around the stimulation focus and the frontal right channels (F2 and F6) contralateral to the stimulation.

To investigate whether a change in the hemispheric relationship in theta power took place, we calculated the average of the theta power in the right hemisphere minus the average in the left hemisphere, named frontal asymmetry (right–left) (Gianotti et al., 2009). Moreover, we compared the changes in theta as well as gamma power in the parietal channels before and after stimulation to analyze how focal the stimulation effects were.

The effects of stimulation in each condition were compared within participants for an interval of three minutes, followed by a *post hoc* analysis of the first minute after stimulation to investigate in detail possible fading effects. Moreover, a time frequency analysis was performed to provide a clear view of the power changes across frequencies over time in each condition. Signal processing and EEG data preprocessing were conducted using Matlab (The Mathworks Inc., Massachusetts, USA) custom scripts and the Fieldtrip toolbox (Oostenveld et al., 2011). The difference in theta power across conditions was correlated with the behavioral results using both the theta-asymmetry before stimulation as a covariate and the changes in theta and gamma frequencies as dependent variables by performing a repeated measures ANCOVA with Bonferroni correction.

CRediT authorship contribution statement

Aline M. Dantas: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Data curation, Writing - original draft, Visualization, Project administration. **Alexander T. Sack:** Methodology, Resources, Writing - review & editing, Supervision. **Elisabeth Bruggen:** Methodology, Resources, Writing - review & editing, Supervision. **Peiran Jiao:** Methodology, Resources, Writing - review & editing, Supervision. **Teresa Schuhmann:** Conceptualization, Methodology, Resources, Writing - review & editing, Supervision, Project administration.

Acknowledgements

We would like to thank our research assistant Kira Temme for her help during data collection.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brainres.2021.147365.

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