



Changes in Brain Activity Immediately Post-Exercise Indicate a Role for Central Fatigue in the Volitional Termination of Exercise

GRANT A. CHESBRO^{†1#}, CAMERON OWENS^{†1,2,3#}, MELODY REESE^{‡4,5,7}, LISA DE STEFANO^{‡6}, J. MIKHAIL KELLAWAN^{‡1}, DANIEL J. LARSON^{‡1}, MICHAEL J. WENGER^{‡7}, and REBECCA D. LARSON^{‡1}

¹Department of Health & Exercise Science, University of Oklahoma, Norman, OK, USA; ²Oklahoma Center for Geroscience and Healthy Brain Aging, University of Oklahoma Health Science Center, Oklahoma City, OK USA; ³Vascular Cognitive Impairment, Neurodegeneration and Healthy Brain Aging Program, Department of Neurosurgery, University of Oklahoma Health Sciences Center, Oklahoma City, OK, USA; ⁴Duke Aging Center, Duke University Medical Center, Durham NC, USA; ⁵Department of Anesthesiology, Duke University Medical Center, Durham, NC USA; ⁶Division of Child and Adolescent Psychiatry, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA; ⁷Department of Psychology, University of Oklahoma, Norman, OK, USA

These authors share first authorship, *Denotes undergraduate student author, †Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 17(1): 220-234, 2024. Electroencephalography (EEG) allows for the evaluation of real time changes in brain (electrocortical) activity during exercise. A few studies have examined changes in electrocortical activity using stationary cycling, but the findings have been mixed. Some of these studies have found increases in brain activity following exercise, while others have found decreases in brain activity following exercise. Hence, it is of importance to identify post-exercise changes in brain activity. Sixteen healthy, untrained subjects (8 males; 8 females) participated in the study. All 16 participants performed a graded exercise test (GXT) to volitional exhaustion on an upright cycle ergometer. Continuous EEG recordings were sampled before (PRE) and immediately following (IP) the GXT. Regions of interest were primarily the dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), and left and right motor cortex (MC). In the DLPFC, a frontal asymmetry index was also identified. There was a statistically significant increase in theta power in the DLPFC, VLPFC, and left and right MC from PRE to IP (all $p < 0.05$). There was also a shift towards right hemisphere asymmetry at the IP time point in the DLPFC ($p < 0.05$). Finally, there was an increase in alpha power from PRE to IP in the right MC ($p < 0.05$). EEG could prove to be an important way to measure the effects of central fatigue on brain activity before and immediately following exercise.

KEY WORDS: Electroencephalography, graded exercise test, exhaustion

INTRODUCTION

Electroencephalography (EEG) has been deemed the best use for non-invasive neuroimaging during and following exercise due to its excellent temporal resolution and ability to record cortical activity during movement (38), allowing for changes in brain activity immediately post-exercise to be recorded following volitional fatigue. The neurons within the brain produce electrical and chemical activity that produce currents that reach the surface of the scalp and can be recorded with EEG. Most of the efforts to identify cortical activity responses to exercise have used stationary cycling as the mode of exercise due to reduced upper body movement that can interfere with EEG recording (2, 10, 16). Some of these studies observed increases in brain activity before, during, and after cycling (2, 10, 21, 25, 33). The increase in cortical activity has been attributed to a variety of factors including elevated body temperature (36, 37), increased cardiovascular and respiratory activity (23, 44), enhanced cerebral blood flow (13, 24), and changes in emotional state (20, 41), an excited or urgent state of mind, increased attention or arousal, or increased effort and task difficulty (16, 33). While some studies have found increases in cortical activity following exercise, other studies have found decreases in brain activity following bouts of longer duration exercise (27, 28, 44). The contrary findings in these studies could be due to differences in methodology including intensity and duration differences in the protocols. These differences are of interest to investigate as it could help exercise physiologist and psychologists gain a better understanding of the impact that intensity, duration, and accumulated fatigue have on brain activity.

Fatigue is a complex area of study and has broad reaching applications to healthy individuals, athletes, and chronic disease populations (29). Fatigue encompasses both physiological and psychological factors and can be defined as a generalized response to stress over a given period (11). During exercise, fatigue is commonly defined as an inability to maintain the desired force output adequate to continue a task and is divided in two categories: peripheral and central fatigue (3, 4). Peripheral fatigue consists of factors that occur at the neuromuscular junction or in the muscle itself (1), while central fatigue relates to spinal and supraspinal changes that lead to decreased motor output (17).

Given the disparate findings about changes in electrocortical activity after cycling and the implications of these changes, further investigation into brain activity after different types of fatiguing exercise are needed. Increased knowledge about the cortical response to exercise could help exercise physiologists and psychologists gain a better understanding of the impact that intensity, duration, and accumulated fatigue have on brain activity. Additionally, understanding how the brain responds to exercise could prove to be important in rehabilitation practice to provide appropriate workload and during of exercise to enhance motivation and increase adherence to physical therapy programming. Therefore, the purpose of the present study was to investigate whether an acute bout of cycling would show increased post exercise brain activation compared to baseline pre-exercise activity.

