

STIMULATING THE PERFORMANCE MONITORING NETWORK: EFFECTS OF
TRANSCRANIAL DIRECT CURRENT STIMULATION ON ERROR PROCESSING

by

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Dedication

This thesis is dedicated to those who have selflessly committed themselves to helping me reach my goals. Specifically, those who have invested their time, their love, and their energy to help me academically or to provide emotional support. First, to my family, especially my mother Carmen, father Romeo, and grandmother Celestina, for their fierce love and guidance throughout my undergraduate and graduate career. To my brother Joe, confidant Chris, and close personal friends Samantha and Katelyn, for always keeping me afloat and never letting me forget why I began this journey. To my thesis committee, including Dr. Pamela Greenwood, Dr. Matthew Peterson, and especially Dr. Craig McDonald, for their countless hours of guidance and understanding. Finally, I would like to specifically thank my mentors, Dr. Mark Neider and Dr. Chi Tran, for their tireless support, conviction in my abilities, and willingness to do whatever possible to facilitate my success.

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Abstract

STIMULATING THE PERFORMANCE MONITORING NETWORK: EFFECTS OF TRANSCRANIAL DIRECT CURRENT STIMULATION ON ERROR PROCESSING

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The human performance monitoring network (PMN) is a collection of neural components that modify behavior to generate successful task performance. However, activation of the involved cortical areas can hinder successful task performance when limited time is available to modify behavior. Presently, little is known about how the PMN functions under different time constraints. To investigate the link between the neural correlates of the PMN and commonly observed post-error behaviors, this study manipulated PMN activation via transcranial Direct Current Stimulation (tDCS) at the Medial Frontal Cortex (MFC). This study implemented a difficult visual discrimination task in which response-

stimulus intervals (RSIs) were varied, coupled with tDCS, to investigate the effects on performance monitoring of different RSI lengths. A within subjects design was used, in which participants took part in both the experimental (anodal) and the control (sham) stimulation conditions. Critically, anodal stimulation significantly improved overall accuracy. Analyses of post-error behaviors also showed that anodal stimulation produced a significant increase in post-error accuracy (PEA) and post-error slowing (PES). These findings are consistent with the notion that post-error behavioral adjustments facilitate better overall task performance when sufficient processing time is available. The findings also provide causal evidence for the role of the MFC in PMN activity and resulting post-error behavioral adjustments.

Introduction

The Performance Monitoring Network (PMN) is a critical component of human cognition. It is a specialized network of cortical and subcortical structures responsible for adaptive skills such as conflict monitoring, error-processing, and behavior correction (Botvinick et al. 2001). The PMN provides the ability to evaluate ongoing performance in the context of environmental or social factors and uses these evaluations to adjust behavior to better fit specific goals or criteria (Holroyd & Coles, 2002). The manner in which this PMN system performs assessments and corrections has been a major focus of the performance monitoring literature. More specifically, research has been focused on delineating the specific cognitive and neural processes underlying the successful assessment and modification of ongoing behavior. Presently, there is evidence that PMN activation acts as both a facilitator and a hindrance to performance. It has been shown that insufficient error-processing time often results in performance decrements following error commission (Jentsch & Dudschig, 2009; Dudschig & Jentsch, 2009). Yet, opposing findings have shown that performance also improved following failures (Botvinick et al. 2001).

Despite this dichotomy within the literature, current hypotheses suggest that performance decrements are generally a result of ongoing performance monitoring activity impeding task-related attention and sensory processing (Rabbitt, 1966; Ullsperger & Danielmeier, 2016; Notebaert et al., 2009). Specifically, ERP studies have found inverse relationships between ongoing error-processing and stimulus processing on subsequent trials following short response-stimulus intervals (RSIs), supporting the notion of an error-processing induced decrement and attentional bottleneck (Buzzell et al. 2017). This suggests that the PMN, a network dedicated to improving performance, actually hinders performance when behavioral modifications must be rapidly made.

Previous studies investigating decrements in error-processing have used transcranial direct current stimulation (tDCS) to modulate error-processing (Reinhart et al. 2015, Bellaiche et al., 2013). Findings show that DC stimulation can be used to increase (Reinhart et al., 2015; ERN) or reduce (Bellaiche et al., 2013; Pe) mechanisms of error processing. One critical remaining question that is addressed in this thesis concerns whether manipulation of PMN activation changes the time needed to process and correct errors. To that end, we used tDCS to manipulate PMN activation and better assess the degree of influence of stimulation and PMN activation on task performance at differing RSIs.

Background

i. Behavioral Indices of Error Processing

Error-processing is a critical decision-making task that engages multiple cognitive processes that work together to compare ongoing behaviors against previously defined standards or criteria (Steinhauser & Yeung, 2010). These evaluative cognitive processes are all facets of the Performance Monitoring Network (PMN) that combine to detect errors, signal error commission, and ultimately modify task-related behaviors in real time. Previous research has examined behavioral activity under various conditions to identify which behaviors reflected ongoing performance monitoring. Post-error slowing (PES; Rabbitt, 1966) is the tendency to respond more slowly following an error and has been commonly observed following error commission. PES has been associated with reduced performance on the trial after an error (Jentzch & Dudschig 2009; Buzzell et al., 2017). The PES has been claimed to be due to processing of an error on the previous trial during ongoing processing of the current trial. The specific processes proposed to underlie PES include attentional orienting to the error and changes in motor and/or sensory response thresholds (Ullsperger & Danielmeier, 2016; Notebaert et al., 2009). Through modulation of the PMN we can further delineate the nature of these decrements in PES.

