

EFFECTS OF ACUTE AEROBIC EXERCISE ON NEURAL INDICES OF
EMOTION REGULATION AND COGNITIVE CONTROL
IN INDIVIDUALS WITH PTSD SYMPTOMS

By

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ABSTRACT

Posttraumatic Stress Disorder (PTSD) is associated with emotion regulation difficulties and cognitive control deficits, which may exacerbate symptoms and negatively affect treatment outcomes. Cognitive emotion regulation strategies, such as cognitive reappraisal, can be especially challenging for individuals with PTSD due to heightened attentional bias toward negatively arousing stimuli and limited cognitive resources needed to regulate their emotional response. Therefore, it is crucial to identify alternative strategies that target both emotion regulation and cognitive control in PTSD. Aerobic exercise has emerged as a promising alternative intervention to target emotion regulation and cognitive control. However, research on the acute effects of exercise on these mechanisms in PTSD populations is limited. This study investigated the acute effects of a single bout of moderate-intensity aerobic exercise on subjective (e.g., self-reported emotional arousal) and neural (e.g., LPP) indices of emotion regulation, as well as behavioral (e.g., flanker task accuracy and reaction time) and neural (e.g., P300 and ERN) indices of cognitive control in individuals with clinically significant PTSD symptoms. Using a within-subject, pre- to post-test crossover design, 67 female participants completed a 20-minute moderate-intensity aerobic exercise session and a 20-minute time-matched sitting control session. Prior to and following each experimental session, participants completed an emotion regulation task and a letter flanker task to measure emotion regulation and cognitive control, respectively. I hypothesized that exercise would 1) enhance cognitive control – evidenced by increased P300, decreased ERN, and improved behavioral performance; and 2) improve emotion regulation – evidenced by decreased LPP and perceived emotional arousal during cognitive reappraisal. Lastly, I predicted improvements in cognitive control would mediate the effect of exercise on cognitive reappraisal success. Contrary to predictions, exercise

did not lead to significant changes in P300 or ERN amplitudes during the flanker task, relative to sitting. Behaviorally, although no significant difference in response accuracy was observed, following exercise, participants demonstrated a greater reduction in reaction time relative to sitting, indicating more efficient performance. For the emotion regulation task, exercise led to a significant increase in the early and late LPP during cognitive reappraisal trials relative to view-negative trials. However, this effect appeared primarily driven by a significant decrease in LPP during view-negative trials from pre- to post-test. Exercise also did not lead to a significant change in perceived emotional arousal relative to sitting. However, exercise led to a significant decrease in perceived effort using cognitive reappraisal. Lastly, results from the mediation analysis did not support my hypothesis that exercise-induced improvements in cognitive control mediate the effect of exercise on cognitive reappraisal success. Overall, while acute aerobic exercise did not enhance neural markers of cognitive control and cognitive reappraisal success, reductions in LPP during view-negative and decreased perceived effort during cognitive reappraisal suggest that aerobic exercise may positively influence cognitive emotion regulation processes. These findings provide preliminary support for the use of exercise to enhance emotion regulation processes in PTSD. Future research is needed to further investigate the acute and long-term effects of exercise on emotion regulation and cognitive control in PTSD, as these insights may inform future research on exercise-augmented treatments for PTSD to enhance treatment outcomes.

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INTRODUCTION

Posttraumatic stress disorder (PTSD) is a trauma-related disorder that develops following exposure to a life-threatening, shocking, or highly upsetting event (American Psychiatric Association, 2022). Symptoms of PTSD include intrusive thoughts (such as flashbacks), avoidance of stimuli associated with the traumatic event, negative changes in cognition and mood (such as feelings of guilt or shame), and hyperarousal/reactivity (American Psychiatric Association, 2022). PTSD affects many individuals, with a lifetime prevalence rate of 5.7% and a 12-month prevalence rate of 3.7% (Kessler et al., 2012).

PTSD is a debilitating mental illness associated with a wide range of mental health, physical health, and social consequences (Pacella et al., 2013; Ryder et al., 2018). Individuals with PTSD are more likely to suffer from comorbid psychiatric conditions such as depression (Campbell et al., 2007), substance use (Debell et al., 2014), and increased suicidal risk (Krysinska & and Lester, 2010), along with decreased social functioning (American Psychiatric Association, 2022; Kessler et al., 2012). Research has also found PTSD to be associated with adverse physical health outcomes such as obesity (Farr et al., 2014), cardiovascular disease (Gradus et al., 2015; Ryder et al., 2018; Sumner et al., 2015), and metabolic conditions such as type-2 diabetes (Vancampfort et al., 2016). Furthermore, PTSD is associated with sedentary behavior (Hall et al., 2015), a significant risk factor for many health problems (Rezende et al., 2014). Indeed, PTSD has been linked to reduced quality of life (Pacella et al., 2013) and higher healthcare utilization (Karthä et al., 2008). Collectively, these mental and physical health consequences highlight the importance of identifying effective treatments for PTSD.

PTSD, Emotion Regulation, and Cognitive Control

Emotion regulation impairment is a common factor linked to increased risk for many psychological disorders and often contributes to the maintenance of these disorders (Gross & Jazaieri, 2014; Sheppes et al., 2015). Emotion regulation is broadly defined as the process of influencing the emotions we experience, and the duration and intensity with which we experience them (Gross, 1998; Gross & Thompson, 2007). Individuals with psychological disorders have been shown to experience difficulties in regulating negative emotions (Aldao et al., 2010; Gross & Jazaieri, 2014; Sheppes et al., 2015). A meta-analysis of emotion regulation studies in clinical populations found that greater use of maladaptive emotion regulation strategies (e.g., suppression, avoidance, distraction) was associated with increased psychopathology, whereas greater use of adaptive strategies (e.g., cognitive reappraisal, problem-solving, acceptance) was associated with decreased psychological symptoms (Aldao et al., 2010).

PTSD, in particular, has been shown to be associated with elevated emotional reactivity (McLean & Foa, 2017) and impaired emotion regulation (Ehring & Quack, 2010; Fitzgerald et al., 2018; McLean & Foa, 2017; Seligowski et al., 2015). Exposure to a traumatic event may elicit intense and persistent negative emotions, and can lead to heightened emotional reactivity (e.g., hyperarousal). Furthermore, many PTSD symptoms are thought to be a function of or exacerbated by emotion dysregulation. For instance, PTSD is associated with hyperarousal and heightened negative emotions, such as fear, anger, guilt, or shame. Individuals may also find it difficult to express their emotions (i.e., emotional numbing) and tend to engage in emotional suppression or avoidance of trauma-related cues.

The relationship between emotion dysregulation and PTSD symptomology aligns with findings from a meta-analysis of 57 studies investigating the relationship between posttraumatic

stress symptoms and emotion regulation (Seligowski et al., 2015). Results from this meta-analysis revealed that greater posttraumatic stress symptom severity was associated with greater overall emotion dysregulation and greater reliance on maladaptive strategies such as rumination, suppression, and experiential avoidance (Seligowski et al., 2015). Additionally, a meta-analysis from Pencea and colleagues (2020) found that individuals with emotion regulation difficulties were at greater risk of developing PTSD symptoms following trauma exposure. Furthermore, recognizing the central role of emotion regulation in PTSD, evidence-based PTSD treatments, such as Cognitive Processing Therapy (CPT) and Prolonged Exposure (PE) therapy, may directly target cognitive emotion regulation or indirectly improve emotion regulation outcomes (McLean & Foa, 2017; Pencea et al., 2020). Therefore, identifying the mechanisms underlying emotion dysregulation in PTSD and developing targeted interventions remains a critical area of research.

There is considerable evidence supporting the role of cognitive control in emotion regulation processes. Cognitive control is broadly defined as a set of cognitive processes implicated in the implementation of goal-related behaviors (Cohen, 2017). There has been a growing literature highlighting the role of cognitive control deficits in the etiology and maintenance of PTSD (Aupperle et al., 2012; Bomyea et al., 2012). Effective cognitive control involves the engagement of attentional, goal-directed processes while inhibiting distracting or unrelated stimuli (Cohen, 2017). Deficits in cognitive control may increase vulnerability to posttraumatic stress symptoms following a traumatic event, such as re-experiencing symptoms (Anderson & Levy, 2009). Specifically, cognitive control deficits may lead to increased attentional bias toward trauma-related thoughts and difficulty disengaging from emotionally salient stimuli. For example, a cross-sectional study by Bomyea and colleagues (2012) found that

lower performance on the OSPAN task – a behavioral measure of attentional control and working memory – was associated with greater self-reported re-experiencing symptoms.

Cognitive control difficulties in PTSD have been shown to negatively affect processes specifically implicated in cognitive emotion regulation strategies (Hayes et al., 2012; Miller et al., 2017). Effective implementation of cognitive emotion regulation strategies relies on cognitive control processes such as attentional control, response inhibition, and cognitive flexibility (Ochsner et al., 2012; Ochsner & Gross, 2005). For instance, cognitive reappraisal is a commonly studied emotion regulation strategy that involves the reinterpretation of an emotionally salient stimulus in order to reduce its emotional impact (Gross, 1998; Gross & Thompson, 2007). Although cognitive reappraisal ability is consistently associated with positive psychological outcomes (Aldao et al., 2010; Gross & Jazaieri, 2014), it has also been shown to be a cognitively demanding strategy (Buhle et al., 2014; Ochsner & Gross, 2005). In order to implement cognitive reappraisal successfully, an individual needs to inhibit their initial emotional response and shift their mental state toward an alternative interpretation (Pruessner et al., 2020; Schmeichel & Tang, 2015).

Neuroimaging research supports the relationship between cognitive control deficits and emotion dysregulation in PTSD. Successful cognitive reappraisal relies on the activation of the prefrontal cortex and anterior cingulate cortex to modulate activity in parietal and subcortical (e.g., amygdala) regions implicated in emotional reactivity (Buhle et al., 2014; Ochsner et al., 2012). However, research has shown that individuals with PTSD exhibit decreased activation of cognitive control brain regions implicated in emotion regulation, such as the prefrontal cortex and anterior cingulate cortex, and hyperactivation of the amygdala in response to threatening stimuli (Hayes et al., 2012a; Hayes et al., 2012b). These findings suggest that individuals with

PTSD may experience an exaggerated response to trauma-related stimuli and limited cognitive resources needed to downregulate their emotional response. Therefore, it is possible that individuals with PTSD struggle to implement emotion regulation strategies due to deficits in cognitive control processes. Given the relationship between cognitive control deficits and emotion dysregulation in PTSD, identifying approaches to strengthen cognitive control could improve emotion regulation and enhance PTSD treatment outcomes.

Current Treatments for PTSD

Further supporting the importance of emotion regulation and cognitive control in the etiology and maintenance of PTSD are the theoretical models for psychological treatments for PTSD. The leading treatments for PTSD are cognitive processing therapy (CPT; Resick & Schnicke, 1992) and prolonged exposure therapy (PE; Foa & Kozak, 1986). CPT and PE are considered “gold standard” treatments that are “strongly recommended” by the American Psychiatric Association (American Psychological Association, 2017). Although there is considerable research supporting the efficacy of these interventions for PTSD (American Psychological Association, 2017; Watkins et al., 2018), each has its limitations that impact outcomes.

CPT is a form of cognitive-behavioral therapy that assists patients in identifying maladaptive thoughts, known as "stuck points," related to their traumatic event (Resick & Schnicke, 1992). For example, a trauma survivor may have maladaptive thoughts such as “It is my fault that it happened.” CPT aims to help patients challenge their maladaptive thoughts and adopt a more flexible interpretation of the consequences of their traumatic experiences. Identifying and challenging maladaptive thoughts can be difficult, as it requires significant demands on one’s cognitive resources (Buhle et al., 2014; Ochsner & Gross, 2005). Given that

individuals with PTSD exhibit deficits in cognitive emotion regulation skills, such as cognitive reappraisal, they may struggle to reap the benefits of a cognitively demanding treatment such as CPT. This may also be true for other cognitively demanding treatments for PTSD, such as Cognitive Therapy (CT) and Cognitive Behavioral Therapy (CBT).