METHODS

Participants

Twenty-two healthy, untrained (cycle < 2 days/week) individuals participated in the study. After data processing, 16 (8 males/8 females) participants (age: 25.1yrs \pm 3.1; height: 170.7cm \pm 9.7; weight: 73.8 kg \pm 16.2) were included in statistical analysis. A power analysis, using G*Power 3.1, for a pair samples *t*-test with an α of 0.05, β of 0.80, and a large effect size of 0.8 found that a minimum sample of 15 was needed for the study. Participants were recruited from the University of Oklahoma and the surrounding area by word-of-mouth, flyers, and emails. Participants were included if they answered no to all questions on the Physical Activity Readiness Questionnaire (PAR-Q) and had no limitations for exercises or diagnosed psychiatric conditions, or diagnosed diseases that would affect neuromuscular or cardiorespiratory function. Written informed consent was obtained from each participant prior to the study, and all methods were approved by the University of Oklahoma Institutional Review Board and complied with the Declaration of Helsinki. This research was carried out fully in accordance with the ethical standards of the International Journal of Exercise Science (34).

Protocol

The participants completed a graded exercise test (GXT) on an upright cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, Netherlands). EEG was recorded continuously before and after the GXT. Once seated on the cycle ergometer, two-minutes of baseline EEG recording was collected (PRE). The GXT began with an intensity equal to 1 watt per kilogram of body weight (W/kgBW) and increased by 0.5 W/kgBW every minute (26). The participants were instructed to maintain a self-selected cadence throughout the duration of the test, as freely chose cadence has been shown to minimize EMG activity and could minimize neuromuscular fatigue (6, 49). To limit eye movement artifact in the EEG signal, the participants were instructed to focus on the revolutions per minute (RPM) device before and after the test. Having participants focus on the RPM device to limit eye motion artifacts also ensured that the participants were maintaining their cadence. The cycling test was terminated when participants met one of the three following criteria: 1) If the participant could not maintain upper body stability, which could affect the ability to obtain clear EEG signal, 2) If the participant could not maintain their chosen RPM (drop by more than 10%), or 3) If the participant voluntarily ended the test (volitional exhaustion). Immediately following the test (IP) another 2-minutes of resting EEG was recorded.

A 32-electrode EEG cap (Hydrocel Geodesic Sensor Net; EGI, Eugene, OR, USA) was fitted and placed on the participant's head. All impedances were below 40 kilohms k Ω . The EEG signals sampled at 1000 Hz and were amplified using an EGI Net Amps 3000 (EGI, Eugene, OR, USA). Due to electrical current from the cycle ergometer, a grounding strap (Nasafes Grounding Cord and Strap, Nasafes, Clearwater, FL, USA) was attached to the participant's wrist to help lower impedance levels.

EEG data was pre-processed and processed using MATLAB R2018b. The data was band pass filtered (0.5 and 120 Hz) with a notch filter at 57-63 Hz. Pre-processing was conducted using manual rejection and rejection based on independent components through ICLabel. If more than 15 components were removed using ICLabel, the data was rejected. For data processing, a minimum of 20 1 second epochs for PRE and IP were used (19). Following data processing, 6 individuals were removed due to insufficient data for analysis. A Fast Fourier Transform with hamming windows was applied to the data, giving power spectral information in the following frequencies, which are the most commonly used in exercise studies: theta (θ ; 4.00-7.99 Hz), alpha (α ; 8.00-12.99 Hz), beta (β ; 13.00-29.99), and gamma (γ ; 30.00-80.00 Hz). The EEG channels were divided into 4 regions of interest (ROI) based on Brodmann's areas (8) corresponding with the 10-20 international EEG system. The ROIs for the present study were the dorsolateral pre-frontal cortex (DLPFC; electrodes: F3 and F4); ventrolateral pre-frontal cortex (VLPFC: F7, F8), motor cortex (MC; C3 and C4), and somatosensory cortex (SS; P3 and P4) (For electrode locations see Figure 1). The frontal cortex is involved with executive function, the motor cortex is involved with initiating and planning movement, and the somatosensory cortex is involved with detecting and processing sensory and motor information in the body. These are the most commonly used ROIs in exercise studies using EEG (2, 44). Frontal alpha asymmetry (FAA) was operationalized using the asymmetry index of alpha power in the DLPFC ($\log R$ alpha power - $\log L$ alpha power) (51). Positive scores represent greater alpha activity in the right DLPFC and greater activation in the left DLPFC; negative scores indicate greater alpha activity in the left DLPFC and greater activation in the right DLPFC.

All data were checked for normality using the Shapiro-Wilk test and were normally distributed ($p > 0.05$). Paired samples t -tests were used for all data analysis. Cohen's d was used to determine effect size for any significant findings where < 0.20 were considered negligible, between 0.20-0.49 were considered small, 0.50-0.79 were considered moderate, and ≥ 0.80 were considered large. Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The significance level for all tests was set *a priori* at $\alpha = 0.05$.

