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EFFECTS OF EXCITATORY TRANSCRANIAL MAGNETIC STIMULATION ON
DECEPTIVE BEHAVIOUR

Master's thesis

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Running head: TMS effects on deceptive behaviour

Tartu 2018

Effects of excitatory transcranial magnetic stimulation on deceptive behaviour

Abstract

The present study investigated the effects of excitation of the dorsolateral prefrontal cortex (DLPFC) with repetitive transcranial magnetic stimulation (rTMS) on deceptive behaviour. The event-related potential (ERP) component P300 is well known as a neural marker of deception. P300 amplitude was examined in response to critical, familiar, and neutral stimuli in a task similar to the concealed information test. The electroencephalography (EEG) of 13 volunteers was recorded combined with rTMS. We did not find a difference in response to rTMS between right and left DLPFC as initially expected. However, TMS elicited a higher mean P300 amplitude to the critical stimulus compared to sham condition. Therefore, noninvasive prefrontal cortex excitation by rTMS can be used to increase the sensitivity of P300 to critical items in an analogue of the concealed information test.

Keywords: deception, P300, dorsolateral prefrontal cortex, repetitive transcranial magnetic stimulation

Ergastava transkraniaalse magnetstimulatsiooni mõju petukäitumisele

Kokkuvõte

Käesolevas magistritöös uuriti dorsolateraalset prefrontaalkoort (DLPFC) korduva transkraniaalse magnetstimulatsiooniga (rTMS) ergastamise mõju petukäitumisele. Sündmuspotentsiaalide komponenti P300 kasutatakse petukäitumise neuraalse indikaatorina teatud tüüpi eksperimentides. Uurisime P300 amplituudi vastusena kriitilistele, familiaarsetele ja neutraalsetele stiimulitele varjatud informatsiooni testi. Uuringus osalesid 13 vabatahtlikku, kelle EEG salvestati kombineerituna magnetstimulatsiooniga. Me ei leidnud rTMSi mõju erinevust paremas ja vasakus DLPFCs. Samas, TMS suurendas vastust kriitilisele stiimulile võrreldes sham-stimulatsiooniga. Seega, mitteinvasiivset prefrontaalkoort ergastamist rTMS-iga on võimalik kasutada P300 tundlikkuse tõstmiseks varjatud informatsiooni testi analoogis.

Märksõnad: petukäitumine, P300, dorsolateraalne prefrontaalkoor, korduv transkraniaalne magnetstimulatsioon

Introduction

Throughout history, deceptive behaviour has been studied with different methods, such as observing people's behaviour, analysing their speech, measuring brain activity and physiology associated with deception (Granhag, Vrij, & Verschuere, 2015, p. 16).

Unfortunately, a lie detector that would reliably detect a lie has not been found to this day and people's ability to detect deception by observing behaviour or listening to speech is limited. Based on meta-analyses, people achieve 54% correct lie-truth judgments on average, being more accurate in judging audible than visible lies (Bond & DePaulo, 2006). It is important to reveal the inner workings of deception (Ganis & Keenan, 2009) and that is one of the reasons why many researchers have turned their attention to studying the cognitive processes underlying deceptive behaviour (Bachmann, 2015; Granhag et al., 2015).

Attempts to detect concealed information using recording of physical indices have a rather long history and more recently the psychophysiological detection of concealed information has been extended by the measurement of brain potentials and functional magnetic resonance imaging (Ambach, Bursch, Stark, & Vaitl, 2010). Functional neuroimaging techniques such as positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) can assess neural response during the actual performance task (Abe, 2011). Over the past decade, researchers have used functional neuroimaging to investigate the neural correlates of deception, especially the processes of inhibiting honest response and generating dishonest ones (Abe, 2011). The brain imaging technology nowadays has allowed to show that the neurobiological markers of the cognitive processes underlying deception and truthful behaviour are indeed different (Ganis & Keenan, 2009).

In this paper, *lying* and *deceiving* are used as synonyms, as there is reason to believe that the neural processes of deceptive behaviour (including lying) are universal (Spence et al., 2004; Abe, 2011).