Prolonged Exposure (PE) Therapy is an exposure-based treatment for PTSD (Foa & Kozak, 1986) that involves repeated exposure to trauma-related memories and stimuli through imaginal and in vivo exposures. The goal of PE is to facilitate fear extinction and reconsolidation of traumatic memories (Foa, 2011; Foa & Kozak, 1986). Although PE does not directly target emotion regulation, emotion regulation has been shown to be a strong predictor of treatment engagement and positive outcomes (Gilmore et al., 2020). Repeated exposure to traumatic memories requires patients to tolerate high emotional distress, which can be particularly challenging without adequate emotion regulation skills (Gilmore et al., 2020).

Although there is considerable research supporting psychological treatments for PTSD, many individuals do not respond to treatment or drop out prior to completing treatment. Multiple meta-analyses of PTSD treatments have shown high dropout rates for PE (24%) and CPT (29%), (Kline et al., 2018) and an average dropout rate of 36% across PTSD treatments (Imel et al., 2013). A recent meta-analysis of non-response rates in treatment studies for PTSD found significant non-response rates for CPT (48%), PE (40%), CBT (41%), and CT (21%; Semmlinger et al., 2024). Among patients who drop out of treatments for PTSD, many report the interventions to be highly distressing and difficult to tolerate (Alpert et al., 2020). Therefore, increasing patients' ability to regulate negative emotions and tolerate emotional distress may help improve treatment adherence. It is clear from this research that although current treatments for PTSD are highly efficacious, many patients do not see benefits from these interventions (Imel

et al., 2013; Kline et al., 2018; Semmlinger et al., 2024; Watkins et al., 2018). Therefore, it is essential to seek alternative or adjunctive treatments to improve PTSD treatment outcomes. Identifying adjunct treatments that target emotion regulation and cognitive control, which are important treatment mechanisms of gold-standard PTSD treatments, could significantly enhance patient outcomes.

Exercise as an alternative treatment for PTSD

In recent years, exercise has gained recognition as a promising treatment for mental health disorders, including PTSD (Alexandratos et al., 2012; Busch et al., 2016; Powers, Asmundson, et al., 2015; Stathopoulou et al., 2006). Exercise is described as planned, structured, and repetitive physical activity intended to improve or maintain physical fitness (American College of Sports Medicine, 2025; Caspersen et al., 1985). Exercise has been shown to have many health benefits, such as improved physical function, quality of life, and reductions in major health concerns such as cardiovascular disease and metabolic disease (CDC, 2020). Indeed, regular engagement in moderate-intensity exercise is associated with decreased all-cause mortality rates (CDC, 2020). In addition to the many physical and mental health benefits of exercise, exercise interventions are widely accessible and easily implementable in various settings (Hegberg et al., 2019). For instance, walking is an easily accessible form of exercise that, at moderate intensities, is associated with positive physical and mental health outcomes (American College of Sports Medicine, 2025).

Although research on exercise as a treatment for PTSD is in its early stages, emerging evidence suggests that exercise is efficacious at reducing PTSD symptoms (Björkman & Ekblom, 2022; Fetzner & Asmundson, 2015; Oppizzi & Umberger, 2018; Powers, Medina, et al., 2015; Rosenbaum et al., 2015; Whitworth et al., 2017). A recent meta-analysis examining the

effects of exercise interventions for PTSD across eleven studies found a small to medium effect ($ES = .46$) of exercise on reducing PTSD symptoms (Björkman & Ekblom, 2022). These findings align with previous systematic reviews and meta-analyses (Oppizzi & Umberger, 2018; Rosenbaum et al., 2015), further supporting the potential utility of exercise as a viable intervention for PTSD. Furthermore, exercise was shown to improve common comorbid symptoms of PTSD, such as decreased substance use, improved sleep, and improved quality of life (Björkman & Ekblom, 2022). In sum, these findings highlight the utility of exercise for improving overall well-being in individuals with PTSD.

Given the mental, cognitive, and physical health benefits of exercise, there is increasing research interest in exercise as an adjunctive treatment for PTSD (Bryant et al., 2023; Powers, Medina, et al., 2015). In a study that investigated the efficacy of a 12-session exercise-augmented prolonged exposure (PE) therapy intervention for PTSD, results revealed that those who engaged in moderate-intensity aerobic exercises prior to PE therapy exhibited significantly greater reductions in PTSD symptoms relative to those who received PE alone (Powers, Medina, et al., 2015). Despite these promising findings, research is still limited in this area, particularly regarding the mechanisms by which exercise may enhance PTSD treatment outcomes. Specifically, little research has examined neural mechanisms by which exercise-induced improvements in emotion regulation and cognitive control could optimize PTSD treatment effectiveness.

The Effects of Exercise on Emotion Regulation and Cognitive Control

Important to this work, exercise has been shown to be associated with enhanced emotion regulation (Bernstein & McNally, 2017, 2018; Ligeza et al., 2019) and cognitive functioning (Olson et al., 2016; Pontifex et al., 2019, 2021), making exercise an ideal candidate to improve

the proposed mechanisms in this study. Exercise has been shown to enhance self-reported (Bernstein & McNally, 2017) and neurophysiological markers (Ligeza et al., 2019) of successful emotion regulation. Research from a cross-sectional study found frequent physical activity to be associated with a greater decrease in a neural marker of emotional arousal (i.e., the late positive potential) when instructed to regulate their emotions while viewing negative images (Ligeza et al., 2019). Although these results are promising, the immediate effect of a single bout of exercise on neurophysiological markers of emotion regulation is understudied. Additionally, research is limited on the mechanisms by which exercise enhances emotion regulation in individuals diagnosed with PTSD. Therefore, research is needed to fill this gap in the literature to better understand the neurophysiological mechanisms by which exercise may lead to improved emotion regulation in PTSD.

Researchers have posited that exercise may improve emotion regulation and cognitive control through the activation of neural mechanisms that are impaired in individuals with PTSD (Hegberg et al., 2019; Ligeza et al., 2019). Mechanistic studies demonstrate that exercise is associated with increased performance on cognitive tasks and enhanced neurophysiological indices of cognitive control (Olson et al., 2016; Pontifex et al., 2015, 2021). Exercise-induced physiological arousal is a potential mechanism that may explain the effects of exercise on cognitive control (Pontifex et al., 2019). Indeed, research has demonstrated that exercise-induced physiological arousal leads to greater recruitment of cognitive resources needed to perform cognitively demanding tasks such as cognitive reappraisal (Lambourne & Tomporowski, 2010; Pontifex et al., 2019).

A significant body of literature has demonstrated enhanced cognitive control following an acute bout of exercise (for review, see Pontifex et al., 2019). However, these effects have yet

to be investigated in PTSD samples, who are more likely to exhibit deficits in cognitive control processing (Aupperle et al., 2012; Bomyea & Lang, 2016). Furthermore, the potential mediating effect of cognitive control on the relationship between exercise and emotion regulation has been largely unexplored. Therefore, this study aims to extend this body of research by exploring the mechanisms by which exercise improves emotion regulation in individuals with PTSD through enhanced cognitive control.

Current Study

Despite strong evidence supporting current treatments for PTSD, many individuals do not respond to these treatments (Alpert et al., 2020; Imel et al., 2013; Semmlinger et al., 2024), underscoring the importance of investigating the mechanisms that may facilitate improved treatment outcomes. The present study aimed to contribute to this knowledge gap by examining the effects of an acute bout of aerobic exercise on neurophysiological indices of emotion regulation and cognitive control – two mechanisms known to be impaired in individuals with PTSD. Specifically, this study aimed to 1) identify the neurophysiological effects of a single bout of aerobic exercise in individuals with clinically significant PTSD symptoms and 2) explore the potential mediating effects of exercise-induced cognitive control on the effect of aerobic exercise on emotion regulation in this population.

Event-related potentials

This study employed a multi-modal design integrating self-report, behavioral, and neurophysiological methods to measure emotion regulation and cognitive control. The neurophysiological modality of interest was event-related potentials (ERPs). ERPs are broadly defined as electrical activity on the scalp measured via an electroencephalogram (EEG) that is time-locked to a specific event (e.g., stimulus presentation or button press response; Luck, 2014;

Luck & Kappenman, 2011). ERPs have been identified in specific brain regions, which provide insight into the cognitive processes engaged during a task (Luck & Kappenman, 2011).

Event-related potentials offer several advantages over self-report and behavioral measures. ERPs are thought to reflect a more “objective” measure relative to self-report, which is more subjective and may be susceptible to bias (Luck & Kappenman, 2011). Additionally, ERPs have high temporal resolution, with the ability to measure the time course of neural processes in the order of milliseconds (Luck, 2014; Luck & Kappenman, 2011). This provides greater temporal specificity when attempting to disentangle the neural processes involved in cognitive tasks. Indeed, ERPs are valuable tools for assessing emotion regulation and cognitive control mechanisms implicated in PTSD (Hansenne, 2006), and when combined with self-report and behavioral outcomes, provide a more comprehensive assessment of the cognitive and affective effects of exercise.

To this end, this study aimed to utilize a multi-modal measurement approach to assess emotion regulation and cognitive control across two separate tasks. Specifically, emotional arousal assessed during and following an emotion regulation picture-viewing task was measured using “online” neural indices (i.e., late positive potential) measured via EEG, and self-report measured via questionnaire. During a letter flanker task (Eriksen & Eriksen, 1974), cognitive control was measured via behavioral performance (i.e., accuracy and reaction time), and via “online” neural indices of cognitive control (i.e., P300, ERN) were measured via EEG.

Late Positive Potential. The late positive potential (LPP) is a positive deflecting waveform that occurs approximately 400ms following the exposure of a visual stimulus and is maximal in the centro-parietal region of the scalp (Hajcak et al., 2010; Luck & Kappenman, 2011; MacNamara et al., 2022). The LPP has been shown to increase in amplitude following the

presentation of both highly arousing positive and negative stimuli (Krompinger et al., 2008) and is modulated by various emotionally arousing stimuli, including images, words, and threatening faces (Foti et al., 2010; Grecucci et al., 2019; MacNamara et al., 2022). The LPP exhibits greater amplitudes when individuals are presented with more intense emotional stimuli, such as erotic or mutilation images (Schupp et al., 2000). Due to the late positive potential's sensitivity to emotionally arousing stimuli, researchers have utilized this ERP as a neural marker of emotional arousal processes in the brain. Additionally, the LPP is sensitive to emotion regulation instructions to increase or decrease emotional arousal (MacNamara et al., 2022; Moser et al., 2009, 2014). Together, these characteristics make the LPP a reliable neural measure of emotional reactivity and regulation.

Research on the time course of emotional processing has differentiated the LPP into two general time windows: the early and late LPP (Thiruchselvam et al., 2011). The early LPP, which occurs 400-100ms post-stimulus onset, reflects early attention orientation to affectively arousing stimuli, whereas the late sustained LPP, occurring 1000 ms post-stimulus onset, reflects late-stage meaning-making processes regarding the emotional significance of the stimulus (MacNamara et al., 2022; Thiruchselvam et al., 2011).

Individuals with PTSD have been shown to exhibit an elevated early LPP (Fitzgerald et al., 2018), which may be due to an attentional bias towards negative arousing stimuli. Additionally, cognitive emotion regulation strategies such as cognitive reappraisal involve reinterpreting the meaning or significance of emotionally salient stimuli (Gross, 2002; Gross & John, 2003), typically reflected by an attenuated late LPP (Thiruchselvam et al., 2011). An EEG study on OEF/OIF/OND veterans demonstrated that successful use of cognitive reappraisal – as evidenced by an attenuated late LPP – was associated with lower PTSD symptoms at follow-up

(Fitzgerald et al., 2018). Although no ERP studies to my knowledge have examined the effect of an acute bout of aerobic exercise on neural indices of cognitive reappraisal success, Ligeza and colleagues (2019) found that individuals with higher self-reported exercise frequency were associated with a smaller LPP amplitude during reappraisal, suggesting that exercise facilitates successful emotion regulation.

