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Differential Patterns of Theta Activation Underlying Various Cognitive Control Strategies

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Differential Patterns of Theta Activation Underlying Various Cognitive Control Strategies

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Abstract

In this study, EEG was recorded from 157 participants at the University of Arkansas as they performed three computer tasks that tested inhibitory control (Go/Nogo Task), proactive and reactive control (AX-Continuous Performance Task), and resolving response conflict (Global/Local Task- modified Flanker Task). Time-frequency analysis (ERSP) was the primary focus of this study, in order to take advantage of the temporal and frequential characteristics of EEG recordings. The ERSPs and following statistical analysis showed significantly higher midfrontal theta band (4-8 Hz) power values for target trials (those that required more cognitive control) than control trials, which indicated that the procedure was implemented correctly. Furthermore, statistical analysis revealed that reactive control and inhibitory control had significantly higher theta power values than both proactive control and response conflict, and that proactive control had significantly higher theta power values than response conflict. Taken together, these results suggest a common underlying physiological mechanism for initiating and executing cognitive control, namely frontal midline theta band oscillations, but how these oscillations are integrated into cognitive processing still remains unclear. The results of this study suggest that theta power might be an important factor in allowing frontal midline brain regions to differentiate cognitive control mechanisms, but further work will need to be completed to investigate the role of theta power and theta phase in establishing and coordinating cognitive control.

1. Introduction

Cognitive control processes are activated in the brain whenever habitual neuronal responses are inadequate to support goal-oriented behavior (Cavanagh & Shackman, 2014).These processes span a wide array of behavioral and physiological mechanisms that are employed to prioritize information and make decisions in the face of uncertainty (Mackie et al., 2013). Often times cognitive control and executive control are used synonymously. While the topic of executive function has been investigated throughout many scientific fields, the underlying mechanisms of how the brain coordinates several regions of the brain to exhibit cognitive control are not completely understood. Furthermore, to our knowledge a differential analysis of various cognitive control strategies has yet to be completed. That is to say, how these various cognitive control mechanisms differ from each other, both in terms of amount of cognitive demand and the physiological processes that underlie them, is not entirely known.

In attempt to understand these control processes further, researchers investigate their underlying mechanisms often through neuroimaging or electrophysiology resources, such as functional magnetic resonance imaging (fMRI; Garavan et al., 1999; MacDonald & Carter, 2003) or electroencephalography (EEG; Folstein & Van Petten, 2008; Cavanagh & Shackman, 2014). These technologies provide more insight into the biological basis of cognitive control, but in distinct manners. Functional MRI is a type of blood oxygen level dependent (BOLD) imaging that is used for its spatial resolution to localize the anatomical basis of brain functions, while EEG is used for its temporal resolution to quantify and characterize the dendritic potentials of the brain's upper cortex. This study focused on the EEG modality, but certainly depended on previous research that was conducted with fMRI (Garavan et al., 1999; MacDonald & Carter, 2003, Botvinick et al., 1999).

EEG has been used in cognitive control research to study the electrophysiological link between theorized control mechanisms and the brain's structure. One form of EEG data analysis that that is frequently used by cognitive control researchers uses event-related potentials (ERPs) in the time domain, which are the average phase-locked voltage distributions recorded from EEG across experimental trials. For example, the "N2", which is the negative voltage deflection that peaks between 200-300 ms post-stimulus in frontal and mediofrontal scalp regions, increases in magnitude during high conflict trials when compared to low-conflict trials in Go/No-Go tasks, Eriksen Flanker tasks, AX-Continuous Performance Task (CPT), and Stop/Signal paradigms, all situations believed to require more cognitive control (Folstein & Van Petten, 2008; Lamm et al., 2013). However, a limiting factor of using ERPs to explore cortical activation is that it collapses the time-domain signal across the frequency bands. This study aimed to make use of both the temporal and frequency components of EEG by analyzing data in the combined time-frequency domain. By convolving the time-domain EEG signal with Morlet wavelets, event-related spectral perturbations (ERSPs) are produced. These scalograms create a profile of the changes in spectral power of the EEG recording, relative to a baseline, at a certain time and frequency point within a given experimental epoch (Figure 4). Time frequency analysis can provide information about the frequencies in EEG recordings that are predominantly active during cognitive control functions as well as the timing and phase angle at which these frequency activations occur with respect to stimulus or response onset. This information could be powerful in the realm of executive function because the oscillatory activity of certain neuron populations might be a fundamental mechanism by which the brain coordinates brain regions for rapid decision-making and information processing during instances demanding cognitive control conflict (Cohen & Donner, 2013; Cavanagh & Frank, 2014; Cavanagh & Shackman, 2014; Cooper et al., 2015).