Brain areas connected to deception

Deception is one of the higher order mental activities and it is likely to involve multiple cognitive processes (Spence et al., 2004). It is not fully understood whether lying occurs in a certain area of the brain or are there specific mechanisms in the brain that underlie deception. Several researchers have found connections between deceptive behaviour and the dorsolateral prefrontal cortex (DLPFC; Spence et al., 2004; Karton, Palu, Jöks, & Bachmann, 2014;

Karton & Bachmann, 2017; Priori et al., 2008; Ito et al., 2012). The prefrontal cortex is associated with adaptive behaviour in novel or difficult circumstances (Spence et al., 2004). Brain imaging studies have shown that lying and deceptive behaviour increase the activity of executive functions areas in the left DLPFC and in the right anterior prefrontal cortex (Abe, Suzuki, Mori, Itoh, & Fujii, 2007). Executive functions include problem solving, planning, initiating and inhibiting behaviour, and manipulation of useful information (e.g. probable consequences of a lie) (Gombos, 2006; Christ, Van Essen, Watson, Brubaker, & McDermott, 2009). Therefore, executive functions (inhibition, working memory etc.) are central processes in producing deceptive responses (Abe et al., 2007) and succeeding in deception might be related to better cognitive control (Spence et al., 2004). Activity in the DLPFC has also been associated with three different aspects of executive control: working memory, inhibitory control, and task switching (Christ et al., 2009). Furthermore, it is also important to view left and right DLPFC separately. The left DLPFC is associated with reality monitoring, strategic behaviour, and executive functions (Abe, 2011; Ito et al., 2012) while the right DLPFC is associated with cognitive control, behavioural inhibition, and avoidance (Knoch & Fehr, 2007; Shackman, McMenamin, Maxwell, Greischar, & Davidson, 2009). Deception is based on several cognitive processes that are linked to the same region (Sip et al., 2010) that may be active without deception as well (Sip, Roepstorff, McGregor, & Frith, 2008), making it difficult to determine whether lying is related to a specific brain region or has its specific brain mechanism.

Noninvasive brain stimulation

The causal relations connected to lying have been studied much less than correlative relations (Ganis & Keenan, 2009). Based on the results obtained with neuroimaging methods (e.g. fMRI, MRI) it is not possible to assertively state that lying causes certain brain activity or vice versa. Deception is a complex act that includes several cognitive processes and functions (Buller & Burgoon, 1998; Abe et al., 2007; Priori et al., 2008; Sip et al., 2008; Christ et al., 2009; Abe, 2011; Verschuere, Schuhmann, & Sack, 2012; Karton, Palu, Jöks, & Bachmann, 2014). Research methods that allow us to observe causal effects are mainly performed with two noninvasive neurostimulation techniques: transcranial direct current stimulation (tDCS) or transcranial magnetic stimulation (TMS). The importance of TMS in brain research has rapidly increased and it is widely used for noninvasive modulation of cortical function (Kaminski, Korb, Villringer, & Ott, 2011). TMS is being increasingly combined with other

brain imaging and neurophysiologic techniques including fMRI and EEG (Rossi, Hallett, Rossini, Pascual-Leone, & Safety of TMS Consensus Group, 2009).

The main advantage of using TMS is the possibility to study causal relations by exciting or inhibiting the functions of certain brain region and the change in behaviour is recorded. Therefore, brain imaging and noninvasive brain stimulation can be viewed as complementary methods. By using brain imaging we can detect more active brain regions during deceptive behaviour and by using TMS we can study if these regions have any causal relations in that behaviour (Kähkönen, Komssi, Wilenius, & Ilmoniemi, 2005; Bachmann, 2015).