In addition to PES is the post-error change in accuracy (PEA; Post-error accuracy). Both PEA and PES have stimulated much of the performance monitoring research. A marked increase in PEA has been attributed to increased cognitive control and allocation of attention (Maier et al., 2011). When observed in conjunction with increased PES, increased PEA has been claimed to be a definitive signature of post-error behavioral adaptation. In contrast, some studies have either not observed significant increases in PEA, or have reported significant decreases in PEA (Jentsch & Dudschig, 2009). This inconsistency in results suggests the relationship between PES and PEA is poorly understood and requires further assessment. PMN modulation through DC stimulation would serve as a mechanism by which to examine this link between the PES effect and PEA decrements.

ii. Influence of Time on Behavioral Indices

Manipulation of the length of response-stimulus intervals (RSI) has helped to better illustrate the connection between post-error slowing (PES) and post-error accuracy (PEA) effects. Studies show that insufficient processing time is often followed by an increase in error rates (decreased PEA) and an increase in response slowing (PES) (Notabaert et al, 2009; Laming 1979; Jentsch & Dudschig 2009). It has been argued that this interference is likely due to greater temporal overlap between error processing and subsequent stimulus processing (Jentsch & Dudschig, 2009). Extent theories also suggest the increased PES can be attributed to a conscious attentional shift from error processing of the previous stimulus to

processing the current stimulus and ultimately to adjusting performance parameters. This is assumed to be the case in situations where PEA shows a notable increase (Laming, 1968; Maier et al., 2011). Ullsperger & Danielmeier (2016) have suggested these behavioral effects can be attributed to either motor and sensory threshold changes following error detection or the reflexive recruitment of attention to error-correction following error detection. When the RSI is short, either of these processes could presumably interfere with task-related attention to the subsequent trial (Ullsperger and Danielmeier, 2016).

Until recently, only a few studies aimed at interpreting the relationship between PES and PEA have manipulated the duration of the response-stimulus interval (RSI) (Jentsch & Dudschig, 2009; Dudschig & Jentsch, 2009). Jentsch and Dudschig (2009) found that as RSI length increased PES decreased but was still present at long RSI's. Yet, Buzzell et al (2017) found minimal PES at long RSI. Thus, the influence of timing constraints on post-error behavior has not been clearly delineated. By manipulating the length of time between response to a stimulus and onset of the subsequent stimulus, this study aimed to define the point in time at which post-error decrements in subsequent task-related performance are produced and whether transcranial DC stimulation of the PMN would alter that point in time.

iii. Neural Correlates of Performance Monitoring

Event-related potential (ERP) data provide one measure of the connection between human cognition and its source cortical structures. Previous research

investigating the error-related negativity (ERN) and error positivity (Pe), two commonly studied neural indices of performance monitoring, have implicated the medial prefrontal cortex as a potential origin of these components. More specifically, source localization and imaging studies implicate the rostral and dorsal Anterior Cingulate Cortex (ACC) as the neural generators for these components (Van Veen & Carter, 2002). The ERN is believed to index a relatively automatic error detection process (ERN; Gehring et al., 1993), whereas the Pe is thought to index error awareness or the accumulation of evidence indicating that an error has occurred (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001).

A study by Buzzell et. al. (2017) used EEG and ERP techniques to assess differences in post-error behaviors and stimulus processing neural activity at varying RSI lengths (long and short). This research identified a negative correlation between Pe amplitude on the current trial and the P1 amplitude of the subsequent trial when RSIs were short but not long. This correlation, combined with the behavioral data, suggested to the authors that as conscious error perception of the previous trial increased (denoted by elevated Pe activity) sensory processing decreased on the following trial (denoted by a reduction in P1 amplitude). This was interpreted as indicating that short RSIs do not allow sufficient processing time. These data helped identify the possible neural underpinnings of common post-error behavioral activity.

iv. Modulating the PMN and Error-Processing: Connecting Neural Correlates to Modulation Methods

Previous transcranial Direct Current Stimulation (tDCS) studies claim to have effectively altered neural activity and significantly influenced error-processing abilities (Reinhart et al. 2015, Bellaiche et al. 2013, Harty et al. 2014). Through the application of excitatory (anodal) tDCS over the medial-frontal cortex (MFC) of schizophrenic patients, Reinhart et al. (2015) induced behavioral and neurophysiological changes in adaptive control (goal-directed behavior modification) resulting in normal error-processing (enhanced ERN) in the schizophrenic group. The patients' results were indistinguishable from the healthy participants, showing normal neural (frontal theta wave phase synchrony) and behavioral (PES) markers of cognitive control. Additionally, Bellaiche et al applied tDCS to the mPFC to test the efficacy of cortical modulation on error-processing behaviors and observed a reduction of Pe amplitude (degree of error processing) under inhibitory (cathodal) stimulation. These studies show that excitatory and inhibitory stimulation of MFC produce predictable changes in neural and behavioral markers of error processing.

The present study sought to better understand the relationship between the PMN and post-error behavioral adjustments that occur when the time between decisions is limited. Following error commission, when time to adjust task parameters, time to acknowledge error commission, and time to subsequent

stimulus onset overlap decisions must be made rapidly, possibly at the cost of subsequent stimulus processing. To assess the degree of influence of PMN activation, RSI length was manipulated and tDCS was applied to the MFC to noninvasively modulate performance-monitoring processes. We hypothesized that anodal stimulation would amplify post-error performance decrements as a result of stimulation-induced increases in performance monitoring activity conflicting with ongoing task-related attention. Specifically, we predicted that anodal stimulation would reduce performance following error trials with short response-stimulus intervals (RSI's), but have a beneficial effect on performance following error trials with long RSIs.