Time-frequency analysis provides more information about the manner in which the brain's structures are communicating to execute goal-oriented behaviors. Many studies have shown that circumstances requiring cognitive control modulate EEG activity in the theta (4-8 Hz) frequency range (Cohen et al., 2008; Cavanagh et al., 2012; Cohen & Cavanagh, 2011), and inflict changes in beta (15-30 Hz; Cohen et al., 2008) and alpha (8-14 Hz; Compton et al., 2011) power, throughout experimental trials. The source of these higher levels of theta power during cognitive control is believed to be anterior cingulate cortex because of the role it plays in action-monitoring and action-selection to optimize goal-driven performance (Cavanagh et al., 2012; Botvinick et al., 1999; Liddle et al., 2001).This study aimed to confirm the theory behind cognitive control and midfrontal theta activation and provide a comparative analysis of four distinct cognitive control strategies: inhibitory control, proactive control, reactive control, and resolving response conflict.

Inhibitory control was operationalized as suppressing a prepotent response in a Go/Nogo task (modified from Garavan et al., 1999), which was administered through E-Prime software on a computer monitor. Event-related fMRI studies have shown that the anatomical basis of response inhibition includes an array of brain regions, such as the supplementary motor area, dorsal and ventral frontal regions, parietal lobe, and the anterior cingulate cortex (Garavan et al., 1999). The role that each of these regions plays in response inhibition is not fully understood, but it has been hypothesized that the anterior cingulate cortex plays a role in successful response inhibition, through making and monitoring decisions (Liddle et al., 2001; Carter et al., 1998; Cavanagh et al., 2012). Along with this idea, there have been several reviews that propose an underlying mechanism of this action monitoring to be frontal midline theta oscillations above the medial

prefrontal cortex (Cavanagh & Frank, 2014; Cavanagh & Shackman, 2014; Cavanagh et al., 2012).

Proactive and reactive control were represented through the AX-Continuous Performance Task (adapted from MacDonald & Carter, 2003). Proactive control processes are the cognitive mechanisms that prepare the brain to be particularly sensitive to incoming goal-relevant stimuli, and reactive control processes are the more reactionary mechanisms that are used to resolve conflict and overcome interference (Cooper et al., 2015). The "BX" and "AY" trials were used to operationalize these processes, respectively, as participants learned that the "B" cue prepared them for the next response and the "Y" probe was the reactionary stimulus that instigated the need for cognitive control to make the correct decision (Figure 2; Procedure section). Both of these cognitive control mechanisms are believed to be controlled by theta frontoparietal oscillatory networks (Cooper et al. 2015), which again supports the notion that frontal midline theta oscillations are a key mechanism to instigating and enacting cognitive control.

Response conflict was examined through the Global/Local task, a modified Eriksen Flanker task that used Navon (Navon, 1977) Letters instead of arrows. Conflict trials were operationalized through mis-matched letter configurations (big "H" made of small "S" or big "S" made of small "H"; Figure 3). The biological structures underlying conflict monitoring and conflict resolution are largely agreed to be located in the medial frontal cortex (Cohen et al., 2008; Nigbur et al., 2011), especially the anterior cingulate cortex (Botvinick et al., 1999; Cavanagh et al., 2012). There have been numerous studies that show conflict control functions are enabled by theta oscillatory networks (Nigbur et al., 2011; Cohen & Cavanagh, 2011; Cohen et al., 2008).

Therefore, we hypothesized that the experimental target trials, which demanded relatively more cognitive control than control trials, would have more frontal midline EEG activity in the theta (4-8 Hz) frequency band than control trials. This is in alignment with the previously mentioned research and theory on the anterior midcingulate cortex, medial frontal regions, and the role of theta oscillations as a lingua franca for cognitive control (Cavanagh et al., 2012). While there has been a considerable amount of research delving into the individual bases of these separate cognitive control mechanisms, there are considerably fewer studies that were designed to compare and contrast these cognitive control mechanisms on their time-frequency profiles within the theta range. Because of the exploratory nature of this study, we are not making specific hypotheses about how various cognitive control strategies will relate to each other.

2. Methods

2.1 Participants

The EEG data for this time-frequency study was collected from 157 undergraduate students in the University of Arkansas general psychology pool (Gender: 73 M, 80 F, 1 Androgyne, 3 N/A; Age: $\bar{x}$ = 19.19 years, s = 1.30 years). All participants included in this study were English-speaking and self-reported that they had no current psychiatric diagnoses, no psychoactive drug use, and no uncorrected visual impairments. Additionally, all subjects used in this study completed at least 12 correct trials without considerable artifact per trial type, otherwise errors occurred while generating the epochs for time-frequency analysis. All students were granted course credit for their participation in this study. This study was approved by the University of Arkansas' Institutional Review Board (IRB#: 1708026820).

2.1 Procedure

In order to test for the four cognitive control strategies (inhibitory control, proactive control, reactive control, and resolving response conflict), three computer-based tasks were completed by the participants. The Go/Nogo task was designed to test response inhibition. The "AX" continuous performance task (AX-CPT) was designed to test proactive and reactive control. The Global/Local task was designed to test one's ability to resolve response conflict. All 3 tasks were presented on a 17-inch computer monitor using E-Prime software (Psychology Software Tools, Inc., Pittsburgh, Pennsylvania; Schneider et al., 2002). Stimuli were displayed on a black screen, and each task was shuffled throughout the entire experimental trial (approximately 1.5 hours).