TMS is a noninvasive, safe, and well-tolerated method (NICE, 2014; Dodick, Schembri, Helmuth, & Aurora, 2010; Lipton & Pearlman, 2010) and a useful tool for neurostimulation of the cortical areas (Brighina et al., 2004). It is based on the principle of electromagnetic induction of an electric field in the brain, changing brain activity in a specific part of the cortex for a short term and having behavioural consequences (Rossi et al., 2009). In particular, rTMS allows to modulate cortical activity: at low frequencies, it has an inhibitory effect (Chen et al., 1997), whereas at high frequencies, it is able to enhance cortical excitability (Pascual-Leone, Valls-Sole, Hernandez, & Hallett, 1994). Karton & Bachmann (2011) found that a change can be observed in people's deceptive behaviour by applying low frequency (1 Hz) rTMS. In an experiment where the participants could freely choose when and how much they name seen objects, inhibiting the functional state of the rDLPFC increased the number of truthful answers whereas the opposite result occurred when IDLPFC was stimulated. Similarly, in a follow-up research, Karton, Palu, Jöks, & Bachmann (2014) found that in simple behavioural tasks excitation of the IDLPFC decreased lying compared to excitation of the rDLPFC, but inhibition had no different effects.

The Concealed Information Test

The Concealed Information Test (CIT), also known as the Guilty Knowledge Test (GKT; Lykken, 1959; Vershuere, Ben-Shakhar, & Meijer, 2011) aims at differentiating “guilty” subjects from “innocent” subjects and has been used in criminal investigations to examine whether a suspect knows crime-relevant information that only people involved in the crime should know (Matsuda, Nittono, & Allen, 2012).

The CIT is often combined with EEG to register event-related potentials (ERPs) related to deceptive behaviour (Farwell & Donchin, 1991; Matsuda, 2018). The endogenous components of ERPs, such as the P300 (also known as the P3), are generally considered as correlates of higher-order cognitive processes, e.g. attention, stimulus evaluation, or context updating (Polich, 1986; Duncan-Johnson & Donchin, 1982; Donchin & Coles, 1988). P300 is also regarded as a relevant electrophysiological marker in the studies of deception (Ambach et al., 2010; Rosenfeld & Labkovsky, 2010; Verschuere, Ben-Shakhar, & Meijer, 2011). Studies have shown clear differences in P300 amplitude between probe and irrelevant stimuli on several conditions. For example, the P300 amplitude is larger when the participants have prior knowledge about the details of the item they were to steal (Winograd & Rosenfeld, 2014). Cutmore, Djakovic, Kebell, & Shum (2009) showed that non-verbal stimuli are more effective in discriminating and detecting guilty knowledge in an oddball paradigm.

Objective

The objective of current study is to explore the causal effects of manipulation of the dorsolateral prefrontal cortex (DLPFC) with excitatory transcranial magnetic stimulation (TMS) on deception-related behavior. We expect to have the opposite results to the study by Karton and Bachmann (2017), where inhibiting the right dorsolateral prefrontal cortex decreased the P300 amplitude in response to the critical stimulus compared to the neutral stimulus.

The main hypotheses we tested are as follows:

1. Excitation (10Hz) of the right dorsolateral prefrontal cortex (rDLPFC) with repetitive transcranial magnetic stimulation (rTMS) increases the P300 to the critical stimulus.
2. Excitation of the left dorsolateral prefrontal cortex (lDLPFC) with repetitive transcranial magnetic stimulation (rTMS) does not change as much the P300 to the critical stimulus.

Method

Participants

The sample of our study was recruited opportunistically from the university environment. All subjects who participated in the study were healthy and had normal or corrected to normal vision. Written informed consent was obtained from all participants. The experiments were

approved by the Research Ethics Committee of the University of Tartu (approval nr 271/T-23). The participants did not receive any compensation for participation.

Overall, there were 20 subjects (9 male, 11 female) participating in the experiment. Data of 3 male and 4 female subjects were excluded due to excessive EEG artefacts or recording problems. The final sample consisted of 13 participants (6 male, 7 female; age range 20-30 years, mean age (*M*) 25.23 years, standard deviation (*SD*) 2.42 years). All of the subjects participated in the study on two separate days and received rTMS and sham stimulation both to the right and left DLPFC.