Methods

Participants

Twenty-six students from George Mason University participated in this study for course credit reimbursement via SONA-systems. Nine total participants were removed; four due to calibration errors, and five due to low “overall,” “same,” or “different” accuracies (accuracy two SD below the mean; defined within stimulation condition). A total of 17 participants (11 male, M age = 20.41, SD = 2.12) remained for behavioral analysis. All participants were right handed, had no history of neurological or psychological disorders (including history of seizures or concussions), were not taking any central nervous system altering medications within 24 hours of the task, and were screened for normal or corrected to normal visual acuity and color vision. All participants provided informed consent prior to participation and were reminded of their ability to end their session at any time. All procedures were approved by George Mason University’s Institutional Review Board.

Experimental Design

The experiment used a 2 (stimulation condition) x 2 (RSI length) within subjects factorial design. Stimulation conditions included anodal stimulation

(experimental) or sham (control). Response-Stimulus Intervals (RSIs) fell within a 200 ms to 1200 ms range but were ultimately binned by type; short (<533 ms) and long (>866 ms). RSIs were presented at a jittered rate and varied across trials and blocks. The task was a visual discrimination task in which participants were to discriminate between two possible stimuli for the duration of the task following a brief period of stimulation.

Experimental Procedures and Stimuli

Participants engaged in a difficult 2-choice visual discrimination task which required them to discriminate between the hues of two concurrently presented concentric circles. Stimuli were presented in 8-bit sRGB red and green values. Participants indicated via button press whether the concentric circles were the “same” color or “different” colors (**figure 1**). Responses were recorded via speeded button press on a keyboard using only the index fingers of each hand (**figure 2**). Participants were instructed to respond with the index finger of one hand for the “same” colored stimuli and the index finger of the other hand for the “different” colored stimuli. Response hand conditions were counterbalanced between participants and occurred an equal number of times. Each stimulus type occurred with equal probability (50% same stimuli and 50% different stimuli) at varying RSIs. Each trial consisted of 200 ms of stimulus presentation, a response window of 2000 ms, and a variable response-stimulus interval (RSI). RSIs were randomly

selected from a flat distribution ranging 200 ms to 1200 ms; range designed to follow previous research of RSI effects on PES and PEA (Jentzsch & Dudschig, 2009; Dudschig & Jentzsch, 2009). Following a response, or the 2000 ms response window, a variable RSI passed before the stimuli were presented once again (**figure 2**). Participants repeated this process for 12 blocks of 84 trials (1,008 trials).

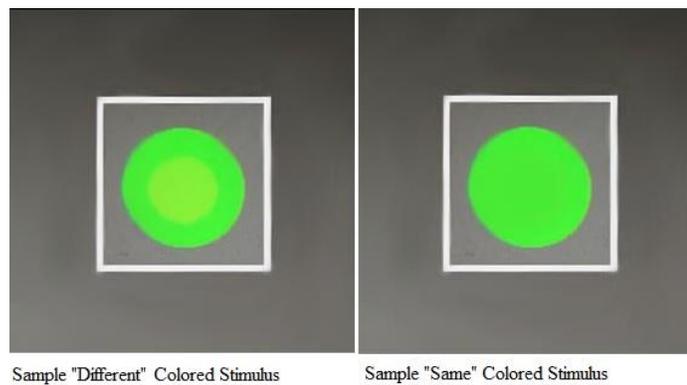


Figure 1: Sample Stimuli. Concentric circles of “different” or “same” hues (8-bit sRGB). Note relative luminance, contrast, and size have been altered for clarity.

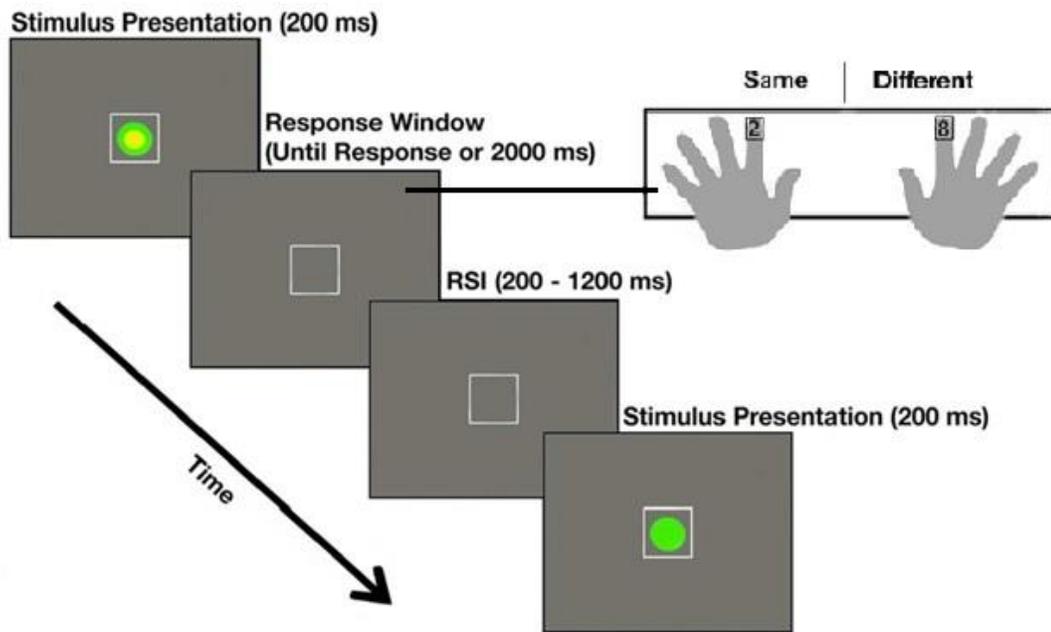


Figure 2: Experimental Paradigm. Note relative luminance, contrast, and size have been altered for clarity.