RESULTS

There was an increase in θ power from PRE to IP in both the Left-DLPFC (Electrode F3; $t = -3.606$, $p = 0.003$, $d = 0.88$) and Right-DLFC (Electrode F4; $t = -3.152$, $p = 0.007$, $d = 0.79$) (Figure 2A). None of the frequency bands were found to be different between the right and left DLPFC (Figure 2A). In addition, there were no differences in percent change from PRE to IP between the left and right DLPFC (Figure 2B). θ increased from PRE to IP in both the Left VLPFC (Electrode F7; $t = -2.423$, $p = 0.029$, $d = 0.61$) and Right VLPFC (Electrode F8; $t = -2.369$, $p = 0.032$, $d = 0.59$) (Figure 3A). There was a difference in θ ($t = -2.369$, $p = 0.032$, $d = 0.59$) and α ($t = -2.025$, $p = 0.041$, $d = 0.51$) between the Left and Right VLPFC at the PRE time point (Figure 3A). The Left and Right VLPFC were found to have a difference in Percent Change from PRE to IP in β ($t = -2.467$, $p = 0.026$, $d = 0.62$). (Figure 3B). There was a shift from left hemisphere asymmetry (PRE) to right hemisphere asymmetry (IP) ($t = 3.225$, $p = 0.006$, $d = 0.27$) (Figure 4).

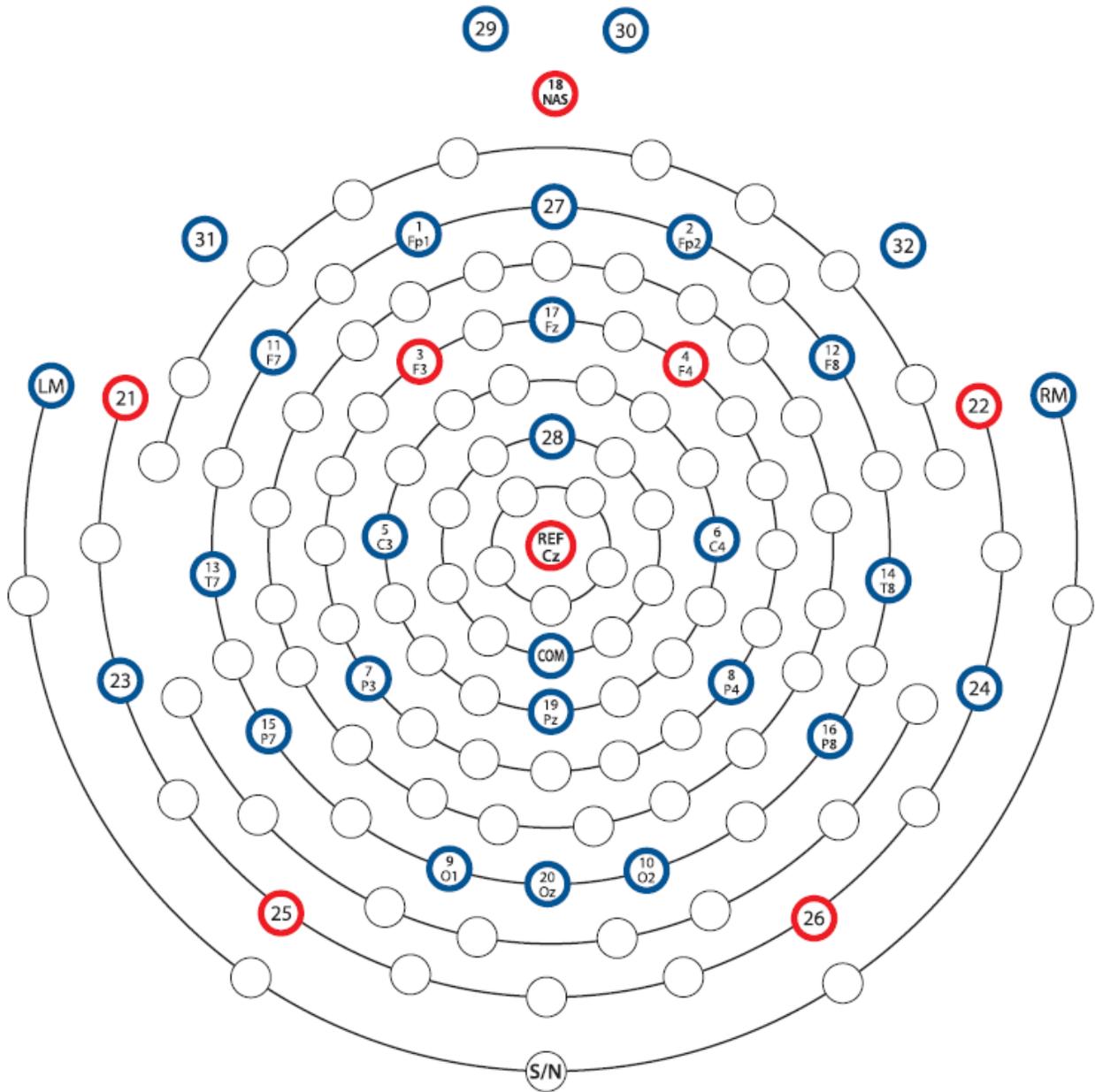


Figure 1. Electrode map for HydroCel Geodesic Sensor Net 32 Channel. EGI, Eugene, OR, USA
Statistical Analysis