Equipment

Neuronavigation system NBS (Navigated Brain Stimulation) Nexstim Ltd. with figure-of-eight coil was used for the TMS stimulation. International extended 10-20 EEG electrode placement system was used to locate the DLPFC area of stimulation (Figure 1). The left DLPFC area is marked as F5 and the right is marked as F6. The computer monitor where the CIT-like task was presented, was SUN CM751U (1024 x 768 pixels, 100 Hz refresh rate).

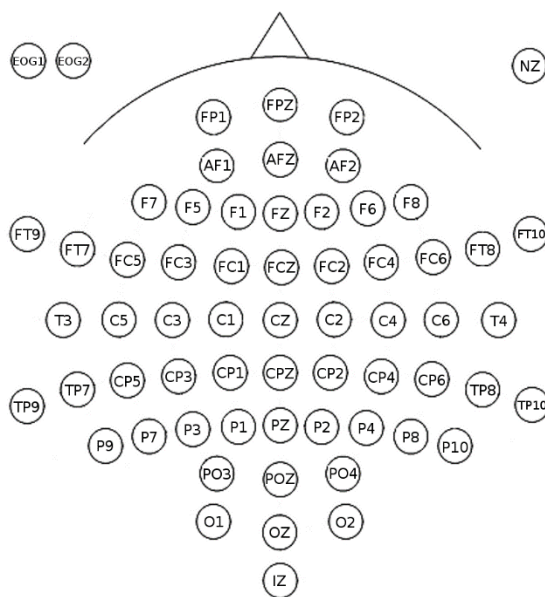


Figure 1. 10-20 EEG electrode placement system.

Experimental procedures

Each participant visited the laboratory on two separate days. Similarly to the work of Karton & Bachmann (2017), an analogue of the Guilty Knowledge Test (Concealed Information Test) was used as our experimental task. We used five small products (e.g., chewing gum,

chocolate bar, candy, etc.) that would be easy to “steal”. In each session, three products from these five were put in a box on a table next door. The experiment started with the subjects simulating a “shoplifting” scenario. For that, one of the three products had to be “stolen” and put in another box that the subjects brought to the room where the TMS/ERP experiment began. The purpose of the experiment as explained to the subjects was to discover “stealing” using EEG. The subjects were instructed to hide the “crime”-related knowledge.

Three types of stimuli were specified for the experiment: (1) two products that were *familiar* from the “stealing” episode, but not “stolen”, (2) the product corresponding to the one actually “stolen” (the *critical* product for each subject, could be different for each subject, the critical object was protocolled after the participant had finished the experiment), (3) two products that were new to the subjects (the *neutral* products that were not present in the box for “stealing” for that subject).

After the “thieving” episode, motor threshold (MT) was measured as a barely noticeable twitch of the thumb on the opposite side of the brain relative to the stimulation side EEG caps were fitted to the subjects, followed by the blocks of sham/TMS and CIT-like task stage of the experiment. All subjects received TMS stimulation to either side of the DLPFC on separate days. The stimulation blocks were given according to AABB/BBAA or BBAA/AABB design (sham - A and rTMS - B), balanced between sessions and subjects.

One session consisted of 4 blocks of the CIT-like task. Each block consisted of 100 stimuli presented on a computer screen in a random order. Each picture stimulus was presented for 1400 ms, 20 times per block. First a fixation cross was presented for 500 ms, followed by the picture stimulus and the third screen was a question “Do you have this product?”, to which the subjects were instructed to always answer “no” by pressing a response key on a standard computer keyboard. After responding the question screen disappeared and the subjects initiated the next trial by pressing the space bar. All items were presented in the middle of the screen.

Each block started with a train of 10 Hz rTMS pulses or sham stimulation pulses and the subjects received the TMS/sham stimulation after every four pictures (25 times during one block, 2x25 rTMS stimulations and 2x25 sham stimulations during both sessions). To mask the sound of rTMS clicks and reduce differences between real rTMS and sham, the

participants wore headphones during the experiment and white noise with clicks imitating the rTMS stimulation was played in the sham condition. Pure white noise was played in the rTMS stimulation. The stimulation intensity was set at 100% of the individual motor threshold. The intensity of stimulation used for different subjects ranged between 39% and 46% of maximal stimulator output.