Participants took part in two procedurally identical experimental sessions. Upon arrival at both sessions participants first provided a signed informed consent after reviewing the exclusionary criteria. They were then screened for normal or corrected to normal visual acuity and color vision. Immediately following the vision screening, participants completed a practice version of the experimental task consisting of 20 trials with accuracy feedback. Participants then continued to the calibration stage of the task, which consisted of 80 trials (without accuracy feedback) in which the QUEST staircase procedure was used to adjust task difficulty,

via incremental changes in red and green sRGB values, until the subject's performance reached ~80% accuracy. Following the calibration, participants completed a second practice consisting of 40 trials of the calibrated experimental task (without feedback) to assess accuracy of calibration. Performance on the second practice was required to fall between 70% to 87.5% accuracy. Participants were not made aware of this requirement. Calibration was repeated until performance (accuracy values) fell within this range. Once performance fell within this range the practice and calibration stages were complete. If performance consistently fell below 70% or above 87.5% following calibration, the participant was not allowed to participate. No participants were lost following this heuristic.

Transcranial Direct Current Stimulation (tDCS) Procedures

Once the calibration and practice were complete the participant would then move on to a set of questionnaires (not discussed here) while the stimulation site, FCZ, was determined according to the international 10/20 system. FCZ was chosen to stimulate the Medial Frontal Cortex (MFC) in accordance with current distribution model findings (Reinhart et al, 2015; Sadleir et al. 2010). Upon completion of the questionnaires, tDCS was administered using a battery-powered, constant-current stimulation device (Mind Alive, Inc.) and a set of two conductive rubber electrodes (active: 19.25 cm², reference: 52 cm²). The electrodes were placed in a saline-soaked sponge pocket which covered the entirety of the electrode. The

anodal electrode was placed at site FCZ and the reference on the cheek (**figure 3**), 3 cm from the cheilion (corner of the lip) following the jaw line diagonally up towards the condylion (tip of jaw bone). Anodal stimulation of 1.5 mA was provided for 20 minutes; stimulation took place in a dark, quiet room prior to completion of the experimental task. Sham stimulation comprised of a fixed 10 second ramp up to 1.5 mA, followed by 10 seconds of stimulation, delivered at both the beginning and the end of the 20-minute session. Stimulation sessions were designed to replicate the methods used by Reinhart et al. (2015). Following the end of stimulation and electrode removal the study began shortly thereafter; no later than 5 minutes.

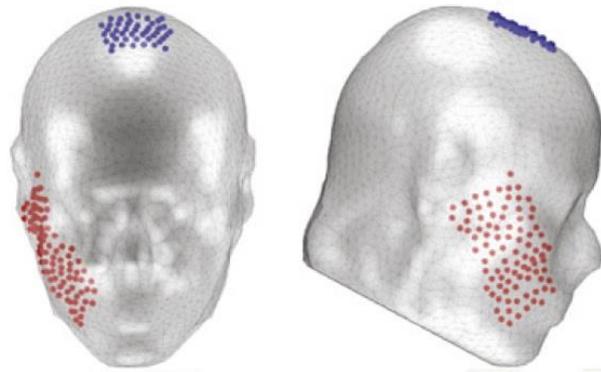


Figure 3: Rendering of tDCS Set-Up (Reinhart et al. 2015). Note: Anodal electrode (Blue) sits at FCZ; Reference (Red) sits along jaw line.

Participants took part in two sessions; one anodal and one sham. The anodal and sham sessions were separated by a minimum 48-hour window to allow for residual stimulation effects to return to baseline. However, completion of the second session was required within seven days of the initial session. Stimulation order was counterbalanced across participants. Upon completion of the task, participants were debriefed and released. No adverse effects were reported.

Behavioral Analyses

All trials in which there was no response, responses were corrected, or responses occurred outside of the 150 ms – 2000 ms response window were removed from analysis. Accuracy and Reaction Time (RT) were calculated for long and short RSIs. RTs were calculated for correct and error trials. Post-Error Accuracy (PEA) and Post-Error Slowing (PES) were also calculated for long and short RSIs. All data were separated and analyzed by stimulation condition and RSI type. Long and short RSIs were specifically chosen to expand on the results of previous studies investigating RSI effects (Jentzsch & Dudschig, 2009; Dudschig & Jentzsch, 2009). Overall accuracy data, RT data (for error and correct trials), PES, and PEA were statistically compared by RSI type (short versus long) and stimulation condition (anodal versus sham). A 2 (RSI Type) x 2 (Stimulation Condition) factorial ANOVA was used for all analyses; if needed these were followed by post-hoc analyses using

paired-samples t-tests. RSIs were designated as short (< 533 ms) or long (> 866 ms) for analysis. Medium RSIs are not discussed here.