P300. The P300 is a commonly studied event-related potential that is associated with various domains of cognitive control, such as attentional control and working memory, and is thought to reflect the accumulation of cognitive resources during stimulus processing (Polich & Kok, 1995). The P300 is a positive deflecting ERP that occurs approximately 300 ms following the presentation of a stimulus. The P300 amplitude is maximal at the parietal region and is thought to reflect attentional control processes and the recruitment of cognitive resources during task-related stimulus processing (Polich, 2007).

Indeed, a smaller P300 amplitude has been associated with various mental health disorders, including but not limited to anxiety, depression, substance use disorder, and PTSD (Araki et al., 2005; Hansenne, 2006; Javanbakht et al., 2011). Individuals with PTSD have been shown to exhibit significantly smaller P300 amplitudes relative to healthy controls (Araki et al., 2005). A diminished P300 amplitude has also been found in individuals with acute stress disorder (Han et al., 2018). This suggests that the P300 would be a suitable marker for investigating cognitive control among individuals with clinically significant PTSD symptoms.

Essential to this work, the P300 is modulated by exercise (Pontifex et al., 2015, 2021). Research has shown moderate-to-large increases in the P300 amplitude following a single bout of exercise (Hillman et al., 2009; Kao et al., 2020; Pontifex et al., 2015). For example, Chang et al. (2017) found an enhanced P300 amplitude during a Stroop task following an acute bout (20

minutes) of aerobic exercise. Researchers posit that moderate-intensity exercise may increase physiological arousal, leading to increased activation of brain regions implicated in cognitive control (i.e., PFC and ACC) – demonstrated by an enhanced P300 (Pontifex et al., 2019).

Error-Related Negativity. The error-related negativity (ERN) is a negative deflecting waveform that occurs approximately 0-100ms following the commission of an error (Falkenstein et al., 1991; Gehring et al., 2011). The ERN amplitude is maximal in the frontal-central brain region (Gehring et al., 2011) and is generated by the anterior cingulate cortex (ACC), a brain region implicated in cognitive control processes (Brázdil et al., 2005). There are different theories on the functional significance of the ERN. For example, the ERN is thought to reflect compensatory error-monitoring processes (Gehring et al., 1993; Moser et al., 2013), conflict monitoring (Yeung et al., 2004), or an affective response to errors (Hajcak & Foti, 2008). Research has demonstrated enhanced ERN amplitudes across clinical samples such as anxiety (Moser et al., 2013), and OCD (Fitzgerald & Taylor, 2015; Hajcak et al., 2008). Research on the effects of exercise on the ERN is currently limited, with considerable variability in findings. Some studies have shown a significant reduction in the ERN following exercise (Drollette et al., 2025), while others have shown a significant increase in ERN (Pontifex et al., 2021) or no change (Brush et al., 2022).

Although research on the effects of exercise on the P300 is promising, research is limited on the effects of exercise on the LPP and ERN. Furthermore, no study, to my knowledge, has examined these effects in PTSD populations. Therefore, this study fills an important gap in the literature by exploring the behavioral and neurophysiological effects of exercise on emotion regulation and cognitive control in a PTSD sample.

Hypotheses

In summary, I hypothesized that engaging in an acute bout of moderate-intensity aerobic exercise would enhance cognitive control processes (i.e., attentional control and inhibition). This enhanced cognitive control would be reflected in the increased recruitment of cognitive resources during the flanker task – as evidenced by an enhanced P300 and ERN amplitude. Enhanced cognitive control was also reflected behaviorally, as I predicted that participants would exhibit improved behavioral performance during the flanker task – as evidenced by greater response accuracy and faster reaction times.

Furthermore, I hypothesized that moderate-intensity aerobic exercise would also enhance emotion regulation processes. Specifically, I predicted that following the exercise session, participants would exhibit reduced neural responses to emotional stimuli – as evidenced by an attenuated LPP during an emotion regulation picture-viewing task.

Lastly, I hypothesized that improvements in cognitive control following exercise would mediate the effect of exercise on emotion regulation. Specifically, I predicted that individuals who demonstrated greater exercise-induced enhancements in cognitive control during the flanker task (as reflected in increased P300 amplitude) would also exhibit greater reductions in LPP amplitude during the emotion regulation task. This mediating effect would suggest that exercise facilitates greater emotion regulation by strengthening cognitive control processes necessary for cognitively demanding emotion regulation strategies such as cognitive reappraisal.

METHOD

Sample Size Justification

A power analysis conducted using G*Power (3.1.9.7) indicated that a sample size of 50 would be sufficient to detect a minimum effect size of $d = 0.40$ (power = 0.80) using a Session $\times$ Time $\times$ Trial Type univariate multi-level model. Previous research has found medium-to-large effects of a single bout of exercise on the P300 (Cohen's $d = 0.60$ to 1.90 ; Pontifex et al., 2019) and has been demonstrated in both healthy and clinical samples (Pontifex et al., 2021). Currently, no studies have measured the acute effects of aerobic exercise on neural indices of emotion regulation success (i.e., late positive potential), making it difficult to predict an effect size for emotion regulation. Therefore, a minimum sample size of at least 50 participants should be sufficient to detect a significant effect using a Session $\times$ Time $\times$ ER Instruction univariate multi-level model.

Participant Eligibility and Recruitment

Sixty-seven female adults (ages 18-34) were recruited from the Michigan State University student research pool (SONA) for partial course credit. Inclusion criteria for this study were female adults currently experiencing clinically significant levels of PTSD using the Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5; Weathers et al., 2013). Participants were eligible if they endorsed a PCL-5 total score > 30 , which is the cutoff score for clinically significant PTSD symptoms and reflects a probable diagnosis of PTSD (Blevins et al., 2015).

Exclusion criteria for this study were assessed using the Physical Activity Readiness Questionnaire (PAR-Q; Adams, 1999), which screened for any cardiovascular (e.g., Coronary Artery Disease, Heart Failure, High Blood Pressure) or metabolic disease (e.g., Diabetes), or any orthopedic limitations (e.g., Osteoporosis) that may interfere with safely participating in aerobic

exercise. Individuals with substance use disorder and severe psychopathology (e.g., schizophrenia) were excluded, as these conditions are also associated with decreased cognitive functioning (Bowie & Harvey, 2006; Bruijnen et al., 2019). Participants with current stimulant use were also excluded, given that stimulants are associated with increased cognitive control (Smith & Farah, 2011). Participants were also excluded if they had a history of head trauma resulting in loss of consciousness for more than five minutes, epilepsy, or hearing, visual, or other physical or mental impairments that could interfere with the collection of quality neurocognitive data.

Sixty-seven participants were enrolled in the study. Of the participants enrolled, 14 did not complete their second session. Due to my use of a repeated measures multi-level model data analytic strategy, participants with partial data were retained in the study (see statistical analyses for additional details). Therefore, only two participants were excluded due to excessive EEG artifacts across both sessions, which led to unusable EEG data. Thus, the final sample size consisted of 65 participants. See Figure 1 for the CONSORT flow chart and Table 1 for demographics.

Experimental Design and Procedure

Figure 2 presents a visual depiction of the study design. During the first session, eligible participants completed the informed consent process and were then fitted with a Polar OH1 heart rate monitor (Polar Electro Oy, Kempele, Finland) across their chest to assess participants' heart rate during the experimental tasks. Participants then completed the baseline questionnaire during EEG setup.

Following convention (Pontifex et al., 2019), this study utilized a within-subject crossover design with pre-and post-test assessments. Each participant visited the laboratory on

two separate days, approximately 5-10 days apart. Each participant was randomized into two different session orders (Session 1: exercise, Session 2: sitting; or Session 1: sitting, Session 2: exercise). The order of the sessions was counterbalanced across participants to ensure that the results were not affected by the order in which the sessions were completed. As described in a review by Pontifex and colleagues (2019), a within-subject crossover design has several strengths. This design allows for pretest and posttest comparisons to assess change in hypothesized outcomes, and the inclusion of a sitting control session helps isolate the unique effects of the exercise intervention. Additionally, the within-subjects design allows participants to serve as their own control, which controls for individual differences in cognitive control and emotion regulation. Lastly, the counterbalanced design controls for potential practice effects.

Aerobic Exercise Session

The exercise session was adapted from Pontifex et al. (2021). During this session, participants walked on a treadmill at a moderate intensity (65-75% age-predicted HR_{max}) for 20 minutes. Exercise intensity was continuously monitored using a Polar OH1 heart rate monitor (Polar Electro Oy, Kempele, Finland), which was strapped to the participant's chest prior to starting the exercise session. Age-predicted HR_{max} was calculated for each participant using the following formula: ($HR_{max} = 220 - \text{Age}$; Fox et al., 1971). If a participant's heart rate exceeded 75% of their age-predicted HR_{max} , the research participant would adjust the treadmill speed or incline to help the participant decrease their workload. Alternatively, the participant's workload was increased if the participant's heart rate fell below 65% of their age-predicted HR_{max} . Additionally, participant ratings of perceived exertion (OMNI scale of perceived exertion; Robinson et al., 2011) and ratings of distress were assessed verbally by a research assistant in two-minute intervals. These subjective measures were used in conjunction with the heart rate

measure to ensure that participants were exercising at moderate intensity. To ensure that the observed effects were not due to differences in participant-research assistant interaction and non-exercise-related stimuli, during the exercise session, participants watched an emotionally neutral video (Wonders of the World, 2011). Following the exercise session, participants rested until their heart rate returned to within 10% of their resting heart rate (approximately 5 minutes) before starting the post-assessments.

Sitting Session

During the control session, participants completed a 20-minute sitting session. The sitting session was also adapted from Pontifex et al. (2021) and served as a time-matched attentional control. Participants sat silently in a chair while viewing an emotionally neutral video (Wonders of the Universe, 2011). This attentional control was selected to isolate the effects of aerobic exercise on cognitive control and emotion regulation while also controlling attentional differences between the exercise and control sessions (Pontifex et al., 2019). The sitting task controls for attentional differences by providing participants with a task that is stimulating but does not increase physiological or psychological arousal due to the neutral content in the video. Furthermore, sitting and watching a TV show would represent a typical activity that an individual would engage in on a given day. Similarly to the aerobic exercise session, participants' heart rate, perceived exertion using the OMNI scale of perceived exertion (Robertson et al., 2000), and perceived distress were assessed in two-minute intervals by the research assistant. Following the sitting session, participants rested for approximately 5 minutes to match the resting period in the exercise session.

Pre- and Post-Intervention Tasks

Prior to and following the aerobic exercise (or sitting) session, participants completed a letter flanker task (Eriksen & Eriksen, 1974), followed by an emotion regulation picture viewing task (Moser et al., 2006, 2014) while their electrical brain activity was continuously recorded via electroencephalogram (EEG). For both tasks, stimuli were displayed on a Dell computer monitor, and stimulus presentation and timing were controlled using PsychoPy (Peirce, 2007).

Letter Flanker Task

Participants completed a letter flanker task (Eriksen & Eriksen, 1974) adapted from Moser et al. (2011) and Pontifex et al. (2021). The stimulus for the letter flanker task consisted of a series of five letters presented in rapid succession. The participants were instructed to identify the centrally presented letter amid congruent ('UUUUU') or incongruent ('UUVUU') flanking letters by pressing a button assigned to each letter. The letter flanker task consisted of 240 trials grouped into six blocks of 40 trials. Each block consisted of a different pair of perceptually similar letters (i.e., block 1: M and N, block 2: E and F, block 3: O and Q, block 4: I and T, block 5: U and V, block 6: P and R). To increase task difficulty and promote errors, the button assignment for the letters was switched halfway through each block (i.e., left button click for 'U' during the first half and right button click for 'U' during the second half). Furthermore, participants were encouraged to respond as quickly as possible while maintaining accuracy. Each trial began with the flanking letters displayed on the screen for 35 ms, followed by the target letter, which was displayed with the flanking letters for an additional 100 ms. The total stimulus was displayed for 135 ms. Following the stimulus presentation, a blank screen was presented to allow for the participant's response. The intertrial interval was equally distributed and varied between 1200, 1325, 1450, 1570, and 1700ms.