All of the experiments were conducted at the cognitive psychology laboratory of the School of Law of the University of Tartu situated in Tallinn. Protocols were filled for every session of each subject, stating the date, participant's native language, handedness, and individual details of every session. After the experiments, the participants gave feedback about the critical stimulus and they were asked whether they remembered the products in the box. Most of the participants felt the difference in TMS and sham conditions but they were assured that this was only due to different TMS regimes.

EEG and data analysis

We used Nexstim eXimia EEG-system with 60 carbon electrodes cap (Nexstim Ltd.) for EEG recording. The EEG signals were referenced to a calculated mean electrode. The vertical electro-oculogram (VEOG) was recorded via two additional electrodes placed above and below participants' left eye. The system recording was filtered with 1 Hz for analysis. All recorded EEG data was analyzed with EEGLAB (Delorme & Makeig, 2004), an open-source Matlab (MathWorks, USA) toolbox.

Bioelectrical activity was recorded from 17 electrodes: frontal (AF1, F1, F5, F7, AF2, F2, F6, F8), parietal (P3, P4, PO3, PO4), temporal (TP7, TP8), and central (C3, C4, CZ). After the initial recording, data was cleaned from artefacts first with appropriate Matlab code and after that manually checked for additional artefacts. All trials contaminated by artifacts were discarded from further analysis. ICA was computed and for 4 participants 1 electrode was interpolated. For analysis, data was filtered with a 30 Hz low-pass filter. Data was segmented into trials from 0 ms to 600 ms relative to stimulus onset. Data were baseline-corrected with a -100 ms to 0 ms window prior to the stimulus onset. For ERP figures, data was additionally filtered with a 10 Hz low-pass filter.

Statistical analysis

All statistical analyses were performed with IBM SPSS Statistics 25. Repeated-measures analysis of variance (ANOVA) was used to assess the effects of our experimental conditions. Greenhouse-Geisser method was used to correct p -values if the sphericity assumption was violated according to Mauchly's test for sphericity. Only the corrected p -values are reported. Effect sizes are shown using partial eta-squared (η_p^2) for ANOVA. Post hoc comparisons were conducted using t-tests with Bonferroni's correction. For the paired-samples t-test, Cohen's d is reported as an estimate of effect size.

Results

First, a four-way repeated-measures ANOVA with the factors electrode group (frontal and parietal), stimulus type (neutral, familiar, critical), stimulation type (TMS and sham), and stimulation side (left and right) as within-subject factors was performed for assessing differences in P300 amplitude (the epoch used for mean P300 amplitude measurement was 300 ms to 500 ms). The main effect of electrode group was significant: $F(1, 12) = 84.935, p < .0001; \eta_p^2 = .876$ and shows that the mean amplitude of P300 was higher in parietal electrode group (Table 1). The main effects for stimulus type, stimulation type, and stimulation side were not significant ($F(2, 24) = 2.478, p = .133; \eta_p^2 = .171, F(1, 12) = 1.018, p = .333; \eta_p^2 = .078$, and $F(1, 12) = .124, p = .731; \eta_p^2 = .01$, respectively) showing that significant effects arise from interactions although not exactly as we hypothesized.

Table 1
P300 amplitude means (M) and standard deviations (SD) in electrode groups for all stimulus types.