To investigate differences in post error-behavior, post-error accuracy (PEA) and post-error slowing (PES) were calculated. PES was determined by calculating the difference between reaction times for correct trials followed by correct trials (correct-correct) and correct trials followed by error trials (correct-incorrect). Similarly, PEA was determined by calculating the difference in accuracy between trials followed by a correct response and trials followed by an incorrect response. PES and PEA were calculated as the percent change in reaction time and accuracy, respectively.

Results

Overall Accuracy: A 2x2 factorial ANOVA showed main effects of stimulation ($F(1,16) = 8.961, p = .009$) and RSI ($F(1,16) = 5.19, p = .037$) on overall accuracy (**figure 4**). Critically, anodal stimulation produced greater overall accuracy compared to the sham condition. Additionally, accuracies for both stimulation conditions were significantly lower at short RSIs than at long RSIs. These results support the notion that increased PMN activation improves overall task performance, although performance is reduced when RSIs are short. This could also be suggestive of an attentional bottleneck following post-error instantiation of top down control.

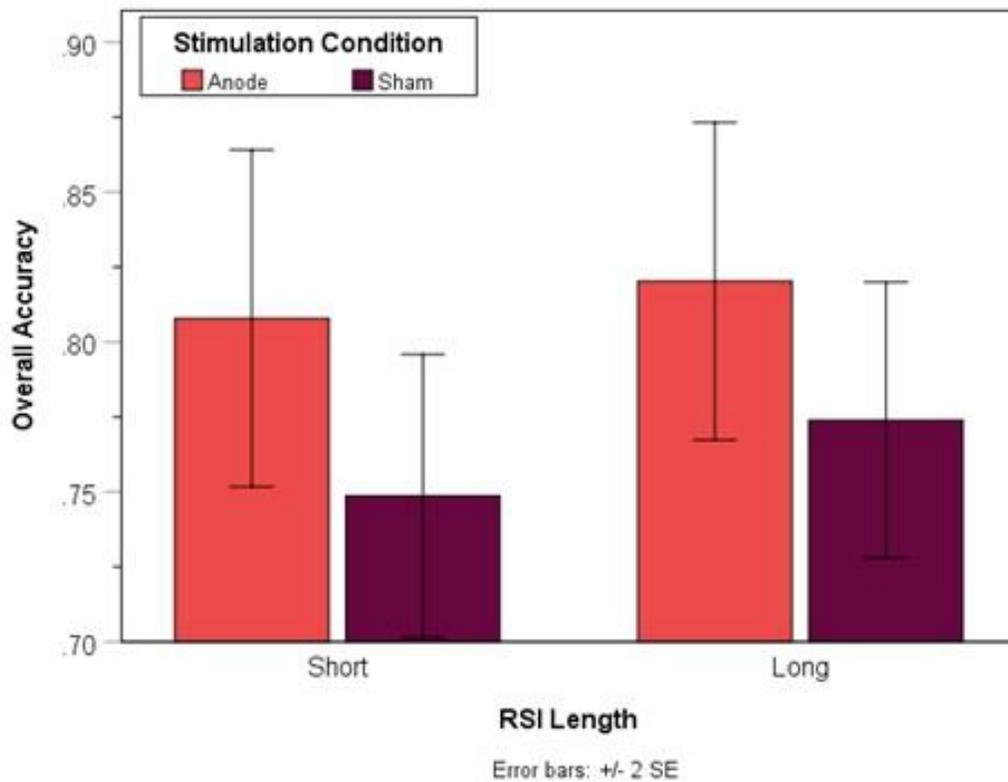


Figure 4. Main Effects of RSI and Stimulation on Overall Accuracy. Note the increase in accuracy for the anodal condition and additionally at long RSIs.

Post-Error Accuracy: The post-error accuracy (PEA) data further support this result PMN activation. A 2x2 factorial ANOVA revealed no significant main effects for RSI length ($F(1,16) = 2.173, p = .160$) or stimulation condition ($F(1,16) = 3.078, p = .098$) on PEA. There was, however, a significant interaction effect between RSI and stimulation on PEA, $F(1,16) = 5.078, p = .039$. Post-hoc analyses using paired-

samples t-tests showed no difference between anodal and sham PEA at short RSIs, $t(16) = .184, p = .856$, whereas at long RSIs, anodal PEA was significantly higher than the sham condition, $t(16) = 3.443, p = .003$. Critically, this shows that following stimulation, participants showed less of a decrement in PEA at long RSIs when compared to the sham group (**Figure 5**). This evidence supports the interpretation that the PMN activation facilitates positive changes in task-oriented behavior only at long RSIs, or when sufficient time is available.