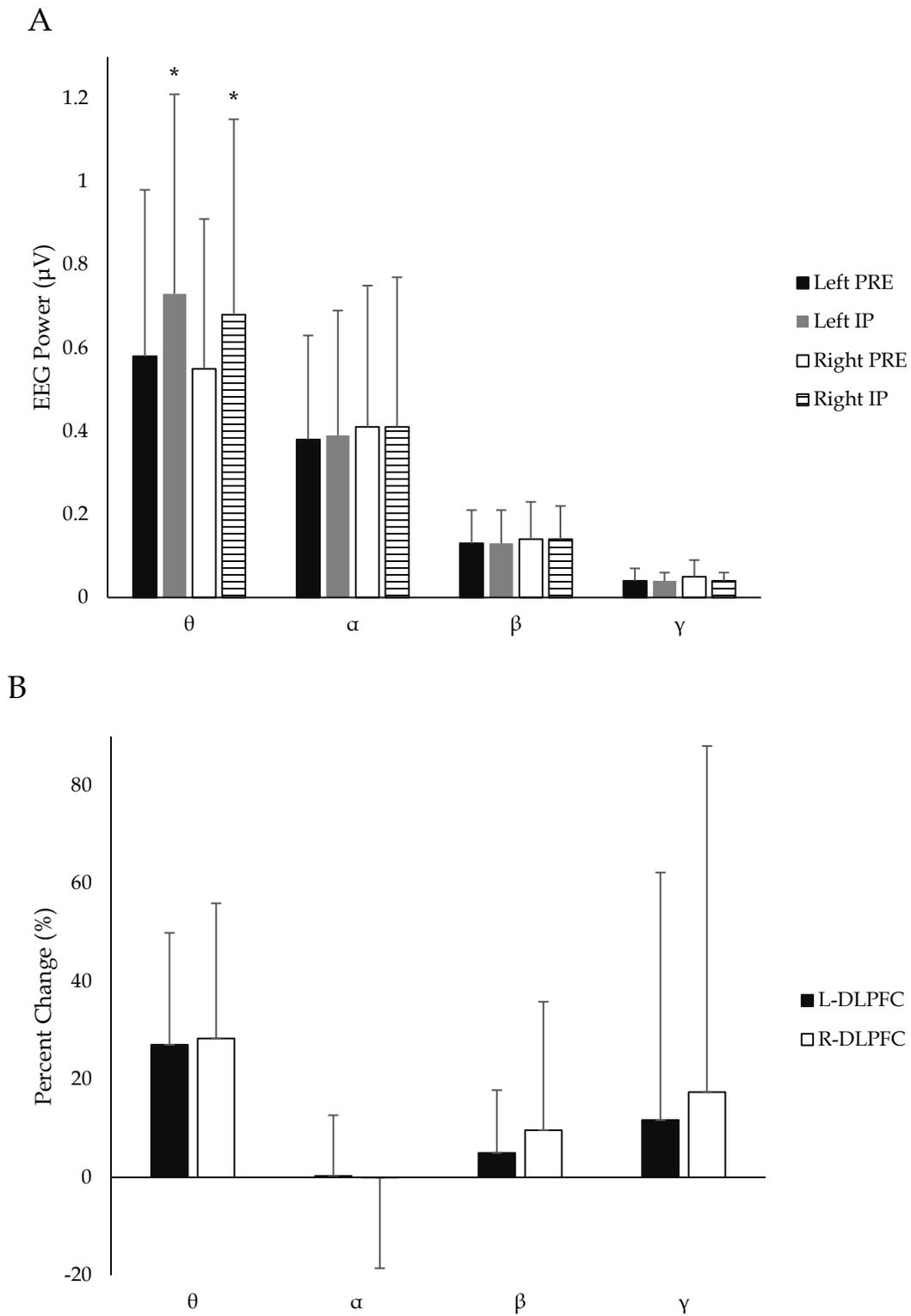


Figure 2. DLPFC. A: EEG Power. B: Percent Change from PRE to IP. * - Difference from PRE. $p < 0.05$. All Values are Mean \pm SD.