Frontal electrodes		Neutral		Familiar		Critical		Total	
		M	SD	M	SD	M	SD	M	SD
Left+right		-0.99	2.47	-1.4	2.19	-1.47	3.38	-1.29	2.68
Stimulation	Sham	-1.35	2.31	-1.59	1.79	-2.16	2.46	-1.7	2.19
	TMS	-0.64	1.64	-1.21	1.98	-0.78	2.60	-0.88	2.07
Parietal electrodes		Neutral		Familiar		Critical		Total	
		M	SD	M	SD	M	SD	M	SD
Left+right		6.78	3.92	5.25	3.45	4.98	3.98	5.67	3.78
Stimulation	Sham	7.07	2.80	5.90	3.15	4.66	3.51	5.88	3.15
	TMS	6.50	3.55	4.61	2.91	5.30	2.77	5.47	3.08

There were significant interactions between electrode group and stimulus type ($F(2, 24) = 6.643, p = .005; \eta_p^2 = .356$). Paired-samples t-tests were carried out to investigate which

conditions differed significantly from each other (means in Table 1). There was a significant difference in the response to neutral vs. familiar stimulus in both frontal and parietal electrodes ($t(12) = 2.243, p = .045, d = .622$ and $t(12) = 4.322, p = .001, d = 1.199$, respectively). Nearly significant difference is evident in the parietal electrodes between neutral and critical stimulus ($t(12) = 2.079, p = .06, d = .577$). However, there was no significant difference between neutral and critical stimulus in the frontal electrodes ($t(12) = .633, p = .539, d = 0.176$) and between familiar and critical stimulus in frontal or parietal electrodes ($t(12) = -.387, p = .706, d = -.107$ and $t(12) = .177, p = .863, d = .049$, respectively).

There were significant interactions between electrode group and stimulation type ($F(1, 12) = 8.118, p = .015; \eta_p^2 = .404$). Means of P300 in response to different stimulation types within electrode groups are in Table 1. There was a near significant difference in P300 response in the parietal electrodes between TMS stimulation and sham stimulation ($t(12) = 2.157, p = .052, d = .598$), but there was no significant difference in the frontal electrodes between TMS and sham stimulation ($t(12) = -1.793, p = .098, d = -.497$).

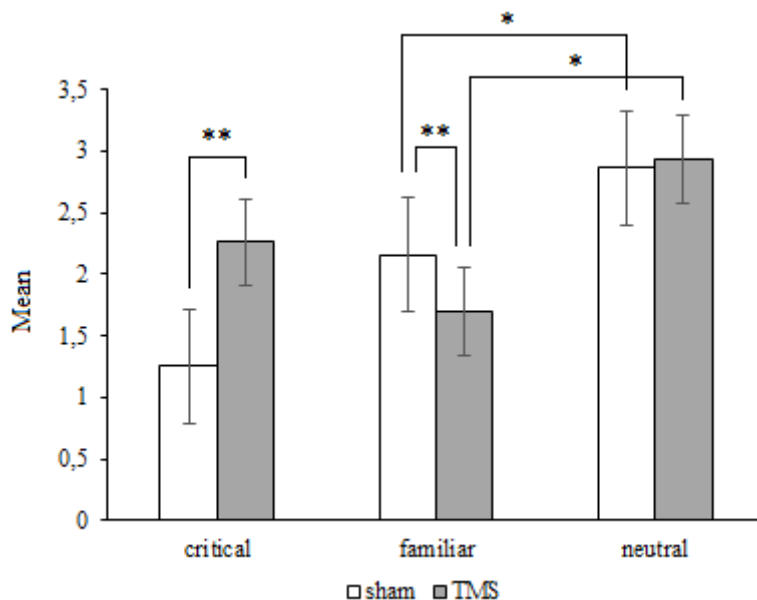


Figure 2. Mean amplitudes (μV) of stimulus types by stimulation condition, averaged over stimulation sides (left/right) and electrode groups (frontal/parietal). “Whiskers“ depict standard errors. Paired t-test differences: $*p < .05$, $**p = .06$.