The Go/Nogo task was adapted from the task described in Garavan et al. (1999). The task began with a fixation cue shown in the middle of the screen for 100 ms to focus the participant's eyes on where the next stimulus would arrive. Stimuli consisted of a single, white letter displayed for 200 ms. Participants were instructed to respond to any letter besides the letter "X" by pressing the button labeled #1. After the stimulus cue, another fixation cue appeared for 600 ms, during which the participants responded to the stimulus cue. If the letter presented was "X", then the participant was instructed to refrain from pressing the button (apply inhibitory control). After this fixation cue, another fixation cue was displayed for an inter-trial interval that varied from 0-500 ms. Go trials, in which the participant was instructed to respond, constituted 75% of the trials in this task, in order to establish a prepotent response. Nogo trials, in which the participant was instructed not to respond, represented the other 25%. A depiction of this task is shown below in Figure 1.

Figure 1. Depiction of the Go/Nogo Task.

The AX-CPT task was adapted from MacDonald & Carter (2003). Instead of being shown one letter, as in the Go/Nogo task, participants were shown pairs of letters in this task. The four types of pairs were “AX”, “AY”, “BX”, and “BY”, with the “AX” pair being the special pair with distinct instructions from the rest. The task began with a 500 ms fixation stimulus in the middle of the screen, as shown in Figure 2. Then the cue was presented (first letter of the pair) for 200 ms and colored light blue to let participants know when a new pair began. After the first letter (cue) was shown, the participant was allowed an additional 1300 ms to respond, after which a fixation stimulus was shown for 2000 ms. This was the case for every trial, regardless of the condition. After this fixation stimulus, the second letter within the pair was shown in white for 200 ms. This stimulus was termed the probe. After the probe was shown, the participant had an additional 1300 ms to respond, after which another fixation stimulus was displayed for an

inter-trial interval that varied from 1000-2000 ms. There were 4 trial types: “AX”, “AY”, “BX”, “BY”. If the second letter was an “X” preceded by an “A” (making it an “AX” pair), then participants responded to the letter “X” with the button labeled #5. Hence, for “AX” trials, participants responded with button #1 after the “A” letter and with button #5 after the “X” letter, as shown in Figure 2. These “AX” trials comprised 70% of the trials for each round of the AX-CPT, while the other three trial types comprised the remaining 30% equally (10% each condition). If the second letter was a “Y” preceded by an “A” (making it an “AY” pair), then participants responded to the “Y” letter with the button labeled #1. So, for “AY” pairs, the participant was expected to respond with button #1 after “A” and button #1 after “Y”. During these “AY” trials, participants were first primed with the “A” cue, and since the “AX” condition was prepotent, they expected that the second letter would be “X”. However, since the second letter was “Y” in “AY” trials, participants had to react to the “Y” probe and change their second response to button #1. This requires reactive control. During the “BX” trials, participants were shown a “B” first, to which they were supposed to respond with button #1, and then they were shown “X”, which designated a response with button #1. In these “BX” trials, participants had to remember that the letter “B” preceded the letter “X”, otherwise they might be tempted to respond with button #5 after being shown the letter “X”. Hence, keeping in mind that they saw a “B” letter for the cue required proactive control. In the last trial type, the letter pair “BY” was shown to participants, to which they were supposed to respond with button #1 after the “B” and button #1 after the “Y”(control trial type).