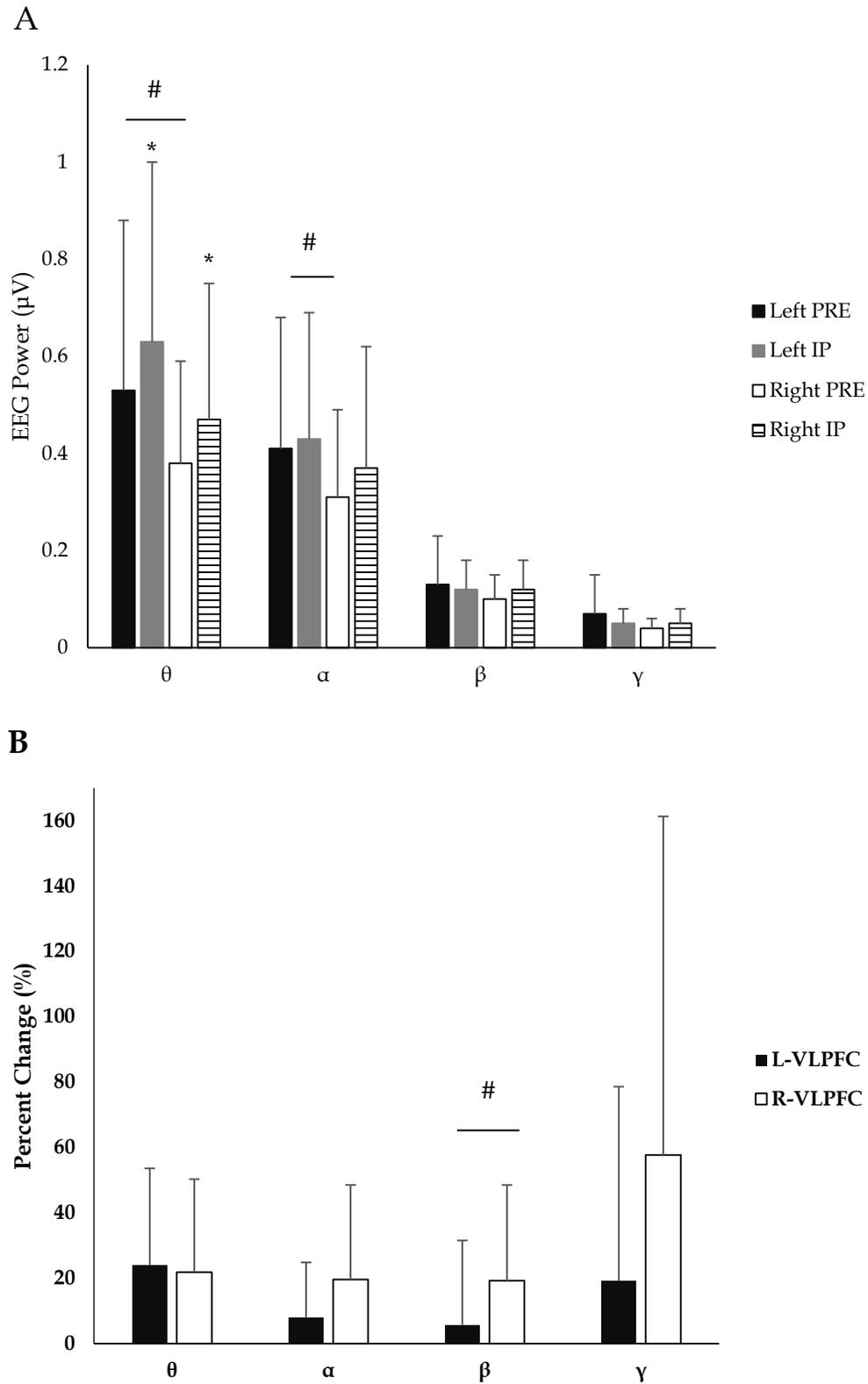


Figure 3. VLPFC. A: EEG Power. B: Percent Change from PRE to IP. * - Difference from PRE. # - Difference between limbs. Difference $p < 0.05$. All Values are Mean \pm SD.

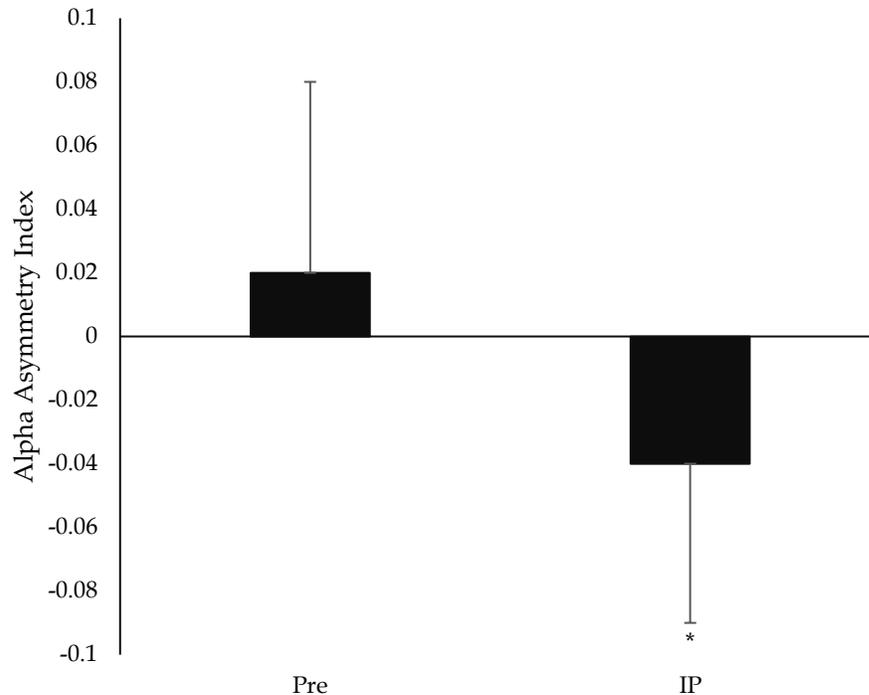


Figure 4. Frontal Alpha Asymmetry. Positive values indicate greater activation of the left hemisphere. Negative values indicate greater activation of the right hemisphere. * - Difference from PRE. Values are Mean \pm SD.

There was an increase in θ power following cycling in both the left motor cortex (L-MC; Electrode C3; $t = -3.056$, $p = 0.008$, $d = 0.36$) and the right motor cortex (R-MC; Electrode C4; $t = -3.737$, $p = 0.002$, $d = 0.35$) (Figure 5A). In addition, α increased in the R-MC from PRE to IP ($t = -2.882$, $p = 0.011$, $d = 0.35$) (Figure 5A). None of the frequency bands were found to be different between the R-MC and L-MC at either time point (Figure 5A). There was a difference in Percent Change from PRE to IP in the α ($t = -3.174$, $p = 0.006$, $d = 0.79$) and γ ($t = -2.791$, $p = 0.014$, $d = 0.70$) between the Left and Right MC (Figure 5B).