Repeated-measures ANOVA also showed a significant interaction between stimulus type and stimulation type ($F(2, 24) = 5.264, p = .013; \eta_p^2 = .305$). Post-hoc analysis of means (Figure 2)

was conducted, paired-samples *t*-tests revealed that there was a near significant difference between the conditions of critical stimuli presented after TMS and sham stimulation ($t(12) = -2.074, p = .06, d = -.575$) and between the conditions of familiar stimuli after TMS and sham stimulation ($t(12) = 2.045, p = .063, d = .567$). Conditions of neutral stimuli (after TMS vs. after sham $t(12) = .027, p = .979, d = .007$) did not differ. In TMS condition, P300 was significantly different in response to familiar vs. neutral stimuli ($t(12) = 4.273, p = .001, d = 1.185$) but there was no significant difference in critical vs. neutral ($t(12) = .718, p = .487, d = 0.199$) and critical vs. familiar stimuli ($t(12) = -1.182, p = .26, d = -.328$). The same occurred in sham condition: familiar vs. neutral stimuli differed significantly from each other ($t(12) = 2.415, p = .033, d = .67$) while critical vs. neutral and critical vs. familiar stimuli did not ($t(12) = 1.741, p = .107, d = .483$ and $t(12) = .885, p = .394, d = .245$, respectively). Other comparisons showed insignificant results ($p > .113$). All other interactions were not statistically significant (all $F_s < 2.983$, all $p_s > .07$, all $\eta_p^2 < .199$).

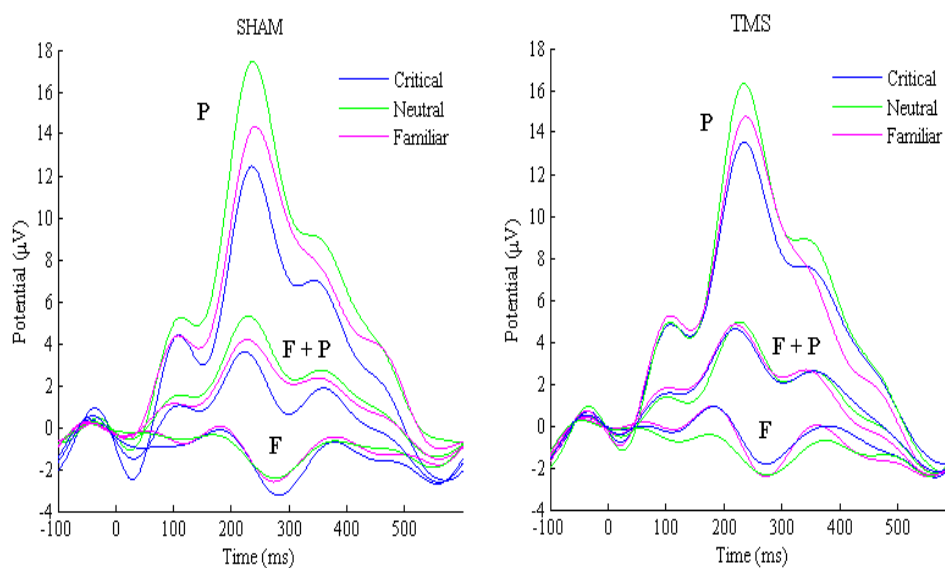


Figure 3. The grand average ERPs per stimulation conditions.

Discussion

The aim of this study was to examine the effects of repetitive transcranial magnetic stimulation (rTMS) over dorsolateral prefrontal cortex (DLPFC) on deceptive behaviour. More specifically, we intended to explore whether rTMS changes the P300 amplitude in a task similar to the Concealed Information Test (CIT) and whether this effect is different

depending on which hemisphere is stimulated. Karton and Bachmann (2017) have previously found that disrupting the functionality of DLPFC with 1-Hz rTMS strongly reduces the P300 amplitude in response to the critical items compared to familiar and neutral items. We expected that excitation of the right DLPFC with rTMS would increase P300 to the critical stimulus whereas excitation of the left DLPFC would have the opposite result. However, we did not find any differences in P300 amplitude between stimulation sides. There was a main effect of electrode group: parietal electrodes elicited a much higher P300 amplitude compared to the frontal electrodes, which is not a surprising result as P300 amplitude typically increases in magnitude from frontal to parietal electrode sites (Johnson, 1993). There was also a significant interaction between electrode group and stimulation type. We found that excitatory rTMS had a diminishing effect to P300 in parietal areas but not in frontal areas.