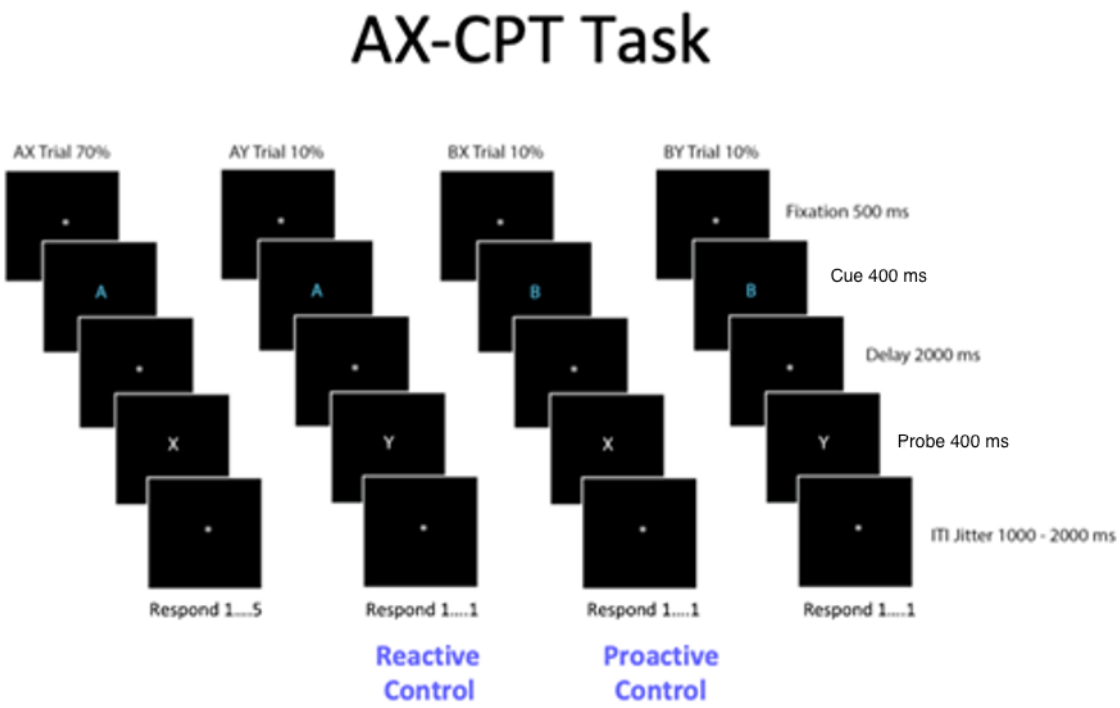

Figure 2. Depiction of the AX-Continuous Performance Task.

The Response Conflict task was a Flanker task that presented Navon Letters (Navon, 1977) as the conflict inducing stimulus rather than arrows, thereby increasing the difficulty of the task. In this task, participants were shown a large letter comprised of smaller letters. Sometimes the bigger and smaller letters matched ("congruent") and sometimes the bigger and smaller letters did not match ("incongruent"). For example, in Figure 3 below, the global incongruent trial shows a big "S" made of small "H". The other conditions were a big "H" made of small "S" ("incongruent"), big "H" made of small "H" ("congruent"), and big "S" made of small "S" ("congruent"). During each trial, participants were first shown the local/global indicator for 2000 ms, which was the word "Big" or "Small". This let participants know if they should respond to the larger, overall letter shape or to the smaller letter, respectively. Then participants were shown the actual letter stimulus for 200 ms. After this interval, participants were shown a fixation cue

for 1100 ms, during which they responded to the previous stimulus. Regardless of the "Big" or "Small" conditions, if the participant believed the correct response was the letter "H", they responded by pushing button #1. If the correct response was believed to be "S", participants responded by pushing button #5. Next, another fixation stimulus was shown for an inter-trial interval that varied from 0-500 ms. For this task, the "incongruent" trials were of particular interest, since the participant was required to resolve the conflicting "S" and "H" stimuli in order to respond correctly. Hence, this task tested the participants' ability to resolve response conflict.

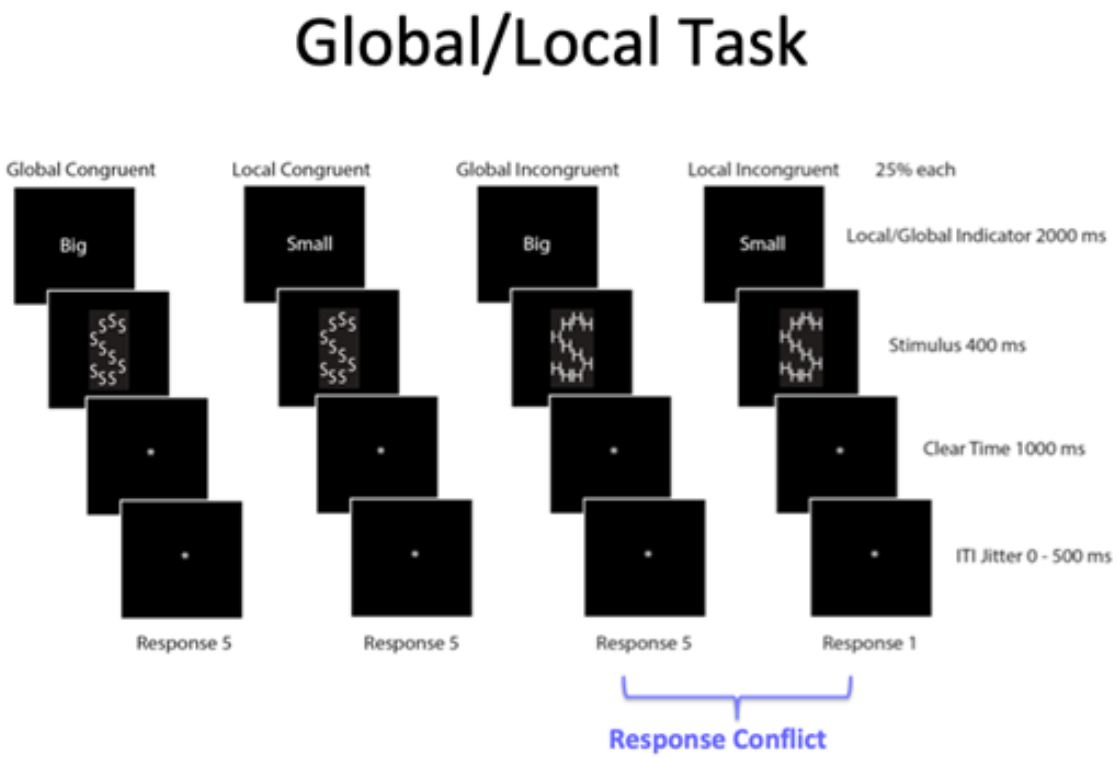

Figure 3. Depiction of the Global/Local Task.