Practice Block. Prior to starting the letter flanker task, participants completed a practice block consisting of 12 trials to familiarize themselves with the timing and events of the task. The practice block was only completed prior to the pre-flanker task during both sessions.

Emotion Regulation Picture Viewing Task

The emotion regulation picture viewing task was adapted from Moser et al. (2014). Participants were presented with a series of negative high-arousing (e.g., car accident) and neutral low-arousing (e.g., spoon) images from the International Affective Picture System (IAPS; Lang et al., 2008). The task consisted of three ER Instruction trials: Reappraise-Negative, View-Negative, and View-Neutral. Prior to being presented with each image, participants were cued to either passively view (View-Negative and View-Neutral) the image or to use reappraisal (Reappraise-Negative) to “reinterpret aspects of the image in order to adopt a more neutral or positive perspective.”

The reappraisal instructions were adapted from Moser and colleagues (2014). Cognitive reappraisal instructions were tailored to reflect how reappraisal is typically implemented in psychotherapy. For example, Cognitive Processing Therapy emphasizes developing neutral rather than overly positive reinterpretation of trauma-related stimuli (Resick & Schnicke, 1992). Therefore, cognitive reappraisal instructions were adapted as follows: “Imagine the pictured scene from a neutral, less emotional perspective to decrease the intensity of your negative emotions. For example, when viewing an image of a car crash, one might imagine that everyone in the car crash survived.” Furthermore, participants were instructed to refrain from using detached reappraisal (e.g., interpreting an image as fake or from a scene in a movie). See Appendix C for verbatim instructions.

The emotion regulation task consisted of 90 trials (30 Reappraise-Negative, 30 View-Negative, and 30 View-Neutral), across two blocks. For each trial, participants first viewed an instruction phrase (“Reappraise Negative,” “View Negative,” or “View Neutral”) for 2000ms that instructed them on how to react to the following image. After the instruction phrase, a blank screen was presented for 500 ms, followed by a centrally presented white fixation cross for 500 ms. Following the fixation cross, an IAPS image was displayed for 6000 ms. Finally, a blank screen was displayed for 2500 ms to allow participants to relax and clear their minds before the next instruction phrase (See Figure 3 for a visual depiction of the trial sequence).

Practice Blocks. Prior to beginning the emotion regulation picture-viewing task, participants were given task instructions from a research assistant and completed two practice blocks. During the first practice block, a research assistant guided participants through a trial walkthrough and instructed participants to use reappraisal out loud for each picture presented. Research assistants provided feedback and examples to participants who exhibited difficulty using reappraisal during the practice. During the second practice block, participants were instructed to practice the instructions silently to familiarize themselves with the timing and events of the task. The first practice block consisted of 3 Reappraise-Negative trials, and the second practice block consisted of 12 trials (4 Reappraise-Negative, 4 View-Negative, and 4 View-Neutral). The practice blocks were only completed prior to the pre-emotion regulation task during both sessions.

Post ER Task Questionnaire. Consistent with previous emotion regulation research (Moser et al., 2014; Webster et al., 2022), following the emotion regulation task, participants completed a questionnaire that assessed self-reported reactions of emotional arousal for each ER Instruction (Reappraise-Negative, View-Negative, View-Neutral) using a 1 (Very Weak) to 7

(Very Strong) Likert scale. Similarly, perceived effort was assessed for each ER Instruction using a 1 (Very Little) to 7 (Very Much) Likert scale. Participants were asked to what extent they used reappraisal when viewing images during the “Reappraise-Negative” trials using a 1 (Not at all) to 7 (The whole time) Likert scale. Lastly, as a quality check, participants were instructed to briefly describe the strategies they used during each ER Instruction.

ERP Recording

Continuous EEG activity was recorded using a Neuroscan Quik-Cap (Compumedics, Inc., Charlotte, NC) with 64 electrodes embedded in a stretch-lycra cap following the International 10-10 system (Chatrian et al., 1985). Electrode recordings were referenced to averaged mastoids (M1, M2), with AFz serving as the ground electrode. Electrode impedance was maintained below 10 k Ω . Electrooculographic (EOG) activity generated by eye movements and blinks was recorded via electrodes placed above and below the left pupil and on the left and right outer canthi. EEG signals were digitized at a sampling rate of 1 kHz, amplified 500 times, and filtered from DC to 70 Hz using a Neuroscan SynAmps RT amplifier. EEG data was then imported into EEGLAB (Delorme & Makeig, 2004) and prepared for temporal ICA decomposition. To restrict ICA computation to task-related activity, data more than 200 ms prior to the first event marker and 200 ms after the final event marker was removed. Continuous data was filtered using a 0.05 Hz high-pass filter, and the mastoid channels were removed prior to ICA decomposition. ICA decomposition was performed using the extended infomax algorithm to extract sub-Gaussian components using the default settings called in the MATLAB implementation of this function in EEGLAB with the block size heuristic drawn from MNE-Python (Gramfort et al., 2013). Following the ICA decomposition, the eyeblink artifact components were identified using the *icablinkmetrics* function (Pontifex et al., 2017), and the

EEG data was reconstructed with eyeblink artifacts removed. Following ICA ocular correction, offline data processing and extraction of ERP data was conducted using Brain Vision Analyzer 2 (BrainProducts, Gilching, Germany).

For the letter flanker task, stimulus- and response-locked data were segmented into individual epochs -200 to 800 ms around the stimulus and response, respectively, and baseline corrected at -200 to 0 ms. Stimulus- and response-locked epochs were filtered using a zero-phase shift low-pass filter at 30 Hz. Physiological artifacts were detected using a computer-based algorithm based on the following criteria: a voltage step greater than 50 μV , a voltage difference greater than 200 μV within a 200 ms interval, and a voltage exceeding $\pm 100 \mu\text{V}$. Participants with fewer than four accepted trials were excluded from the analysis. The P300 component was then extracted at the Pz electrode site as the mean amplitude of 300 to 600 ms following stimulus onset. The ERN/CRN were extracted for error and correct trials at the FCz electrode site as the mean amplitude of the 0 to 100 ms post-response window.

For the picture-viewing task, stimulus-locked data were segmented into individual epochs from -500 to 6000 ms around the stimulus onset, and baseline corrected at -500 to 0 ms. Stimulus-locked epochs were filtered using a zero-phase shift low-pass filter at 20 Hz. Physiological artifacts were detected using a computer-based algorithm based on the following criteria: a voltage step greater than 50 μV , a voltage difference greater than 300 μV within a 200 ms interval, and a voltage exceeding $\pm 200 \mu\text{V}$. Following convention, participants with fewer than six accepted trials in each ER Instruction (View-Negative or Reappraise-Negative) were excluded from analyses (Moran et al., 2013). Consistent with previous research (Moser et al., 2014), the LPP was extracted at the CPz electrode site and averaged at the early (400–1000ms) and late (1-2s, 2-3s, 3-4s, 4-5s, 5-6s) post-stimulus time windows.

Self-Report Measures

Baseline Measures

Upon enrollment in the study, self-reported PTSD symptoms were assessed using the Posttraumatic Stress Disorder Checklist for DSM-5 (Weathers, Litz, et al., 2013), self-reported exercise frequency was assessed using the International Physical Activity Questionnaire, and demographics were collected via Qualtrics (see Appendix for a full description of all baseline intervention measures).

The Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5; Weathers et al., 2013) is a 20-item self-report questionnaire designed to assess PTSD symptoms. The PCL-5 is commonly used in psychological research to screen for PTSD in community populations and can be used to provide a provisional diagnosis (Weathers, Litz, et al., 2013). Items on the PCL-5 ask the extent to which an individual has experienced symptoms of PTSD in the past month. Responses are rated on a 5-point Likert scale (0 = “Not at all” to 4 = “Extremely”). Total symptom severity scores are determined by summing the scores for each of the 20 items. Additionally, the PCL-5 can be used to assess severity scores for the following symptom clusters: intrusive thoughts, avoidance, negative cognitions and mood, and hyperarousal and reactivity (Weathers et al., 2013). A psychometric evaluation indicated that a cutoff score > 30 on the PCL-5 reflects a probable diagnosis of PTSD (Blevins et al., 2015). Although a clinical interview using the Clinician-Administered PTSD Scale (CAPS-5) is needed to confirm a diagnosis of PTSD (Weathers et al., 2018), the PCL-5 is strongly correlated with the CAPS-5. Furthermore, the PCL-5 is a widely used self-report measure of PTSD symptom severity that has been shown to have high validity and reliability (Blevins et al., 2015).

Manipulation Check Measures

Perceived Exertion. Following convention (Pontifex et al., 2019), subjective ratings of physical exertion were measured during the exercise and sitting sessions using the OMNI Rating of Perceived Exertion scale (OMNI RPE; Robertson et al., 2000). The OMNI RPE measures perceived physical exertion on a 0 (not tired at all) to 10 (very, very tired) scale and is a reliable predictor of subjective physical exertion (Robertson et al., 2000). When individuals make subjective ratings of physical exertion, they rely on multiple physiological and psychological cues such as heart rate, pain, and fatigue (Robertson & Noble, 1997). Although physiological measures of exercise intensity (i.e., HR) and perceived exertion are highly correlated (Muyor, 2013), individuals may differ in their perceptions of physical activity intensity. Therefore, obtaining both physiological and subjective ratings of physical exertion provides multiple sources of information about exercise intensity for the purposes of delineating the mechanisms by which exercise increases cognitive control and emotion regulation.

Statistical Analyses

All primary analyses were performed using the lme4 (Bates et al., 2015), lmerTest (Kuznetsova et al., 2017), and emmeans (Lenth, 2017) packages in R version 4.4.2 with Kenward-Roger degrees of freedom approximations. Primary analyses were conducted separately using a univariate multi-level model with Participant and Session $\times$ Participant interaction included as the random intercept. Significance tests were set at $p < .05$. Standardized effect sizes were calculated using Cohen's d (with 95% confidence intervals), with variance corrections for repeated-measures comparisons (d_{rm}).

Flanker Task Analyses

For the Flanker task, analysis of response accuracy was conducted using a 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) univariate multi-level model. Analysis of reaction time was conducted using a 2 (Session: sitting, exercise) $\times$ 2 (Time: pretest, posttest) $\times$ 2 (Accuracy: correct, error) univariate multi-level model. P300 amplitude at Pz was conducted using a 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) $\times$ 2 (Congruency: congruent, incongruent) univariate multi-level model. Post-hoc decompositions of significant Session $\times$ Time $\times$ Congruency interactions were conducted by examining the Time $\times$ Congruency interaction within each Session. Analysis of ERN amplitude at the FCz electrode site was conducted using a 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) $\times$ 2 (Accuracy: error, correct) univariate multi-level model. Post-hoc decomposition of the significant Session $\times$ Time $\times$ Accuracy interaction was conducted by examining the Time $\times$ Accuracy interaction within each Session.

Emotion Regulation Task Analyses

For the emotion regulation picture viewing task, analyses of the early and late LPP amplitude at the CPz electrode site were conducted using separate 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) $\times$ 2 (ER Instruction: Reappraise-Negative, View-Negative) univariate multi-level models. Lastly, analysis of self-report emotional arousal was conducted using a 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) $\times$ 2 (ER Instruction: Reappraise-Negative, View-Negative) univariate multi-level model. Post-hoc decomposition of significant Session $\times$ Time $\times$ ER Instruction interactions was conducted by examining the Time $\times$ ER Instruction interaction within each Session.

Secondary Self-Report Analyses

Analyses of self-report data collected during the pre-and post-test questionnaires were conducted using separate 2 (Session: sitting, exercise) $\times$ 2 (Time: pre-test, post-test) univariate multi-level models. Post-hoc decompositions of significant Session $\times$ Time interactions were conducted using pairwise comparisons of time within each Session.

Mediation Analyses

Exploratory analyses examined whether exercise-induced increases in cognitive control mediated the effect of exercise on emotion regulation. Mediation analysis was conducted in R version 4.4.2. The analysis followed the causal mediation framework, estimating both the direct and indirect effects. Indirect effects were estimated using 1,000 bootstrap resamples using the boot package (Canty, 2002).