There was no significant main effect of stimulus type, but we did find some interesting interactions. Although we expected to find significant differences in the response to critical stimulus compared to other stimulus types, there were differences in P300 amplitude in response to familiar vs. neutral stimuli in both stimulation conditions but there were no differences between the critical stimulus compared to others. This is a very surprising result as the main idea in using different categories of stimuli is to show that brain activity in response to a visual stimulus that has a special meaning to the participant compared to a neutral one should be different. One explanation for this could be that the trials with critical stimulus were most contaminated with artefacts and therefore a large number of those trials were not included in the final data analysis. From the participants' point of view, the experiment took a lot of time (at least two hours on two separate days). During the experiment, fitting the EEG caps was the most time-consuming part and it might have been that by the time the participant could actually start with the CIT-like task, he did not remember the product he had taken. That was also one of the aspects in the feedback that the subjects lead our attention to and that could explain why we did not find a difference in P300 elicited by critical stimulus compared to neutral and familiar stimulus. The issue that the critical stimulus does not appear as critical to the participants in the CIT-like experiments is fairly common (Cutmore et al., 2009; Rosenfeld, Shue, & Singer, 2007).

We found that the highest P300 occurred to the neutral stimuli and the amplitude was lower for stimuli that the participant had seen before (familiar and critical). The reason for this could be that the neutral stimuli were also novel stimuli (not seen during the mock crime). It is known

from EEG studies that novel stimuli may elicit higher P300 amplitudes than familiar stimuli (Gonsalvez & Polich, 2002; Polich, 2007).

However, the mean amplitude of P300 did increase in response to critical stimulus in TMS condition compared to sham condition, meaning that excitation of the DLPFC did change P300 amplitude in the expected direction although the effect was not hemisphere specific. Interestingly, the opposite occurred in response to familiar stimulus.

There was also no significant main effect of stimulation type. An important part of a TMS experiment is masking the sham condition. For that, we used clicks that imitated TMS stimulation. This method is not perfect as TMS stimulation causes very specific physiological responses that do not occur in the sham condition with the imitations. That is why most of the participants could differentiate between the conditions as they also reported in the feedback following the experiments. Other possible methods for masking the sham condition include turning the TMS coil away from the head (Rotenberg, Horvath, & Pascual-Leone, 2014) or stimulating an unimportant area (Jung, Bungert, Bowtell, & Jackson, 2016). Unfortunately, these methods can still have an impact on the cortex. Newer TMS machines have a special coil that also imitates the physiological responses without stimulating the cortex (Rotenberg et al., 2014). Motor threshold (MT) is often used to minimize the physiological response and apply individual stimulation intensity. In the current study, stimulation intensity was set at 100% of the individual MT which was determined via observation of thumb movement. Although this type of MT measurement is widely used, it has also been found that visual observation yields significantly higher MTs compared to determination via electromyography (Westin, Bassi, Lisanby, & Luber, 2014). That might have contributed to the fact that participants could easily tell whether real TMS or sham stimulation was delivered but at the same time many TMS paradigms use an intensity as high as 120% of the individual MT (Rotenberg et al., 2014).

Even though we did not get the results initially expected, it is still noteworthy that the critical stimulus did elicit a higher P300 in response to TMS stimulation. This means that excitation of the dorsolateral prefrontal cortex with rTMS does have a facilitating effect on deceptive behaviour and the P300 sensitivity can be increased. The roles of right and left hemisphere in deceptive behaviour still remain unclear and need follow-up studies on a larger group of people and larger number of useful trials per condition. For further research, it is important to

consider using personal objects of other people as the critical stimuli in the experiments, as this could increase the emotional meaningfulness of the mock crime and critical stimulus. Developing functioning TMS protocols is necessary to improve our understanding of the brain processes related to deceptive behaviour. TMS protocols could have high practical value in cases where discovering deception is very important.

Acknowledgements

I am truly grateful to my supervisors Iris Tuvi and Inga Karton for their patience, support, and guidance.

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