There was an increase in θ ($t = -2.921$, $p = 0.011$, $d = 0.73$), α ($t = -3.442$, $p = 0.004$, $d = 0.86$), β ($t = -3.102$, $p = 0.007$, $d = 0.78$), and γ ($t = -4.318$, $p = 0.001$, $d = 0.59$) in the left somatosensory cortex (Electrode P3; Figure 6A). In addition, there was an increase in θ ($t = -4.318$, $p < 0.001$, $d = 0.1.08$), α ($t = -2.719$, $p = 0.016$, $d = 0.68$), β ($t = -7.777$, $p < 0.001$, $d = 1.94$), and γ ($t = -3.213$, $p = 0.006$, $d = 0.80$) in the right somatosensory cortex (Electrode P4; Figure 6A). There were no differences between the left and right somatosensory cortex electrode at either time point for any of the frequencies (Figure 6A). In addition, there were no differences in Percent Change from PRE to IP in any of the frequency bands in between the left somatosensory cortex and the right somatosensory cortex (Figure 6B).

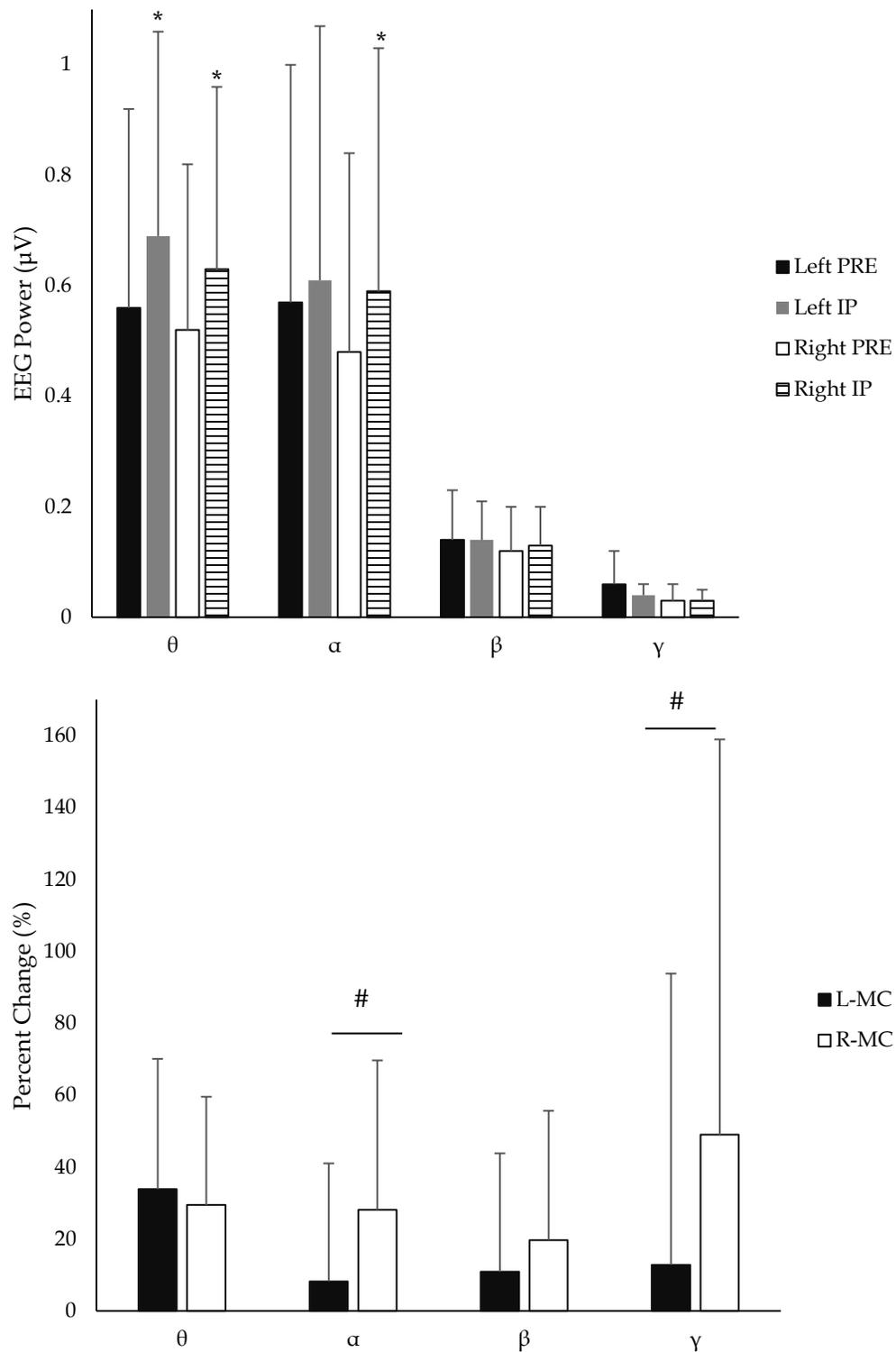


Figure 5. Motor Cortex (MC). A: EEG Power. B: Percent Change from PRE to IP. * - Difference from PRE. # - Difference between limbs. Difference $p < 0.05$. All Values are Mean \pm SD.