2.2 Data Collection and Analysis

All EEG recordings were completed using a 128-channel HydroCel Geodesic Sensor Net with a potassium chloride solution to facilitate the electrical readings. The recordings were sampled at 1000 Hz using EGI Net Station Acquisition software (Electrical Geodesics, Inc., Eugene,

Oregon). Data acquisition began after the impedances on all 128 channels were below 50 kΩ. All channels were referenced to the Cz electrode during data acquisition but re-referenced to an average of all electrodes offline for data analysis.

After EEG data acquisition was complete, data processing was implemented using the EEGLAB (Delorme & Makeig, 2004; https://sccn.ucsd.edu/eeglab/index.php), ADJUST, SASICA, and Signal Processing toolboxes in MATLAB v. R2019b (https://www.mathworks.com/products/matlab.html).

The EEG data were pre-processed as follows. First, each raw data set was band-passed between 0.1 Hz and 35 Hz using a Hamming windowed-sinc FIR filter. Then the data was downsampled to 125 Hz. From here, EEG channels within a participant's data were rejected if the joint probability of that channel's data and all channel data exceeded 4 standard deviations. This helped to detect and remove any channels with considerable noise. Following this, the data was epoched to form stimulus-locked segments ranging from 2500 ms pre-stimulus to 3000 ms post-stimulus. This large range was chosen to account for edge artifact created during wavelet convolution. The time-locked stimuli for the separate cognitive control strategies were: the "B" cue for proactive control (BX trials), the "Y" probe or reactive control (AY trials), the "X" cue for inhibitory control (Nogo stimulus), and the incongruent cues for resolving response conflict (e.g. big H made of small S). All non-time-locked events were removed. Next, independent component analysis (ICA) was performed on the data using the *runica* EEGLAB function (Makeig et al., 1997) and the ADJUST MATLAB plugin ((Mognon et al., 2011) to identify and delete motion artifact related to eye blinks, eye movements, and other stereotyped sources. After ICA, remaining artifacts were removed by thresholding the epochs ±140 μV, which detects and deletes values outside of ±140 μV. Then, all channels that contained deleted data points were

interpolated using a superfast spherical interpolation and average referenced. This concluded the pre-processing pipeline. If any subject completed less than 12 correct, clean trials for any cognitive control condition, they were removed from further analysis. The 157-subject sample only includes the subjects that met these criteria.

Following pre-processing, the EEG data was ready for time-frequency analysis. The processed EEG data was acquired in the time domain. Using EEGLAB's *newtimef* function, individual trials from -500 ms to 1000 ms (stimulus-locked) were convolved with a series of complex Morlet wavelets, focusing on 25 logarithmically-spaced frequencies ranging from 1-25 Hz, to create a time-frequency depiction of the EEG signal. A complex Morlet wavelet is a complex, Gaussian-tapered, sine wave represented by the equation $e^{i2\pi tf}e^{t^2/(2*\sigma^2)}$, where t represents time, f represents frequency, and σ represents the width of each frequency band according to s/(2πf). In this sub-formula, s represents the number of cycles. An adaptive number of cycles was used in this analysis, in which 3 cycles were used at 1 Hz and the number of cycles was increased equally until 10 cycles were used at 25 Hz (Cohen et al., 2008). This improves the temporal resolution at low frequencies and the frequency resolution at high frequencies. The baseline measurement, used in the calculation of spectral power in decibels (dB), was taken from -500 to -200 ms pre-stimulus. Decibel power was calculated via the formula 10 * log10[power(t)/power(baseline)], which used the power that was calculated previously by the formula real[z(t)]^2 + imag[z(t)]^2 (Cavanagh et al., 2012). Z(t) represented the magnitude of the analytical, convolved signal. The plots in Figure 4 only show power values that were significantly different from the baseline ($p < 0.01$). This was calculated using custom-written MATLAB code that performed permutation testing with 500 permutations per cognitive control mechanism to test the null hypothesis that the event-related data and baseline data were

interchangeable (adapted from Cavanagh et al., 2012). Because this study focused on frontal midline theta oscillations believed to be generated by the mid-cingulate cortex (Cavanagh & Shackman, 2014), the electrodes that were chosen for time-frequency analysis were sensors 6 (FCz), 129 (Cz), and 11 (Fz). These electrodes lie on the frontal midline of the scalp. The average of the activation at sensors 6, 129, and 11 at each data point was used for generating the scalograms (Figure 4). For statistical analysis, frontal midline theta activation for each condition was computed as the average activation (or decibel spectral power) per subject within 4-8 Hz frequency and 200-450 ms time post-stimulus (outlined in Figure 4). This time period was chosen because it roughly underlies the time period of the N2, an ERP consistently associated with cognitive control. (Cavanagh & Frank, 2014; Cooper et al., 2015; Nigbur et al., 2011; Cavanagh et al., 2012).