RESULTS

Manipulation Check

Overall, the exercise protocol achieved my criteria for moderate-intensity exercise. Participants' mean heart rate (HR) during the exercise session was 151.1 (8.9), which was within the target heart rate of 150-160 bpm. During the exercise sessions, the average treadmill speed was 2.7, and the average incline was 2.2. Participants reported significantly greater perceived exertion during the exercise session compared to the sitting session ($t(50) = 12.8, p < 0.001, d_{rm} = 2.23$ [95% CI: 1.67 to 2.78]). Additionally, participants did not report increased emotional distress during the exercise session as no significant difference on the Feeling Scale was observed between sessions ($t(50) = 0.0, p = 1.0, d_{rm} = 0.00$ [95% CI: -0.30 to 0.31]). See Table 2 for mean and standard deviation values for each manipulation check.

Flanker Task Results

Behavioral Results

Response Accuracy. Response accuracy was high for the entire sample ($M = 90.5\%$, $SD = 6.4$). Results revealed a main effect of Time $F(1, 344) = 5.6, p = 0.018, f^2 = 0.70$ [95% CI: 0.31 to 1.41], such that across both sessions, response accuracy increased from pre-test ($M = 90.3\%$, $SD = 6.9$) to post-test ($M = 90.8\%$, $SD = 5.8$; $t(344) = 2.4, p = 0.018, d_{rm} = 0.13$ [95% CI: 0.02 to 0.25]). Results revealed no significant main effect of Session ($F(1, 52) = 1.7, p = 0.2, f^2 = 0.21$ [95% CI: 0.01 to 0.49]), and no significant interaction of Session $\times$ Time ($F(1, 344) < 0.1, p > 0.99, f^2 < 0.01$ [95% CI: 0.0 to 0.00]).

Reaction Time. Consistent with previous research, participants exhibited a speed-accuracy tradeoff as reaction times (ms) on error trials ($M = 348.1, SD = 60.8$) were significantly faster than reaction times on correct trials ($M = 412.6, SD = 49.6$; $t(340) = 20.4, p < 0.001, d_{rm} =$

1.67 [95% CI: 1.47 to 1.87]). Results did not reveal a significant main effect of Session or Time ($F_s < 1.3$, $p_s > 0.26$).

Further analyses revealed a marginal Session $\times$ Time ($F(1, 341) = 3.0$, $p = 0.08$, $f^2 = 0.01$ [95% CI: 0.0 to 0.05]) and Accuracy $\times$ Time interaction ($F(1, 341) = 3.7$, $p = 0.056$, $f^2 = 0.01$ [95% CI: 0.0 to 0.05]). All other interactions were not significant ($F_s < 1.3$, $p_s > 0.25$). Post-hoc analyses were conducted by creating a post-test minus pre-test difference score (negative scores reflect a decrease in reaction time from pre-test to post-test) and conducting a Session $\times$ Time $\times$ Accuracy univariate repeated measures multi-level model with participant entered as a random intercept. Results revealed a main effect of Session ($F(1, 189) = 3.8$, $p = 0.053$, $f^2 = 0.80$ [95% CI: 0.38 to 1.60]), such that participants exhibited a larger reduction in reaction time (ms) from pre-test to post-test during the exercise sessions ($M = -9.3$, $SD = 41.9$), relative to the sitting sessions ($M = 1.9$, $SD = 47.6$; $t(189) = 1.9$, $p = 0.053$, $d_{rm} = 0.39$ [95% CI: -0.01 to 0.79]). However, that difference did not remain significant following false discovery rate control (Benjamini-Hochberg critical alpha = 0.05). Results also revealed a main effect of Accuracy ($F(1, 166) = 4.3$, $p = 0.039$, $f^2 = 0.82$ [95% CI: 0.39 to 1.64]), such that participants exhibited a significantly larger reduction in reaction time (ms) from pre-test to post-test during correct trials ($M = -9.3$, $SD = 28.2$) relative to error trials ($M = 2.6$, $SD = 56.8$; $t(160) = 2.2$, $p = 0.032$, $d_{rm} = 0.33$ [95% CI: 0.02 to 0.64]).

ERP Results

Table 3 displays the mean (SD) amplitudes for the P300, ERN, and CRN. Figures 4 and 6 depict the stimulus-locked (P300) and response-locked waveforms (ERN/CRN) for the Flanker task, respectively. Figures 5 and 7 depict the bar graphs for the P300 and ERN, respectively.

P300. Results revealed a significant main effect of Congruency ($F(1, 313) = 47.0, p < 0.001, f^2 = 0.86$ [95% CI: 0.42 to 1.71]), such that participants exhibited a larger P300 amplitude during incongruent trials ($M = 6.8, SD = 3.9$) relative to congruent trials ($M = 5.6, SD = 3.9; t(309) = 6.9, p < 0.001, d_{rm} = 0.32, 95\% \text{ CI: } 0.23 \text{ to } 0.42$]). Across both sessions, the P300 decreased from pre-test ($M = 6.3, SD = 3.5$) to post-test ($M = 6.0, SD = 4.4$), but this difference was marginal ($t(313) = 1.9, p = 0.06, d_{rm} = 0.15, 95\% \text{ CI: } -0.01 \text{ to } 0.31$]). Results did not reveal a significant main effect of Session ($F(1, 51) = 1.9, p = 0.18, f^2 = 0.03$ [95% CI: 0.00 to 0.13]). Further analysis did not reveal any significant interaction effects ($F_s < 1.2, p_s > 0.28$).

ERN. Consistent with previous literature, results revealed a significant main effect of Trial Type ($F(1, 307) = 231.2, p < 0.001, f^2 = 0.99$ [95% CI: 0.50 to 1.95]), such that participants exhibited a larger ERN amplitude during error trials ($M = -4.4, SD = 5.6$), relative to correct trials ($M = 1.2, SD = 6.2; t(307) = 15.2, p < 0.001, d_{rm} = 1.75$ [95% CI: 1.48 to 2.01]). Results did not reveal any main effects of Session or Time, and no significant interactions were observed ($F_s < 0.5, p_s > 0.47$).

Emotion Regulation Task Results

Table 3 displays the mean (SD) early and late LPP amplitudes. Figures 8 and 9 depict the stimulus-locked (LPP) waveforms and topographic head maps during the exercise and sitting sessions, respectively. Figures 10 and 11 depict the bar graphs for the early and late LPP, respectively.

ERP Results

Early LPP. Results revealed a significant main effect of ER Instruction ($F(2, 1152) = 143.9, p < 0.001, f^2 = 1.22$ [95% CI: 0.66 to 2.38]). Consistent with previous emotion regulation literature (MacNamara et al., 2022), participants exhibited significantly larger early LPP

amplitude during View-Negative ($M = 4.2$, $SD = 5.0$) and Reappraise-Negative ($M = 4.8$, $SD = 5.0$) trials, relative to View-Neutral trials ($M = 1.1$, $SD = 4.3$; $t_s > 13.2$, $p_s < 0.001$). Furthermore, participants exhibited difficulty regulating emotions to negative images, as evidenced by a significantly larger early LPP amplitude during Reappraise-Negative trials relative to View-Negative trials ($t(1152) = 2.7$, $p < 0.001$, $d_{rm} = 0.19$ [95% CI: 0.05 to 0.33]). Results did not reveal a significant main effect of Session ($F(1, 51) = 0.5$, $p = 0.48$, $f^2 < 0.01$ [95% CI: 0.00 to 0.02]) or Time ($F(1, 1162) = 0.3$, $p = 0.6$, $f^2 < 0.01$, [95% CI: 0.0 to 0.02]). There was a significant main effect of Time Window ($F(1, 1152) = 80.4$, $p < 0.001$, $f^2 = 0.34$, [95% CI: 0.09 to 0.75]), such that the early LPP amplitude significantly increased from the 400-700ms ($M = 0.5$, $SD = 5.3$) to 700-1000ms ($M = 4.2$, $SD = 4.6$) time window.

Results also revealed a marginal Session $\times$ Time $\times$ ER Instruction interaction ($F(2, 1152) = 2.9$, $p = 0.053$, $f^2 = 0.03$ [95% CI: 0.0 to 0.10]). Post hoc decompositions of this interaction were conducted by creating Reappraise-Negative minus View-Negative difference scores to reflect the emotion regulation effect (Δ Reappraise: negative scores reflect a smaller early LPP amplitude during Reappraise-Negative trials relative to View-Negative trials). View-Negative minus View-Neutral difference scores were created to reflect emotional reactivity (Δ View-Negative: positive scores reflect a larger early LPP amplitude during View-Negative trials relative to View-Neutral trials). Analyses of the emotion regulation effect and emotional reactivity were conducted using separate Session $\times$ Time univariate repeated measures multi-level models with Participant and Session $\times$ Participant interaction entered as a random intercept.

For the emotion regulation effect (Δ Reappraise), results did not reveal a main effect of Session ($F(1, 59) = 1.7$, $p = 0.20$, $f^2 = 0.013$ [95% CI: 0.00 to 0.34]), or Time ($F(1, 323) = 0.7$, $p = 0.42$, $f^2 = 0.05$ [95% CI: 0.00 to 0.17]). However, there was a significant interaction of Session

× Time ($F(1, 323) = 9.8, p = 0.002, f^2 = 0.76$ [95% CI: 0.35 to 1.50]). During the exercise session, participants exhibited a significant increase in the early LPP from pre-test ($M = -0.4, SD = 3.4$) to post-test ($M = 0.9, SD = 4.4; t(321) = 2.7, p = 0.007, d_{rm} = 0.53$ [95% CI: 0.14 to 0.92]). No significant change in early LPP was observed during the sitting session ($t(325) = 1.7, p = 0.09, d_{rm} = 0.32$ [95% CI: -0.05 to 0.68]). For emotional reactivity (Δ View-Negative), results did not reveal a main effect of Session ($F(1, 56) = 0.1, p = 0.80, f^2 = 0.01$ [95% CI: 0.00 to 0.06]), or Time ($F(1, 321) = 0.1, p = 0.8, f^2 = 0.01$ [95% CI: 0.00 to 0.06]). There was a significant Session × Time interaction ($F(1, 321) = 5.1, p = 0.024, f^2 = 0.84$ [95% CI: 0.41 to 1.65]). However, no significant change in early LPP amplitude was observed during the exercise ($t(320) = 1.7, p = 0.09, d_{rm} = 0.29$ [95% CI: -0.04 to 0.63]) and sitting sessions ($t(323) = 1.5, p = 0.14, d_{rm} = 0.25$ [95% CI: -0.08 to 0.58]).

Late LPP. Results revealed a significant main effect of ER Instruction ($F(2, 3101) = 103.1, p < 0.001, f^2 = 1.25$ [95% CI: 0.67 to 2.42]). Consistent with previous emotion regulation literature (MacNamara et al., 2022), participants exhibited significantly larger late LPP amplitude during View-Negative ($M = 1.4, SD = 4.8$) and Reappraise-Negative ($M = 2.3, SD = 5.3$) trials, relative to View-Neutral trials ($M = -0.2, SD = 4.7$). Furthermore, consistent with previous studies examining emotion regulation in PTSD populations (Ehring & Quack, 2010; Pencea et al., 2020), participants exhibited difficulty regulating emotions to negative images, as evidenced by a significantly larger late LPP amplitude during Reappraise-Negative trials relative to View-Negative trials ($t(3101) = 5.2, p < 0.001, d_{rm} = 0.53, 95\% \text{ CI: } 0.32 \text{ to } 0.75$). Results did not reveal a significant main effect of Session ($F(1, 52) < 0.1, p = 1.0, f^2 > 0.01$ [95% CI: 0.00 to 0.00]). Results did reveal a main effect of Time ($F(1, 3116) = 8.6, p = 0.003, f^2 = 0.05, [95\% \text{ CI: } 0.0 \text{ to } 0.17]$), such that the late LPP significantly decreased from pre-test ($M = 1.4, SD = 4.7$) to

post-test ($M = 0.9$, $SD = 5.4$; $t(3116) = 2.9$, $p = 0.003$, $d_{rm} = 0.33$, [95% CI: 0.22 to 0.56]). There was a significant main effect of Time Window ($F(4, 3101) = 36.3$, $p < 0.001$, $f^2 = 0.88$, [95% CI: 0.43 to 1.74]), such that the late LPP amplitude reduced from the 1 to 6 second time window.