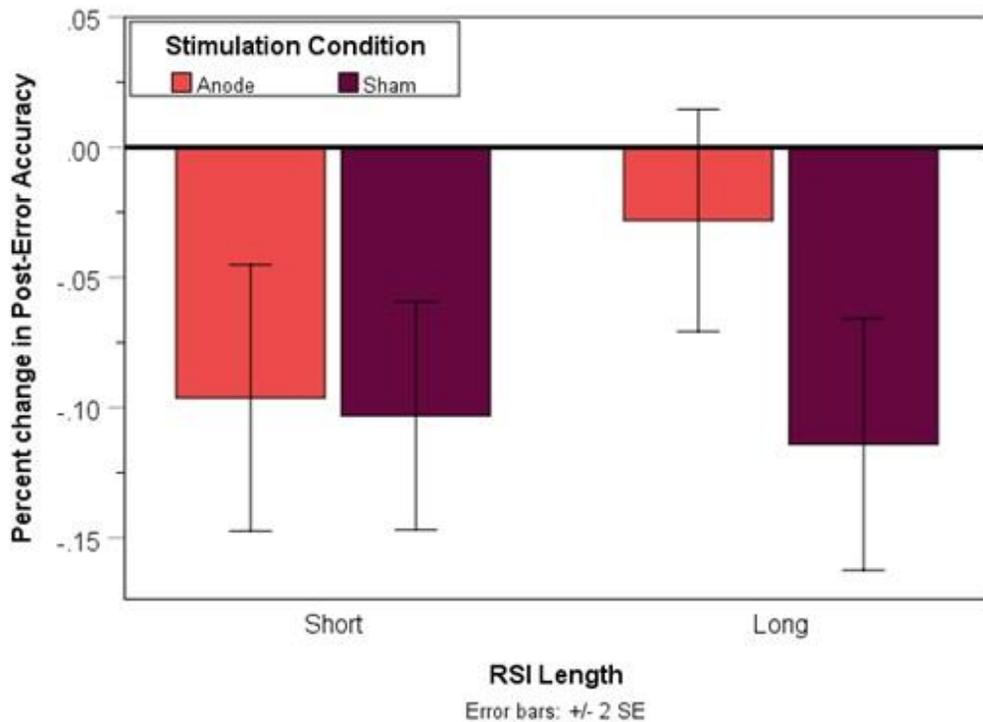


Figure 5. Interaction Effect of Stimulation and RSI Length on PEA. There is a significant difference in PEA between stimulation conditions at long, but not short, RSIs.

Reaction Time: Correct and error RTs were analyzed using a 2 (accuracy type) x 2 (Stimulation type) x 2 (RSI length) ANOVA to determine effects of RSI length (short/long), accuracy type (correct/error), and stimulation (anode/sham) on reaction times. Results showed no main effect of accuracy type ($F(1,16) = .278, p = .605$) or stimulation type ($F(1,16) = .025, p = .876$) on RT. There was, however, a main effect of RSI length ($F(1,16) = 6.503, p = .021$) with shorter RSIs generally producing significantly longer RTs than Long RSIs across both error and correct trials (**figure 6**). No effects on RT were found for the interaction of accuracy type by stimulation type ($F(1,16) = 1.849, p = .193$) or the interaction of stimulation condition by RSI length ($F(1,16) = .567, p = .463$) on RT's. The data do however show a weak trend for an interaction between accuracy type and RSI length ($F(1,16) = 3.182, p = .093$) suggesting a larger difference in RT at short as compared to long RSIs (**figure 7**). Finally, the Accuracy by stimulation by RSI length three-way interaction was not significant ($F(1,16) = .003, p = .961$).

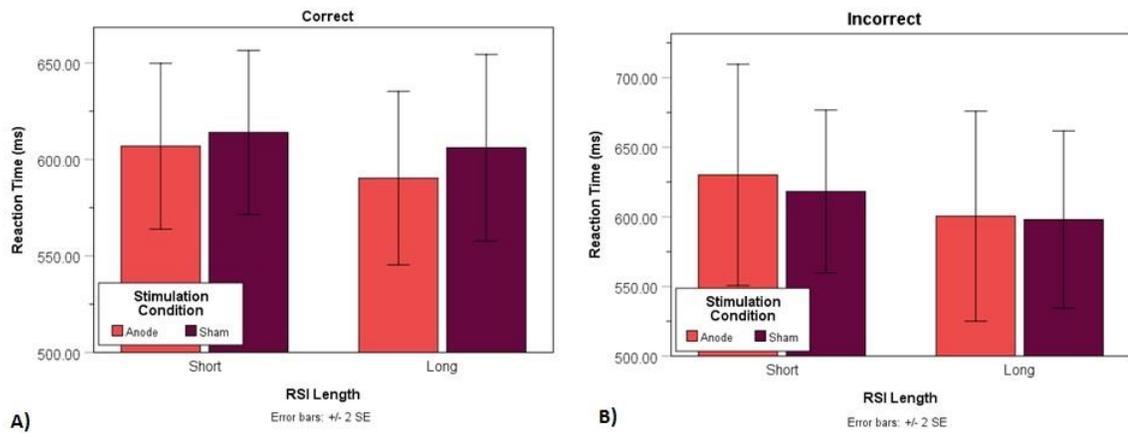


Figure 6. (a: correct trials; b: error trials) Main Effect of RSI Length on Reaction Times. Note the increase in RT following short RSI compared to long RSI for both stimulation groups and response types.