3. Results

The ERSP plots that were produced from the time-frequency analysis across 157 participants are shown below in Figure 4. The plots are organized so that the left column represents control trials and the right column represents target trials, or the ones that demand relatively more cognitive control. The peak significant power values and the time and frequency at which they occurred for target trials of each cognitive control mechanism were noted (proactive: max. power = 7.334 dB at 372 ms and 3.82 Hz; reactive: max. power = 8.971 dB at 340 ms and 5.00 Hz; inhibitory: max. power = 8.295 dB at 316 ms and 5.00 Hz; response conflict: max. power = 6.181 dB at 380 ms and 2.92 Hz). The plots were produced using custom-written MATLAB code that implemented several EEGLAB functions (Delorme & Makeig, 2004).

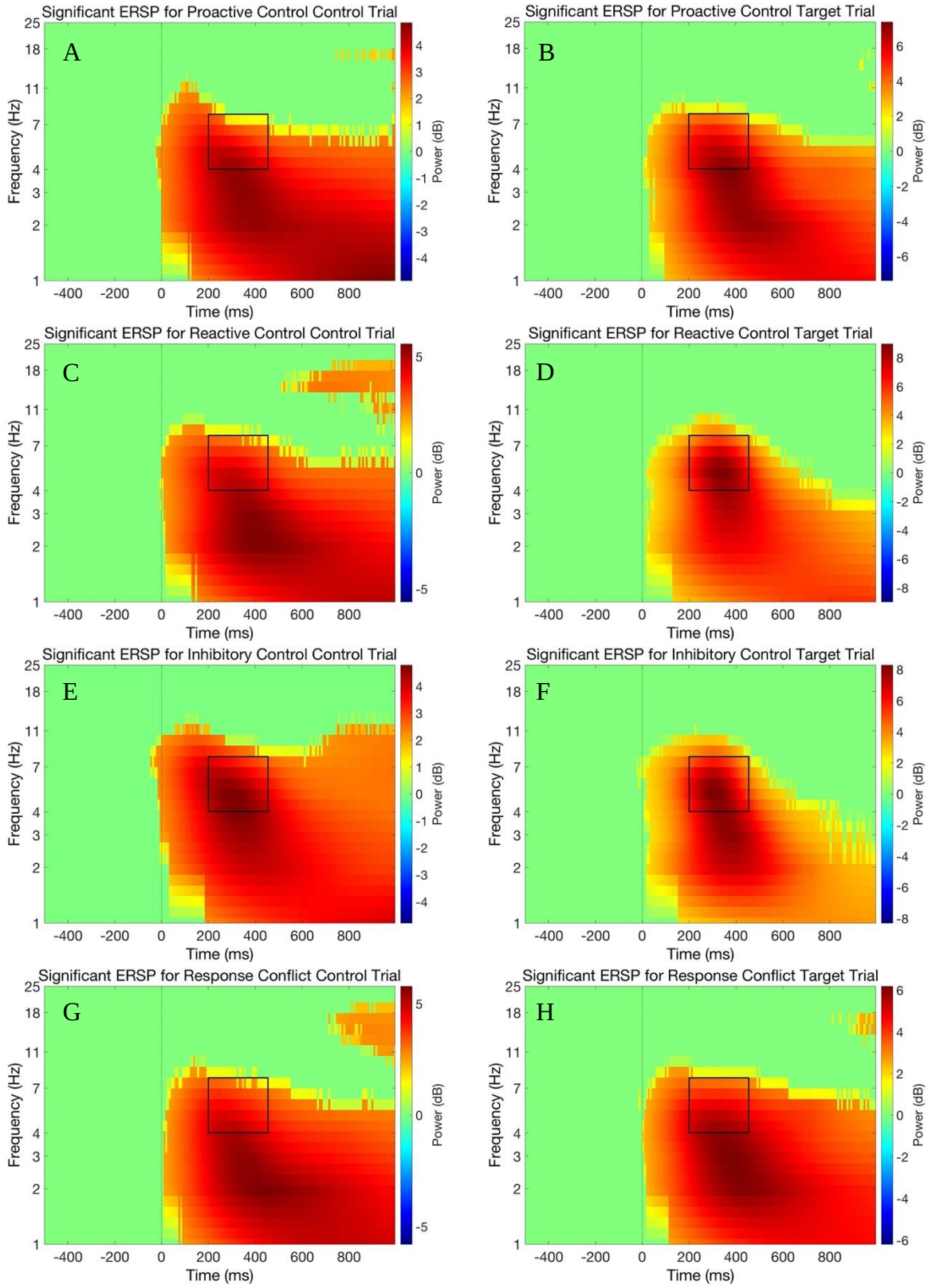

Figure 4. Event-related spectral perturbations (ERSPs) for each trial and task epoched from -500 ms to 1000 ms in relation to the time-locking stimulus for each trial. The baseline was taken from -500 to -200 ms to compute the event-related power in decibels. Only the power values that were significant ($p < 0.01$) from the baseline were included in these plots, which was calculated using permutation testing.