Further analysis revealed a significant ER Instruction $\times$ Time interaction ($F(2, 3101) = 4.5$, $p = 0.011$, $f^2 = 0.05$ [95% CI: 0.00 to 0.18]). Post-hoc decomposition of the ER Instruction $\times$ Time interaction was conducted by examining the difference in late LPP amplitude between each ER instruction within the pre-test and post-test. During the pre-test, the late LPP amplitude was larger during the Reappraise-Negative trials ($M = 2.2$, $SD = 4.8$), relative to the View-Negative trials ($M = 1.8$, $SD = 4.8$; $t(1537) = 2.0$, $p = 0.041$, $d_{rm} = 0.26$, 95% CI: 0.01 to 0.51]). However, this difference did not remain significant following false discovery rate control (Benjamini-Hochberg critical alpha = 0.037). During the post-test, the late LPP amplitude was significantly larger during the Reappraise-Negative trials ($M = 2.3$, $SD = 5.8$), relative to View-Negative trials ($M = 1.0$, $SD = 4.7$; $t(1510) = 5.2$, $p < 0.001$, $d_{rm} = 0.71$ [95% CI: 0.45 to 0.98]). The greater difference in late LPP amplitude between Reappraise-Negative and View-Negative from pre-test to post-test was due to a reduction in late LPP amplitude from pre-test to post-test during View-Negative trials ($t(3101) = 3.1$, $p = 0.002$, $d_{rm} = 0.44$ [95% CI: 0.16 to 0.72]). During Reappraise-Negative trials, there was no significant difference in late LPP amplitude from pre-test to post-test ($t(3146) = 0.7$, $p = 0.46$, $d_{rm} = 0.10$ [95% CI: -0.17 to 0.38]).

Results also revealed a significant Session $\times$ Time $\times$ ER Instruction interaction ($F(2, 3101) = 5.3$, $p = 0.003$, $f^2 = 0.08$, 95% CI: 0.00 to 0.23]). Post hoc decompositions of this interaction were conducted by creating Reappraise-Negative minus View-Negative difference scores to reflect the emotion regulation effect (Δ Reappraise: negative scores reflect a reduction in late LPP amplitude during Reappraise-Negative trials relative to View-Negative trials). View-

Negative minus View-Neutral difference scores were created to reflect emotional reactivity ($\Delta\text{View-Negative}$: positive scores reflect an increase in late LPP amplitude during View-Negative trials relative to View-Neutral trials). Analyses of the emotion regulation effect and emotional reactivity were conducted using separate Session $\times$ Time univariate repeated measures multi-level models with Participant and Session $\times$ Participant interaction entered as a random intercept. For the emotion regulation effect ($\Delta\text{Reappraise}$), results did not reveal a main effect of Session ($F(1, 59) = 0.5, p = 0.47, f^2 = 0.03$ [95% CI: 0.00 to 0.12]), but revealed a significant main effect of Time ($F(1, 986) = 13.1, p < 0.001, f^2 = 0.78$ [95% CI: 0.36 to 1.55]), such that across both sessions, participants exhibited significantly greater emotion regulation difficulty from pre-test ($M = 0.4, SD = 5.0$) to post-test ($M = 1.3, SD = 6.0; t(986) = 3.6, p < 0.001, d_{rm} = 0.67$ [95% CI: 0.31 to 1.03]). However, there was no significant interaction of Session $\times$ Time ($F(1, 986) = 2.3, p = 0.13, f^2 = 0.14$ [95% CI: 0.0 to 0.35]). For emotional reactivity ($\Delta\text{View-Negative}$), results did not reveal a main effect of Session ($F(1, 58) = 0.1, p = 0.7, f^2 = 0.01$ [95% CI: 0.0 to 0.04]) or Time ($F(1, 985) = 0.2, p = 0.64, f^2 = 0.01$ [95% CI: 0.0 to 0.06]). However, there was a significant Session $\times$ Time interaction ($F(1, 985) = 19.7, p < 0.001, f^2 = 0.94$ [95% CI: 0.48 to 1.84]), such that during the exercise session, participants exhibited a significant reduction in late LPP amplitude from pre-test ($M = 2.1, SD = 4.8$), to post-test ($M = 0.8, SD = 5.5; t(986) = 3.3, p < 0.001, d_{rm} = 0.72$ [95% CI: 0.29 to 1.14]). The opposite effect was observed during the sitting session, such that participants exhibited a significant increase in late LPP amplitude from pre-test ($M = 1.1, SD = 4.9$) to post-test ($M = 2.2, SD = 6.2; t(985) = 2.9, p = 0.003, d_{rm} = 0.48$ [95% CI: 0.16 to 0.80]). These results suggest that while participants exhibited difficulty regulating their emotions using cognitive reappraisal, participants experienced reduced emotional reactivity during View-Negative trials following the exercise session.

Self-Report Results

Self-Reported Emotional Arousal. Results revealed a significant main effect of ER Instruction ($F(2, 560) = 161.5, p = 0.001, f^2 = 1.66, [95\% \text{ CI: } 0.95 \text{ to } 3.18]$), such that overall, participants reported greater emotional arousal during reappraise-negative trials ($M = 4.2, SD = 1.5$) relative to view-negative trials ($M = 4.0, SD = 2.1; t(560) = 2.3, p = 0.04, d_{rm} = 0.17, [95\% \text{ CI: } 0.01 \text{ to } 0.33]$). However, this difference did not remain significant following false discovery rate control (Benjamini-Hochberg critical alpha = 0.039). Additionally, participants reported significantly greater emotional arousal during the reappraise-negative and view-negative trials relative to view-neutral ($ts > 16.5, ps < 0.001$). There was a significant effect of Session ($F(1, 53) = 10.2, p = 0.002, f^2 = 0.05, [95\% \text{ CI: } 0.00 \text{ to } 0.17]$), such that participants reported significantly lower emotional arousal during exercise sessions ($M = 3.4, SD = 1.6$) relative to control sessions ($M = 3.9, SD = 1.6; t(53) = 3.2, p = 0.002, d_{rm} = 0.35, [95\% \text{ CI: } 0.12 \text{ to } 0.57]$). There was also a significant main effect of Time ($F(1, 564) = 15.1, p < 0.001, f^2 = 0.08, [95\% \text{ CI: } 0.0 \text{ to } 0.23]$), such that across both sessions, self-reported emotional arousal decreased from pre-test ($M = 3.8, SD = 1.6$) to post-test ($M = 3.5, SD = 1.6; t(564) = 5.4, p < 0.001, d_{rm} = 0.27, [95\% \text{ CI: } 0.13 \text{ to } 0.41]$). Furthermore, results revealed a significant ER Instruction $\times$ Time interaction ($F(2, 560) = 4.5, p = 0.011, f^2 = 0.05 [95\% \text{ CI: } 0.0 \text{ to } 0.16]$). Post-hoc decomposition of the ER Instruction $\times$ Time interaction was conducted by examining the change from pre-test to post-test within each ER Instruction. Across both sessions, participants reported significant reductions in emotional arousal from pre-test to post-test for the reappraise-negative ($t(561) = 3.5, p < 0.001, d_{rm} = 0.31 [95\% \text{ CI: } 0.13 \text{ to } 0.49]$), and view-negative trials ($t(561) = 3.5, p < 0.001, d_{rm} = 0.29 [95\% \text{ CI: } 0.13 \text{ to } 0.45]$). Participants did not report a significant change in pre-

test to post-test arousal for the view-neutral trials ($t(561) = 0.2, p = 0.8, d_{rm} = 0.02$ [95% CI: -0.13 to 0.17]). All other interactions were not significant ($F_s < 1.2, p_s > 0.27$).

Self-Reported Emotion Regulation Effort. Results revealed a significant main effect of ER Instruction ($F(2, 560) = 372.0, p < 0.001, f^2 = 1.86$, [95% CI: 1.09 to 3.55]), such that overall, participants reported significantly greater effort during reappraise-negative trials ($M = 4.7, SD = 1.6$) relative to view-negative trials ($M = 3.8, SD = 1.6; t(560) = 8.8, p < 0.001, d_{rm} = 0.91$, [95% CI: 0.70 to 1.12]). Additionally, participants reported significantly greater effort during both reappraise-negative and view-negative trials relative to view-neutral trials ($t_s > 18.0, p_s < 0.001, d_{rms} > 2.39$). There was also a significant main effect of Time ($F(1, 564) = 8.9, p = 0.003, f^2 = 0.02$, [95% CI: 0.00 to 0.10]), such participants reported a significant decrease in effort during the ER Task from pre-test ($M = 3.6, SD = 1.9$) to post-test ($M = 3.3, SD = 1.9; t(564) = 3.0, p = 0.003, d_{rm} = 0.26$, [95% CI: 0.09 to 0.42]). There was no main effect of Session ($F(1, 55) = 2.6, p = 0.12, f^2 = 0.01$, [95% CI: 0.00 to 0.04]). However, results revealed a significant Session $\times$ Time interaction ($F(1, 564) = 10.3, p < 0.001, f^2 = 0.10$, [95% CI: 0.00 to 0.11]). Post-hoc decomposition of the Session $\times$ Time interaction was conducted by examining the main effect of Time across both sessions. For the exercise session, participants reported a significant reduction in effort across all trials during the ER Task from pre-test ($M = 3.6, SD = 1.9$) to post-test ($M = 3.1, SD = 1.8; t(563) = 4.3, p < 0.001, d_{rm} = 0.45$, [95% CI: 0.24 to 0.66]). For the sitting session, no significant difference in effort during the ER Task was observed between pre-test ($M = 3.64, SD = 1.9$) and post-test ($M = 3.6, SD = 1.9; t(565) = 0.3, p = 0.9, d_{rm} = 0.03$, [95% CI: -0.18 to 0.21]). All other interaction effects were not significant ($F_s < 2.2, p_s > 0.11$).

Mediation Analyses

A mediation analysis was conducted to examine whether exercise-induced changes in cognitive control mediated the effect of exercise on cognitive reappraisal success. I predicted that exercise would lead to a decrease (pre-test to post-test) in late LPP amplitude during reappraisal, and exercise-induced increases in the P300 would mediate this relationship. Given that I was only interested in this relationship during the exercise session, all analyses only included exercise. Additionally, given that I was only interested in examining whether increases in P300 mediated the relationship between exercise and reappraisal success, analyses only included the late LPP amplitude during reappraisal. Although the reappraisal effect is typically calculated using a reappraise minus view-negative difference score, as stated above, the significant reduction in late LPP during view-negative at post-test makes this approach less suitable for understanding the unique effects of exercise on reappraisal success in this sample. Therefore, the model consisted of the following steps:

Step 1: Mediator Model

To examine whether there was a significant change in P300 amplitude (mediator) from pre-test to post-test during the exercise session, the following linear multi-level model, with a random intercept for Participant, was conducted:

$$P300 \sim \text{Time} + (1 | \text{Participant})$$

Consistent with the previously presented results, the fixed effect of Time on P300 amplitude was not statistically significant (path a: $b = -0.52$, $SE = 0.31$, $t(143.79) = -1.66$, $p = 0.099$).

Step 2: Outcome Model

The effect of the mediator (P300 amplitude) on late LPP amplitude, controlling for time, was examined using the following linear multi-level model, with a random intercept for participant:

$$\text{Late LPP} \sim \text{Time} + \text{P300} + (1 | \text{Participant})$$

Results revealed that the fixed effect of P300 amplitude (controlling for Time) on late LPP amplitude was statistically significant (path b: $b = -0.16$, $SE = 0.08$, $t(187.50) = -1.98$, $p = 0.049$). This indicates that greater P300 amplitudes were associated with lower late LPP amplitudes.