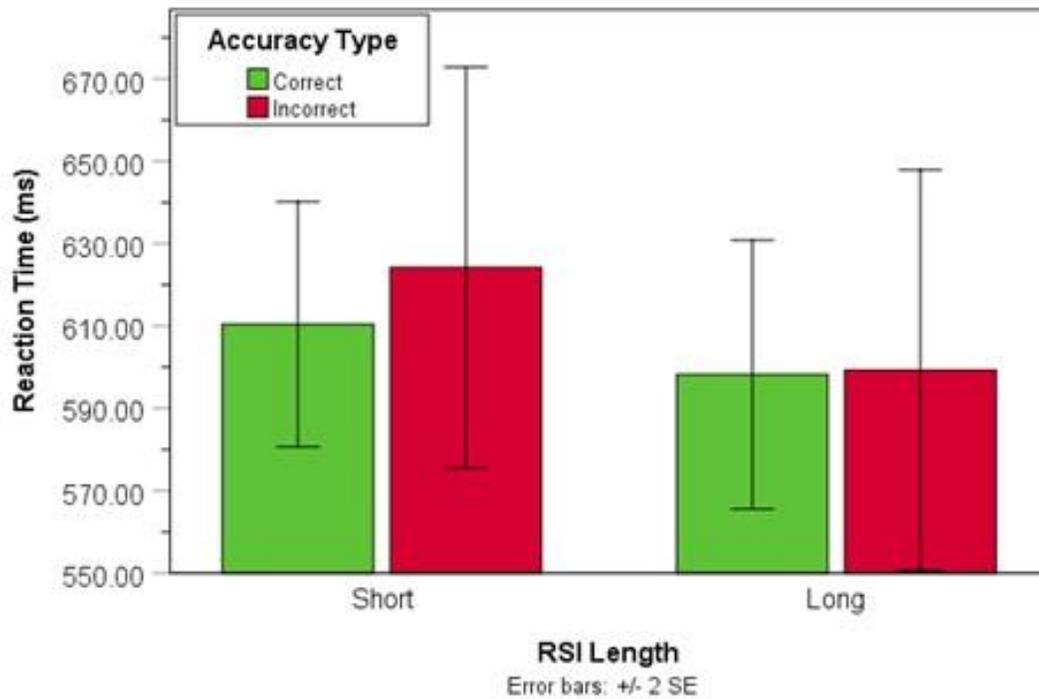


Figure 7. Interaction Between RSI Length and Accuracy was not significant. Note the significantly increased RTs across accuracy type and stimulation type at Short versus Long RSIs.

PES: Analyses of post-error slowing (PES) data revealed no significant main effect of RSI ($F(1,16)=.092, p=.765$) or interaction effect of RSI and stimulation ($F(1,16) = 2.666, p = .122$) on PES. However, participants showed significantly

greater PES, $F(1,16) = 5.088$, $p = .038$, under anodal stimulation (**Figure 8**). These data support the notion that increased activation of the PMN produces greater PES.

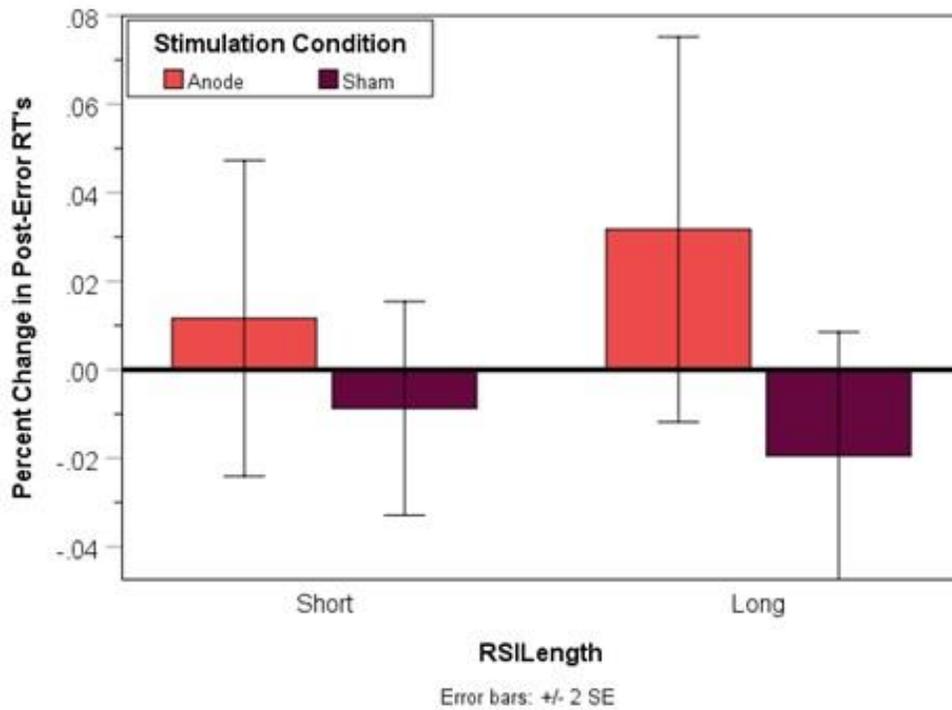


Figure 8: Main Effect of Stimulation on PES. Participants show greater PES at Long RSI than Short RSI in the Anodal condition.

To determine whether experimental parameters other than those evaluated in the primary analysis influenced performance we analyzed post-calibration accuracy, color difference, and luminance difference values between sessions. Critically, post-calibration practice accuracy values did not significantly differ between sessions, $t(16) = .643$, $p = .529$). The sRGB red values also did not differ significantly, $t(16) = 1.461$, $p = .163$). However, the sRGB green values did significantly increase from session one to session two $t(16) = 2.910$, $p = .001$. Reasons for this are unknown, however, the influence of these effects were controlled by calibration methods to provide consistent performance values that fell within the desired accuracy range prior to each experimental session.