Data were exported and statistically analyzed in SPSS (https://www.ibm.com/products/spss-statistics). To control for family-wise error, a Bonferroni correction was applied to all post-hoc analyses. Because some analyses violated the Sphericity assumption, all omnibus ANOVA results reported here had the Greenhouse-Geisser correction applied.

We conducted a 4(Cognitive-Control Strategy: reactive control, proactive control, inhibitory control, resolving response conflict)-by-2(Trial Type: target, control) repeated-measures ANOVA on theta power. Results revealed a main effect of Cognitive-Control Strategy, $F(2,156) = 36.94$, $p < .001$, $\eta^2 = .19$, and a main effect of Trial Type, $F(1,156) = 223.89$, $p < .001$, $\eta^2 = .59$, which were both subsumed by a Cognitive-Control Strategy-by-Trial Type interaction, $F(2,376) = 30.05$, $p < .001$, $\eta^2 = .16$. Bonferroni-corrected contrasts revealed that all target trial types were significantly higher in theta power than control trial types ($p < .001$).

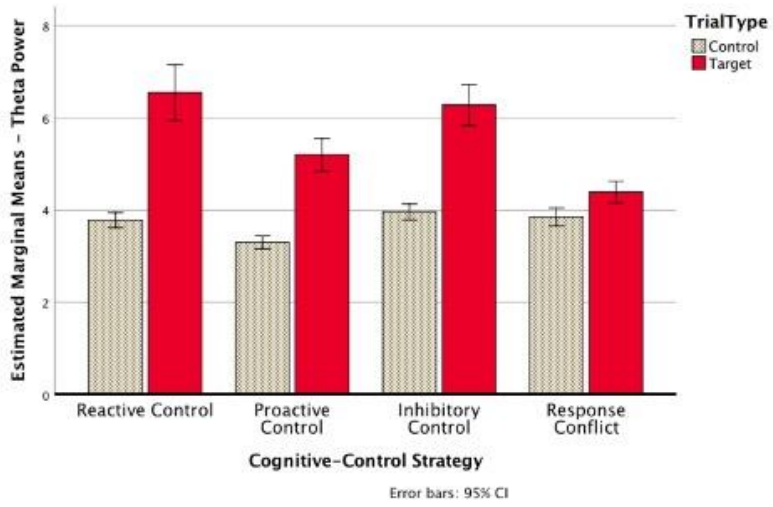

Figure 5. Comparative bar graph of estimated marginal means results for theta power across the four cognitive control strategies.

For control trials, contrasts revealed that go trials showed more theta power than both reactive control ($p = .03$) and proactive control ($p < .001$). Additionally, for control trials, response

conflict ($p < .001$) and reactive control ($p < .001$) showed greater power than proactive control. For target trials, reactive control and inhibitory control showed more power than proactive control ($p < .001$, $p < .001$) and response conflict ($p < .001$, $p < .001$), and proactive control showed more power than response conflict ($p < .001$). A graphical representation of these comparisons is shown in Figure 5.

4. Discussion

When observing the plots shown in Figure 4, it is clear that there are high levels of theta band power (shown by dark red regions) consistent across all target trials. This was an important result, as it supports the hypothesis that theta oscillations are a common substrate, or a lingua franca, for cognitive control (Cavanagh et al., 2012). Furthermore, the statistical analysis concluded that target trials had significantly higher theta band power values than control trials (mean diff: 1.882; $p <0.001$), which meant that the procedure had been designed and implemented correctly and that the surrounding theory behind frontal midline theta and its relation to cognitive control is sound.

While these results were essential to the viability of this study, this study was particularly interested in contrasting the separate cognitive control mechanisms on their ERSP power values in the theta range (reactive control & inhibitory control > proactive control > response conflict). These results allowed us to be one of the first research groups to establish differential relations between separate cognitive control mechanisms using a reasonably large sample.