Direct and Indirect Effects

To examine the indirect and direct effects of exercise on the change in late LPP amplitude from pre-test to post-test, I conducted a bootstrapping procedure with 1000 resamples. Results revealed that the indirect effect of Time on the late LPP via P300 was $b = 0.09$ with a bias of -0.01 and a standard error of 0.08 . The 95% bias-corrected bootstrap confidence interval for the indirect effect was $[-0.04, 0.29]$. This confidence interval included zero, indicating that the indirect effect was not statistically significant. The direct effect of Time on LPP (controlling for P300) was $b = -0.19$, with a bias of 0.02 and a standard error of 0.45 . The 95% bias-corrected bootstrap confidence interval of the direct effect was $[-1.08, 0.74]$. This confidence interval included zero, indicating that the direct effect was not statistically significant. Lastly, the total effect of Time on LPP was $b = -.10$, with a bias of 0.01 and a standard error of 0.46 . The 95% bias-corrected bootstrap confidence interval of the total effect was $[-1.05, 0.82]$. The total effect was also not significant, as the confidence interval included zero.

DISCUSSION

The current study investigated the acute effects of aerobic exercise on cognitive control and emotion regulation in individuals with clinically significant PTSD symptoms. This study employed a multimodal experimental approach, leveraging neurophysiological, behavioral, and self-report measures to examine the acute effects of aerobic exercise. I hypothesized that an acute bout of aerobic exercise would lead to 1) enhanced cognitive control, evidenced by an increased P300, decreased ERN, and improved behavioral performance (accuracy and reaction time) on the flanker task; and 2) improved emotion regulation, evidenced by reduced self-reported and neural indices (LPP) of emotional reactivity. Furthermore, I hypothesized that the effect of exercise on emotional reactivity during reappraisal (LPP) would be mediated by exercise-induced increases in cognitive control (P300). The findings of this study partially supported my hypotheses. To contextualize the overall findings, I first discuss the specific effects of exercise on cognitive control, followed by its effects on emotion regulation and potential mediating mechanisms.

Effect of exercise on cognitive control

Contrary to my hypotheses, the exercise session did not elicit a significant change in P300 amplitude relative to the control (sitting) session. This null result was surprising, as it does not align with extant literature demonstrating a robust effect of aerobic exercise on the P300 (Chang et al., 2017; Kao et al., 2020; Pontifex et al., 2015, 2021). Indeed, a systematic review of studies examining the effect of exercise on the P300 found that exercise increased the P300 amplitude across multiple cognitive tasks, such as the flanker (Ludyga et al., 2017; Pontifex et al., 2013, 2021) and Odd-ball tasks (Kao et al., 2018; for review, see Gusatovic et al., 2022; Kao et al., 2020). However, some studies have found null results (Popovich & Staines, 2015; Stroth et

al., 2009; Zhou & Qin, 2019). Moreover, the vast majority of studies examined the effect of exercise on the P300 in healthy populations, and no studies to date have examined the effect of exercise on the P300 in a PTSD population. Therefore, the unexpected results may be due to the sample characteristics of this study.

This study recruited a sample of trauma-exposed individuals with clinically significant PTSD symptoms. PTSD is associated with impaired cognition (Aupperle et al., 2012; Bomyea et al., 2012). Importantly, cognitive impairment in PTSD has also been demonstrated in various ERP studies (Araki et al., 2005), as greater PTSD symptomology has been shown to be associated with an attenuated P300 to neutral visual stimuli (see Javanbakht et al., 2011 for review). Cognitive control impairment in PTSD is thought to be a function of difficulty disengaging from distracting stimuli in order to attend to more task-relevant information (Aupperle et al., 2012). Difficulty disengaging from distracting stimuli is commonly observed in re-experiencing symptoms for individuals with PTSD. Re-experiencing symptoms – described as repeated, intrusive, and unwanted thoughts (e.g., nightmares, flashbacks) – can be distressing for individuals with PTSD and lead to significant impairment in socio-emotional functioning (American Psychiatric Association, 2022). Due to the difficulty disengaging from these intrusive thoughts, individuals may struggle to focus on work, school, or life-related tasks. Given that individuals with PTSD often exhibit cognitive control impairment, the acute bout of aerobic exercise used in this study may not have been sufficient to modulate the P300 in a cognitively impaired population.

Importantly, this study did not replicate findings from the study from which its methodology was based (Pontifex et al., 2021), which assessed the effects of an acute bout of exercise on the P300 in high and low-anxious college students. Pontifex and colleagues (2021)

found that for both high and low-anxious students, the P300 amplitude significantly increased from pre-test to post-test following exercise, with a greater increase in the low-anxious group. Given that this study shared an identical exercise and flanker task protocol, the differing results provide additional evidence that the null effects found in this study may be due to the sample characteristics. Specifically, these differing results suggest that the effect of cognitive control processes may differ between PTSD and anxiety.

In addition to the P300, I also explored the effects of exercise on the error-related negativity (ERN) as a separate neural measure of cognitive control. Results mirrored the P300 findings, such that no significant change in ERN or Δ ERN was observed from pre-test to post-test across both the exercise and sitting sessions. These results were unexpected, as they also do not align with extant research on the effects of exercise and the ERN. While the literature on the effects of exercise on the ERN is not as robust as research on the P300, there is evidence suggesting that exercise modulates ERN amplitudes (Drollette et al., 2025; Pontifex et al., 2013, 2021). Indeed, a smaller ERN amplitude has been observed following an acute bout of moderate to vigorous exercise (Drollette et al., 2025). However, many of these studies were conducted in child samples (Drollette et al., 2025; Pontifex et al., 2013), and none were conducted in PTSD samples. Studies examining the effects of exercise on the ERN in clinical populations are limited and inconsistent. For instance, acute aerobic exercise has been shown to increase ERN amplitude for high- and low-anxious individuals (Pontifex et al., 2021). In contrast, an 8-week moderate-intensity aerobic exercise intervention did not modulate the ERN in depressed adults (Brush et al., 2022). Overall, there remains considerable variability in research on the effect of exercise on the ERN. These limited and inconsistent findings highlight our incomplete understanding of the mechanisms by which exercise may modulate the ERN in clinical populations.

Notably, while exercise did not modulate the P300 or ERN in this study, participants exhibited the predicted improvement in behavioral performance, as evidenced by greater overall accuracy across both sessions and a greater decrease in reaction time from pre-test to post-test in the exercise session relative to the sitting session. These behavioral results suggest that participants exhibited exercise-induced gains in behavioral performance on the flanker task without recruiting an increase in cognitive attentional resources elicited by the P300 or ERN. Overall, these findings suggest that exercise may potentially enhance behavioral performance in PTSD through neural mechanisms not captured by the P300 and ERN.

Effect of Exercise on Emotion Regulation

Consistent with previous literature on emotion dysregulation in PTSD (Cisler et al., 2010; Seligowski et al., 2015; Woodward et al., 2015), participants in this study exhibited impaired emotion regulation. This was evidenced by larger early and late LPP amplitudes during Reappraisal trials compared to View-Negative trials across all sessions and time points. This pattern aligns with findings suggesting that cognitively demanding emotion regulation strategies, such as reappraisal, can be difficult to implement (Strauss et al., 2016) and may be particularly challenging for individuals with PTSD (Ehring & Quack, 2010; Woodward et al., 2015).

Contrary to my hypothesis, exercise did not enhance emotion regulation, as participants exhibited increased early and late LPP amplitudes (Δ Reappraise: reappraise minus view-negative) from pre-test to post-test. However, this increase was primarily driven by a significant reduction in LPP amplitude during the view-negative trials. Given that the reappraisal effect is typically measured relative to the view-negative trials, a lower LPP amplitude during view-negative can artificially inflate the Δ Reappraise LPP, potentially giving the false impression of increased emotional arousal from pre-test to post-test during reappraisal. Indeed, when

reappraisal was examined independently, participants did not exhibit a significant change in LPP amplitude from the pre-test to the post-test. Nonetheless, the results did not align with my prediction that aerobic exercise would enhance cognitive reappraisal.

Notably, although participants exhibited difficulty using reappraisal to regulate their emotions, exercise reduced the LPP amplitude from pre-test to post-test during Δ View-Negative trials. This reduction in LPP amplitude was not observed during the sitting sessions, which suggests that aerobic exercise may have led to reduced emotional reactivity in individuals with PTSD. This finding is particularly relevant given that hyperarousal, characterized by heightened emotional reactivity, is a core symptom of PTSD (American Psychiatric Association, 2022), and has been observed across neuroimaging studies (Etkin & Wager, 2007). Thus, the observed reduction in emotional reactivity following exercise demonstrates the potential of exercise as a method for alleviating hyperarousal symptoms in this population.

Given that exercise reduced LPP during Δ View-Negative trials from pre-test to post-test, the absence of LPP modulation during Δ Reappraise trials was somewhat surprising. It is expected that the physiological effects of exercise would be consistent across all ER instruction trials. The null results observed during reappraisal further underscore the specific challenges individuals with PTSD may face when employing cognitive emotion regulation strategies such as reappraisal. Successful cognitive reappraisal involves both engaging with negative stimuli and reframing their emotional context to reduce their emotional impact (Gross, 2002; Gross & John, 2003). Given that PTSD is associated with an attentional bias toward negative and threatening stimuli (Fani et al., 2012; Veerapa et al., 2023), it is possible that elevated LPP observed during reappraisal (versus view-negative) reflects an over-engagement with negative, highly arousing stimuli – without sufficient cognitive resources available to reduce their emotional response.

While an acute bout of aerobic exercise may result in immediate reductions in emotional reactivity, it is likely that long-term exercise intervention with multiple bouts may be needed to elicit sustained improvements in emotion regulation in this sample.

The self-report findings of this study differed from neural findings. Specifically, across both sessions, participants reported significantly lower emotional arousal during Reappraisal relative to View-Negative trials, suggesting perceived emotion regulation. Furthermore, participants reported significantly lower self-reported emotional arousal during the exercise sessions relative to the sitting sessions. However, although both experimental sessions experienced significant reductions in emotional arousal from pre-test to post-test, these effects did not differ by experimental session. Notably, exercise was associated with decreased emotion regulation effort, such that during exercise sessions, participants reported a significant pre-test to post-test reduction in self-reported effort during the ER task. Furthermore, this effect was primarily driven by a decrease in perceived effort during reappraisal. These results provide further contextual understanding of how exercise may affect emotion regulation processes. Specifically, while it is unclear whether acute aerobic exercise may have an immediate effect on emotion regulation success in individuals with PTSD, participants found reappraisal to be less effortful following exercise. Given that cognitive reappraisal is a cognitively demanding emotion regulation strategy that is difficult to implement across multiple clinical populations (Aldao et al., 2010; Bartolomeo et al., 2020; Cisler et al., 2010; Pencea et al., 2020), multiple bouts of exercise may be needed before participants experience significant improvements in emotion regulation success. The reduction in perceived emotion regulation effort following exercise suggests that exercise can modulate mechanisms that interfere with emotion regulation success.

Taken together, these findings suggest that while an acute bout of aerobic exercise may not immediately improve the effectiveness of cognitive emotion regulation strategies such as reappraisal in individuals with PTSD, exercise may still provide meaningful benefits by reducing emotional reactivity and perceived effort when regulating emotions. These results are promising, as they provide an initial step in understanding the neural and subjective mechanisms by which exercise can enhance emotion regulation success in this population. Moreover, the current study highlights the complexity of emotion regulation processes in PTSD and the need for long-term interventions that assess the cumulative effects of aerobic exercise on emotion regulation processes.

Mediation Effects

Lastly, I aimed to assess whether the effect of exercise on emotion regulation was mediated by exercise-induced enhancement in cognitive control. Overall, the results suggest that exercise-induced changes in the P300 do not mediate the effect of exercise on the LPP during reappraisal. However, given that exercise did not modulate the P300 or late LPP, results should be interpreted with caution. The significant relationship between the P300 and late LPP (path b) is promising, as it suggests that greater cognitive control is associated with better emotion regulation success in PTSD. Therefore, further research should be conducted on strategies for enhancing cognitive control in PTSD populations.