Discussion

The goals of this study were to assess the degree to which PMN activation, as modulated by tDCS, influenced performance on a demanding visual discrimination task, and, more specifically, to determine how PMN activation impacts post-error behavioral adaptations as a function of RSI. Based on previous findings, we hypothesized that (a) error processing would interfere with task performance at short but not long RSIs and (b) increased PMN activation (induced by tDCS) would exacerbate this decrement. Through excitatory DC stimulation, the present study provides novel findings on the influence of PMN activation on post-error behavior adaptations. Our results suggest that PMN activation results in overall improved task performance (overall accuracy), as seen in the global increase in stimulus discrimination accuracy. Additionally, our results show that PES and PEA are influenced by excitation of the MFC. Most notably, following anodal stimulation, PEA increased at long RSIs and PES increased at both long and short RSIs suggesting that MFC activation does in fact influence error processing. These results also suggest that increased PES, although observed at both short and long RSIs, is only linked to improved performance at long RSIs. The unexpected finding of comparably low PEA at both short and long RSIs in the sham condition contrasts with previous work, and does not lend support to our hypothesis that PMN stimulation would selectively

reduce PEA at short RSIs (Jentzsch & Dudschig, 2009; Houtman & Notebaert, 2013a; Houtman & Notebaert, 2013b; Van der Borgh, Braem, Stevens, & Notebaert, 2016). Nonetheless, these data provide support for the notion that DC stimulation improves post-error performance, with the benefit selectively occurring at long RSIs.

The observed changes in post-error behavior under anodal stimulation indicate that MFC activation significantly altered performance monitoring processes. The overall accuracy increase observed for the anodal group supports the idea that PMN activation improves performance via improved error-processing and behavioral modification (Botvinick, et al., 2001). Our data are consistent with previous stimulation studies by Reinhart et al. 2015, Bellaiche et al. 2013, Harty et al. 2014 and more specifically suggest increased error-processing following tDCS.

Our data fall in line with Reinhart's findings suggesting that a causal relationship exists between MFC stimulation, medial-frontal theta oscillations, and post-error behavioral adjustments. Following stimulation of the MFC, normal theta oscillation phase coherence between MFC and DLPFC and elevated PES were observed in schizophrenic patients, implicating the stimulation as the source of improved cortical dynamics and behavioral adjustments (Reinhart et al., 2014). Critically, the changes observed in Reinhart's study following stimulation, increased ERN and greater learning observed in schizophrenic patients, provide additional

support similar to that presented by Bellaïche et al. (2013) and Harty et al. (2014). Their studies found tDCS successful in reducing Pe amplitude via cathodal stimulation (Bellaïche et al., 2013) and increasing error awareness in older adults via anodal stimulation (Harty et al., 2014), respectively. These data provide evidence that administration of tDCS to the MFC and DLPFC is an effective method by which to modulate activation of the PMN and ultimately improve error-processing when sufficient time between error commission and the subsequent task is provided.

As mentioned above, the present study revealed an unexpected reduction in PEA at both long and short RSIs in the sham condition. We speculate that this phenomenon may reflect an effect of complacency. Participants may have assumed that stimulation was always present and would bolster performance across both sessions and consequently employed minimal or reduced effort on the task during both sessions. This may have resulted in reduced performance in the sham condition given that stimulation was not actually present to bolster performance. However, when stimulation was present, post-error performance was notably improved at long RSIs as a result of stimulation. Additionally, decreased effort may have been counteracted by potential effects of excitatory stimulation.

A model by Ullsperger and Danielmeier (2016) posits that post-error processes can be parsed into distinctive stages. Critically, the early stages present a

trade-off between task-driven attention and error-processing, with error processing taking priority shortly following error commission. Consequently, during this early time-period following the error, task-oriented attention is diminished. The model predicts that performance should improve as more time passes, owing to a progressive reduction in error processing and concomitant increase in task-related attention. The finding that post-error stimulus processing is diminished at short, but not long, RSIs is consistent with this notion (Buzzell et al., 2017). Our data show that increased activation of the PMN resulted in improved post-error behavioral performance at long, but not short, RSIs consistent with this model.

Broadly, our findings support the notion that improved task-oriented behavioral adjustments follow PMN activation. They provide evidence for the suggested use of tDCS in modulating PMN activation to produce successful post-error behavioral adjustments. Further investigation of tDCS as a stimulation method should combine use of EEG or other temporally specific physiological methods to further explore the neural underpinnings of error-monitoring. Future assessments of stimulation effects on the ERN or Pe using similar task parameters could provide neural data to further clarify the nature of the post-error behavioral adaptations observed in this study. Given that the human performance monitoring network notably requires several cognitive processes to work in conjunction with one another to facilitate successful task performance (Ullsperger & Danielmeier, 2016), investigating the influence of task type and difficulty may also prove worthwhile

avenues for future research. Due to the selective effects of task type and task difficulty on error processing, response conflict, attentional control, and perceptual processing, behaviors may show similar selective effects in either error or stimulus processing. The current findings serve as a starting point to further this investigation.

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Biography

Natalie Paquette grew up in Ocoee, FL and attended the University of Central Florida where she earned her Bachelor of Science in Psychology and minor in Cognitive Science in 2013. She then began the graduate program in Cognitive and Behavioral Neuroscience – Psychology at George Mason University in 2015 and went on to earn her Master of Arts in Psychology in 2017. While a graduate student at George Mason University, her research focused on the neural basis of performance monitoring and cognitive control. She will be starting a doctoral program in Human Factors and Cognitive Psychology with a concentration in Cognitive Neuroscience at the University of Central Florida as a McKnight Doctoral Fellow in the Fall of 2017.