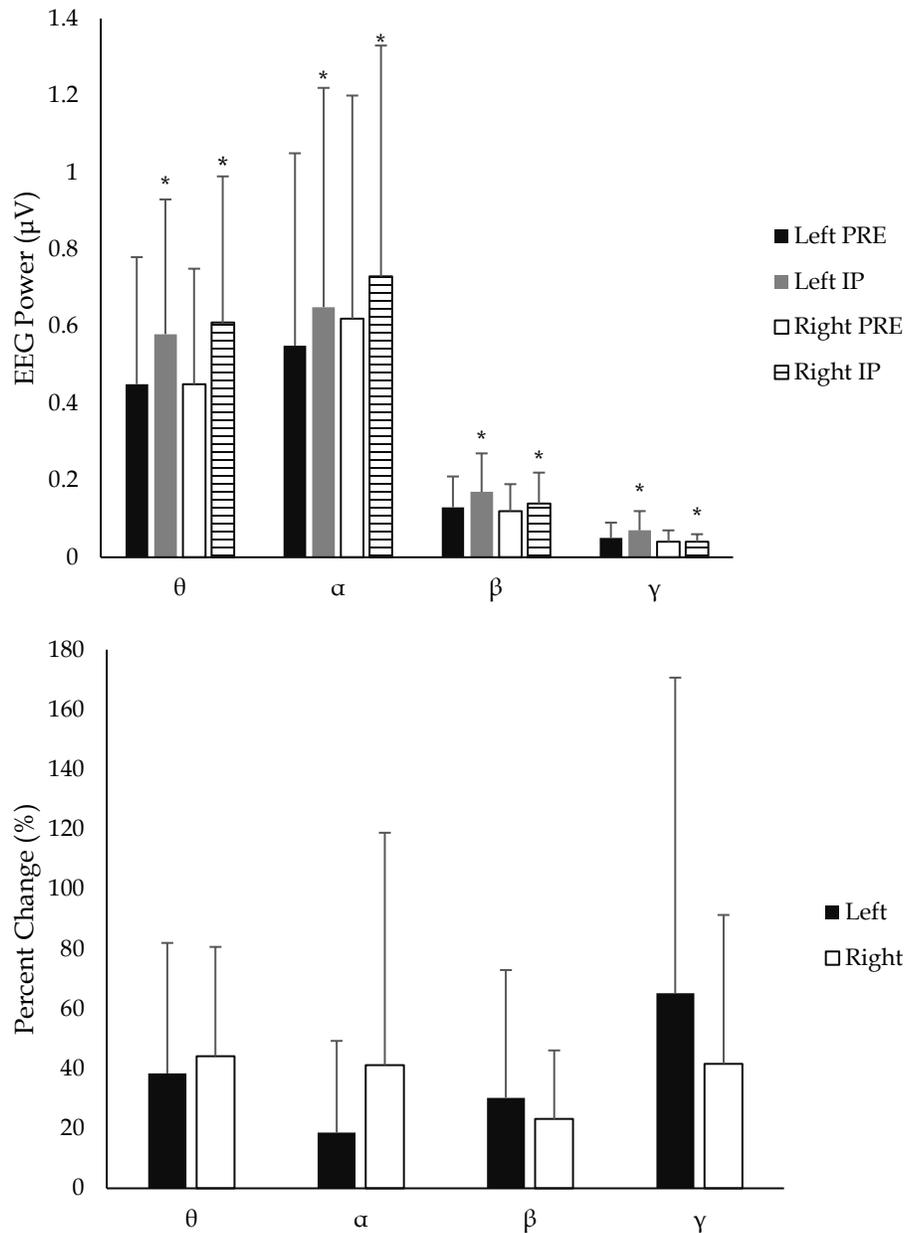


Figure 6. Somatosensory Cortex. A: EEG Power. B: Percent Change from PRE to IP. * - Difference from PRE. $p < 0.05$. All Values are Mean \pm SD.

DISCUSSION

The purpose of the present study was to examine changes in electrocortical activity before and after cycling. The major regions of interest: DLPFC, VLPFC, R-MC, L-MC, L-SS, and R-SS were analyzed for changes in cortical activity. The findings indicated increased activity in all the regions of interest. Overall, the results of the study indicate central fatigue as a mechanism for cessation of exercise evidenced by right hemisphere lateralization in the PFC (Figure 4) and increased α activity primarily in the R-MC.

There were increases in all frequency bands in the electrodes over the somatosensory cortex (Figure 5). These increases could be due to increased sensory information from the body being transmitted to the brain. In MRI studies, task relevant somatosensory stimulation led to increased activity in the contralateral somatosensory cortex (48). This study also found that increased activity in these cortical regions was associated with recruitment of the right dorsolateral prefrontal cortex (48). This activation may occur to help regulate activity in the somatosensory cortex to selectively gate irrelevant inputs (9, 48).