The results from this study suggest that increases in event-related theta power are an underlying mechanism for executing all four cognitive control strategies, in alignment with many previous studies (Cooper et al., 2015; Cavanagh et al., 2009; Cavanagh et al., 2012; Cavanagh &

Frank, 2014; Cavanagh & Shackman, 2014; Cohen & Donner, 2013). The statistical analyses comparing cognitive-control strategies showed that the two cognitive control mechanisms with the largest theta power were reactive control (mean = 6.55 dB; stdv = 3.83 dB) and inhibitory control (mean = 6.28 dB; stdv = 2.83 dB). This result is also reflected in Figure 4, with the darkest regions, and therefore highest significant ERSP power values, of the entire reactive control plot (Figure 4-D) and entire inhibitory control plot (Figure 4-F) being located within their respective outlined boxes. This is a particularly interesting finding, as these two cognitive control mechanisms involve changing a previously engaged action strategy, also known as effortful control. Effortful control is defined as the ability to inhibit a dominant response in order to perform a subdominant response and recruits ACC activation (Rothbart & Posner, 2005). Thus, it may be that theta power is particularly important for applying effortful control and thereby being able to accurately override a prepotent response, as required by both the Nogo trials of the Go/Nogo task and the AY trials of the AX-CPT.

Future research should explore if oscillation phase might be important for these mechanisms. These results could signify the beginning of a numerical framework for comparing cognitive control strategies on their theta power values. It has been suggested that theta power values might be a more sensitive index of between-condition differences than ERP analyses (Cavanagh et al., 2012), so future work in cognitive neuroscience should focus on how the brain specifically uses theta power to interpret and coordinate information, through calculations like power-power correlations (Cohen et al., 2011). It has been theorized that transient theta dynamics are the basis for coordinating distant neural populations for flexible communication and execution of cognitive control (Cohen et al., 2011; Cohen & Donner, 2013), with midfrontal brain regions functioning as nodes for monitoring conflict and directing other brain regions for

goal-oriented behavioral adaptations to conflict (Cohen & Donner, 2013). For instance, the midcingulate cortex (MCC) is heavily connected with cortical and subcortical brain regions, and it has been theorized that the MCC acts as a hub for organizing brain systems across large spatial distances through frontal midline theta oscillations (Cavanagh & Frank, 2014). Understanding the nature of these theta dynamics in terms of their power is important, as it likely impacts forthcoming neural communications and computations that initiate the action selection and action production processes (Cavanagh & Frank, 2014). In fact, it has already been shown that increased theta power is associated with enhanced coupling between single neuron spikes in rats (Narayanan et al., 2013) and monkeys (Womelsdorf et al., 2010). Hence, as stated previously, this study provides support for the theory that frontal midline theta oscillatory activity is key to organizing neural processes underlying several strategies of cognitive control. However, what remains to be understood is precisely how these strategies are differentiated by the brain to result in rapid integration of information and communication with distal networks for goal-driven decision making. It has been suggested that synchronized changes in the phase angle of neural oscillatory activity can create time frames for segregating cortical populations (Cavanagh & Frank, 2014), but this needs to be investigated further. The scope of this project focused only on the power at each data point through convolution with Morlet wavelets, but future work will include phase values and measures such as Inter-Trial Coherence to try to understand the role of the mediofrontal brain regions in conducting cognitive control. To investigate the commonality or differences of the underlying information integration processes, phase values and inter trial coherence plots should be interpreted within target trials and between target trials, for they would provide more information about the temporal synchronization of the recorded EEG signals for each cognitive control mechanism. Integrating phase values into ERP analysis would allow us to

compute phase-locking to external stimuli (Sauseng & Klimesch, 2008; Cavanagh et al., 2012) and phase coherence between target trials. All of this analysis would provide more insight into the manner at which information is communicated to and interpreted by frontal midline regions to execute cognitive control.

5. Conclusion

This study was able to begin a classification system of cognitive control mechanisms on their respective amount of theta power 200-450 ms post-stimulus, or around the N2 interval. We established that reactive control and inhibitory control induced the highest theta power values, followed by proactive control, then response conflict. We also examined the peak theta activation time in order to see if temporal characteristics of the ERSP profiles were distinct. While peak theta activation values differed, the time at which they occurred was strikingly similar, suggesting a common underlying mechanism for information processing in the frontal midline regions of the brain for these cognitive control mechanisms.

By providing a differential analysis of the various cognitive control strategies, we gained more insight into the physiological basis of cognitive control and how this basis varies amongst strategies. Hence, this research added value to forthcoming functional connectivity studies as they try to further understand how neural populations operate to achieve cognitive control. Understanding the underlying mechanisms of cognitive control in a controlled setting could provide more information about the underlying mechanisms of clinical conditions, such as attention deficit hyperactivity disorder, obsessive compulsive disorder, Tourette's syndrome (Garavan et al., 1999), or schizophrenia (MacDonald & Carter, 2003; Ryman et al., 2018). This time frequency study also adds to the growing neurobiological framework for theorizing the mechanisms that can contribute to the development of anxiety and other psychiatric disorders

(Cavanagh & Shackman, 2014). Future work will focus on incorporating phase values, ERPs, and single trial regression (Cohen & Cavanagh, 2011) into the analysis of the various cognitive control strategies in order to elucidate the underlying mechanisms of information integration for executing each cognitive control strategy.

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