The PFC is involved with integration of perceptual information and formulates strategies for motor behavior and instructs the motor cortex to execute the strategy (14). In the present study, increases in θ band power were found from PRE to IP in electrodes over the DLPFC and VLPFC (Figures 1A & 2A), which is in line with findings from previous studies (2). Increases in θ have been shown to increase during more complex tasks and could indicate increased PFC activity to coordinate motor behavior strategy at the end of strenuous exercise. The DLPFC and VLPFC have been shown to be activated in situations where cognitive control is needed (43). In addition, the PFC is connected to sensory and motor cortical systems and may play a role in motor behavior and exercise termination (31, 43, 44).

The present study also found a shift towards right frontal asymmetry in the DLPFC following the termination of exercise (Figure 3). The DLPFC is involved with motivational processing, behavioral inhibition, and planning action (47, 50). Frontal asymmetry is a useful biomarker in research on emotional and behavioral reactions to stressful situations (30). Right PFC asymmetry has been associated with increases in anxiety and these feelings of anxiety may lead individuals to experience the exercise as more difficult (46). It has been shown that there is a negative trend in affective response as exercise approaches an individual's functional limit (15). In addition, pain sensitivity has been shown to be correlated to alpha asymmetry with high pain sensitivity being correlated to right hemisphere activation (39). Decreased activity in the PFC could affect goal attainment due to decreased activation of the PFC due to unpleasant afferent feedback from the body (44). Group III/IV muscle afferent activity due to metabolic perturbation in the working muscles may contribute to constrain neural drive to working muscles to limit further metabolic perturbation (5). Reduced motivation and withdrawal of approach due to unpleasant stimuli from intense exercise could lead to impaired central drive and cessation of activity.

In line with a previous study, increases in θ were observed in both the right and left motor cortex (Figure 4) (2). Increases in θ power possibly represent increased motor activity due to θ activity's role in coordinating motor actions (25). Interestingly, there was an increase in α power in the R-MC, but not a significant increase in α in the L-MC (Figure 4). All the participants in the present study reported that they were right leg dominant. Given that α activity is thought to represent cortical inhibition (18), this could indicate the contralateral cortex to the non-dominant leg experienced more inhibition at exercise termination, even during double limb exercise. In addition, when α activity is low cortical excitability is increased, and when α activity is higher cortical excitability is decreased (45). A previous study found that the same load performed by both arms resulted in higher fatigue level and greater α activity in the non-dominant limb (35).

Limb differences in fatigability could be due to greater failure in central drive in the non-dominant limb (52). Alpha activity has also been found to be inversely correlated with metabolic activity (38). Thus, the decrease in α could also indicate decreased metabolic activity in the R-MC, and perhaps competition for resources in with the PFC. The increases in α activity could be an indication of fatigue induced decrease in central drive. The increased activity in the PFC due to increased pain/discomfort and the increased inhibition in the MC may be related to reduce effort to withdraw from an uncomfortable stimulus.

It has been shown that humans have limited information processing capacity (7, 12) due to the brain's fixed metabolic resources (22) and inability to sustain activation in all neural structures at once (14). There are costs and benefits to information processing (42) and in this case the increased activation of the DLPFC and VLPFC may contribute to decreased activation in other structures in the brain. The increased α activity in the R-MC (Figure 4) may be due to competition for resources between the PFC and MC (32). This competition for resources to coordinate motor behavior strategy could have an opportunity cost where the motor cortex is inhibited to fund the work of the PFC.

The primary limitation of this study was EEG movement artifact during the GXT session, which limited the amount of usable data for further processing. Because of this limitation, we could not analyze the EEG data collected during exercise. Additionally, movement artifact during the GXT could have affected electrode connections and signal quality, leading to unusable data in 6 of the 22 participants, who were thus removed from data analysis. A possible recommendation would be the use of a recumbent cycle ergometer instead of an upright cycle ergometer to limit movement artefact (2). An additional limitation for the study was that no measure of peripheral fatigue was obtained.

There were increases in electrocortical activity from PRE to IP following a graded exercise test to fatigue. These findings are similar to others during cycling exercise to volitional exhaustion (2, 10, 16, 21, 33). Changes in EEG frequency bands, especially θ and α , could be important markers of central fatigue following exercise. In the present study increases in PFC θ activity were found and could indicate increased cortical activity to coordinate motor behavior, which could cause competition for resources in with an opportunity cost of decreased resources for other active areas of the brain, mainly the motor cortex. In addition, increased PFC right hemisphere activation could indicate decreased motivation to continue a task and could lead to a reduction in central drive from the motor cortex. Finally, there was an increase in α activity in the R-MC which could indicate increased inhibition of motor control of the non-dominant leg.

Studying the brain-body interaction during exercise is critically important for understanding physiological processes associated with these brain changes in healthy and clinical populations. Biomarkers of exercise related brain activity changes could be used to optimize athletic training or evaluate the efficacy of clinical trial or exercise interventions in patients who have underlying cognitive or neurological conditions such as Multiple Sclerosis, Alzheimer's disease, Parkinson's disease, etc. In addition, future studies could examine if intensity and duration of exercise have

an effect of brain activity. In this study, potential neurophysiological biomarkers of fatigue following a graded exercise test to exhaustion have been identified. Larger studies will be needed to determine the extent to which these biomarkers are specific to exercise induced-fatigue and the extent to which this fatigue is associated with clinical neuropathology.

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