Clinical Implications

Despite mixed findings, there are several clinical implications that should be noted. While this study did not fully support exercise as a method for improving cognitive control and cognitive reappraisal in individuals with PTSD, the effect of exercise on decreased emotional

reactivity and perceived emotion regulation effort highlights the potential utility of exercise in PTSD treatment.

There is a growing literature on exercise interventions for PTSD symptoms (Björkman & Ekblom, 2022; Speer et al., 2020; Whitworth et al., 2019; Whitworth & Ciccolo, 2016). Furthermore, researchers have begun to integrate exercise into pre-existing interventions in order to capitalize on the benefits of exercise as an augmented intervention (Bryant et al., 2023; Powers, Medina, et al., 2015). Augmented interventions offer the advantage of targeting specific mechanisms that increase the likelihood of treatment response (Bryant et al., 2023; Powers, Medina, et al., 2015). Recent studies such as Bryant and colleagues (2023) and Powers, Medina, and colleagues (2015) have focused on the brain-derived neurotrophic factor (BDNF) as the primary mechanism by which exercise improves PE outcomes. BDNF is a protein that plays a crucial role in the brain's neuroplasticity, which is important for learning and memory (Miranda et al., 2019). Researchers have posited that BDNF is a key mechanism for extinction in exposure-based therapies due to BDNF's influence on cognitive processes such as long-term memory and learning (Lu et al., 2008; Powers, Medina, et al., 2015). Aerobic exercise has been shown to reliably increase BDNF levels (Greenwood et al., 2009), highlighting the potential benefits of exercise as an augmented treatment for PTSD – particularly as a method for enhancing neural mechanisms.

The results of this study provide further support for additional research on exercise as a method for enhancing important mechanisms in psychological treatments for PTSD. Emotion regulation is a common treatment target in PTSD treatments, and is associated with greater treatment outcomes (American Psychological Association, 2017; Pencea et al., 2020). Furthermore, greater emotion regulation ability is associated with a decreased likelihood of

developing PTSD following trauma exposure (J. M. Fitzgerald et al., 2018; Pencea et al., 2020). Therefore, exercise may help facilitate greater emotion regulation success in psychological treatment by decreasing the perceived cognitive effort of engaging in emotion regulation.

Taken together, insights generated by this investigation contribute to our understanding of the neural mechanisms by which exercise may help facilitate improved treatment outcomes in individuals with PTSD and inform future research on the integration of exercise into common psychological treatments for PTSD.

Limitations and Future Directions

Although this study offers valuable insights into the understanding of the cognitive and affective effects of exercise in individuals with PTSD, several limitations should be considered when interpreting the results of this study.

Missing Data

First, similar to many studies with multiple observations, the study had missing data. Specifically, 14 participants were lost to follow-up, as they did not attend their second session. Among the 53 participants who completed all sessions, 2 participants were excluded due to unusable EEG data across both sessions, and 16 participants had missing data on at least one task due to poor data quality. A significant strength of the mixed-effects statistical approach used in this study is its ability to include participants with missing data. Mixed-effects models rely on restricted maximum likelihood (REML) to estimate model parameters using all available data (Muhammad, 2023). Unlike traditional methods such as repeated measures ANOVA, which requires complete data for each participant, mixed-effects models do not discard participants due to missing observations. A mixed-effects model is advantageous because it allows for greater data retention and preserves statistical power in studies involving repeated observations.

However, it is important to acknowledge that with robust statistical methods, missing data can still potentially introduce bias into the results.

PTSD Symptoms

It is important to note that the sample was not formally diagnosed with PTSD. Instead, participants were screened for clinically significant PTSD symptoms using the PCL-5 (Weathers, Litz, et al., 2013). The PCL-5 is a well-validated measure commonly used to screen for PTSD in research and clinical settings (Blevins et al., 2015; Weathers, Litz, et al., 2013). However, given that a structured clinical interview is needed to formally diagnose an individual with PTSD (American Psychological Association, 2017; Weathers et al., 2018), I cannot confirm that all participants met full criteria for PTSD. Additionally, some participants reported lower PTSD scores during the date of participation relative to the date of screening. This is due to the time that passed from screening to enrollment, as eligible participants were allowed to choose any research timeslot within the same semester in which they were screened as eligible for participation. Therefore, participants' PTSD scores may have improved either through receiving psychotherapy or the passage of time. Unfortunately, I did not collect data on participant treatment history. Future research would benefit from investigating the effects of exercise in a population formally diagnosed with PTSD and not currently receiving psychotherapy.

Age range

The study population was limited to college-aged students between the ages of 18 and 34. Given that this sample was limited to a student population, caution should be taken when projecting these results to older samples. Research has shown developmental differences in cognitive control. Research on the developmental differences in the P300 suggests that while the P300 amplitude reaches its maximum at the age of 21, optimal cognitive performance may occur

at the age of 30 and declines in older adults (van Dinteren et al., 2014). Additionally, the effect of exercise on the P300 during the flanker task has been shown to be greater in younger adults (ages 19-25) relative to older adults (ages 60-70; Kamiyo et al., 2009). There are also developmental differences in PTSD, such that younger adults (ages 20-34) have a greater prevalence of PTSD and greater PTSD symptomology relative to middle-aged (ages 35-64) and older (age 65+) adults (Reynolds et al., 2016). The majority of research on the effects of exercise on cognitive control has been conducted in young adults (ages 18-34 years; see Pontifex et al., 2019). Therefore, an age range of 18-34 would be consistent with previous research and would help control for any confounding effects of age. Future studies should assess the effects of exercise on a wider age range.

Acute Exercise

The findings from this study did not fully support my hypothesis that an acute bout of exercise would enhance cognitive control and emotion regulation in individuals with PTSD. Given that I only assessed the short-term effects of exercise, additional research is needed to examine the potential long-term effects of exercise on cognitive control and emotion regulation. Prior work suggests that the benefits of acute exercise are often transient (Crush & Loprinzi, 2017), highlighting the need for regular physical activity to achieve sustained improvements. Although research on long-term exercise interventions is needed, understanding the short-term effects of acute exercise remains valuable. For instance, acute exercise may serve as an adjunct to psychotherapy. Engaging in exercise immediately prior to a therapy session may enhance cognitive flexibility and reduce emotional reactivity, potentially optimizing the treatment. In sum, research is needed to further understand the efficacy of acute and long-term exercise interventions for PTSD.

Exercise Intensity

This study examined the effects of moderate-intensity aerobic exercise on cognitive control and emotion regulation. I selected this intensity, as moderate-intensity exercise is easier to sustain for extended periods – thus increasing accessibility. Additionally, research has demonstrated that acute bouts of moderate-intensity exercise can have immediate effects on cognitive control (Kumar et al., 2012; Ludyga et al., 2016; Pontifex et al., 2021) and emotion regulation (Bernstein & McNally, 2017; Ligeza et al., 2023). However, given that moderate-intensity exercise did not significantly affect these outcomes in the current study, future research should consider the differential effects of exercise intensity on cognitive control and emotion regulation in PTSD. Some research has suggested that the effects of exercise on PTSD symptoms are greatest with multiple bouts of vigorous exercise (Whitworth et al., 2017; Whitworth & Ciccolo, 2016). However, these studies examined the effects of vigorous strength training on PTSD as opposed to vigorous-intensity aerobic exercise. Nonetheless, additional research should be conducted to assess whether exercise intensity moderates the effect of exercise on cognitive control and emotion regulation in PTSD populations.

Conclusion

In summary, the present study offers insights into the acute effects of aerobic exercise on cognitive control and emotion regulation processes in individuals with clinically significant PTSD symptoms. While findings did not fully support my prediction that exercise would enhance cognitive control and emotion regulation, participants did demonstrate reduced emotional reactivity and perceived emotion regulation effort following exercise. These findings highlight the potential for exercise to enhance important affective processes implicated in successful emotion regulation in individuals with PTSD. In conjunction with traditional

psychotherapies, exercise may serve as a promising adjunctive strategy for targeting emotion hyperarousal symptoms and improving emotion regulation success during treatment. Future research is still needed to understand the cumulative effects of repeated bouts of aerobic exercise and to further investigate the mechanisms by which exercise can improve treatment outcomes in PTSD.

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APPENDIX A: TABLES

Table 1. Mean (SD) values for demographics

Measure	
N	65
Age (years)	19.52 (1.83)
Race (%)	
Asian	10.8%
Black/African American	12.3%
Hispanic/Latine	4.6%
White	61.5%
Multi-Racial	10.8%
PCL-5 Score	42.88 (11.12)

Table 2. Mean (SD) values of the experimental manipulation checks by session

Measure	Exercise	Sitting
Treadmill speed	2.7 (0.4)	NA
Treadmill incline	2.2 (0.7)	NA
HR (bpm)	151.1 (8.9)	80.4 (19.5)
OMNI Perceived Exertion	3.8 (1.5)	0.8 (1.2)
Feeling Scale	2.4 (1.2)	2.3 (1.6)

Table 3. Mean (SD) values of event-related potentials by session and time

Measure	Exercise		Sitting	
	Pre	Post	Pre	Post
P300				
Incongruent Trials	6.9 (3.5)	6.4 (4.6)	7.0 (3.5)	6.8 (4.2)
Congruent Trials	5.8 (3.5)	5.0 (4.5)	5.7 (3.3)	5.6 (4.2)
ERN				
Error Trials	-4.7 (6.0)	-4.4 (5.9)	-4.6 (5.6)	-3.8 (5.0)
Correct Trials	1.0 (5.6)	1.2 (6.7)	1.4 (6.6)	1.3 (6.1)
Early LPP (400-1000ms)				
Reappraise Negative	4.5 (5.4)	4.6 (4.8)	4.9 (5.1)	5.2 (4.8)
View Negative	4.9 (5.4)	3.7 (5.2)	3.5 (4.7)	4.6 (4.6)
View Neutral	1.6 (4.4)	1.2 (4.2)	0.8 (4.1)	1.1 (4.4)
Late LPP (1-6s)				
Reappraise Negative	2.2 (4.2)	2.0 (4.6)	2.2 (4.5)	2.6 (5.7)
View Negative	2.3 (4.6)	0.6 (4.3)	1.4 (4.0)	1.3 (4.0)
View Neutral	0.2 (3.9)	-0.2 (3.9)	0.3 (3.6)	-0.9 (5.1)

Note. Values are measured in microvolts (μV). The P300 amplitude was extracted from electrode site PZ and time-locked to stimulus onset. The ERN Amplitude was extracted from electrode site FCz and time-locked to the response. The early (400-1000ms) and late (1-6s) LPP were extracted from electrode site CPz and time locked to stimulus onset.

Table 4. Mean (SD) values of flanker task behavioral performance and emotion regulation task self-report results by session and time

Measure	Exercise		Sitting	
	Pre	Post	Pre	Post
Flanker Task				
Accuracy (%)	90.5% (5.6)	91.0% (5.2)	90.1% (8.0)	90.6% (6.3)
Mean RT (ms)	382.0 (66.2)	372.8 (64.8)	382.0 (64.7)	383.9 (61.2)
ER Task – Arousal				
Reappraise Negative	4.2 (1.6)	3.7 (1.4)	4.7 (1.3)	4.2 (1.5)
View Negative	4.1 (1.4)	3.4 (1.5)	4.4 (1.3)	4.1 (1.6)
View Neutral	2.6 (1.4)	2.4 (1.2)	2.8 (1.3)	2.9 (1.4)
ER Task – Effort				
Reappraise Negative	5.0 (1.4)	4.1 (1.6)	4.9 (1.6)	4.8 (1.5)
View Negative	3.8 (1.6)	3.4 (1.6)	3.9 (1.46)	4.0 (1.8)
View Neutral	2.1 (1.6)	1.8 (1.2)	1.9 (1.4)	1.9 (1.2)

APPENDIX B: FIGURES

Figure 1. Consort Flow Chart

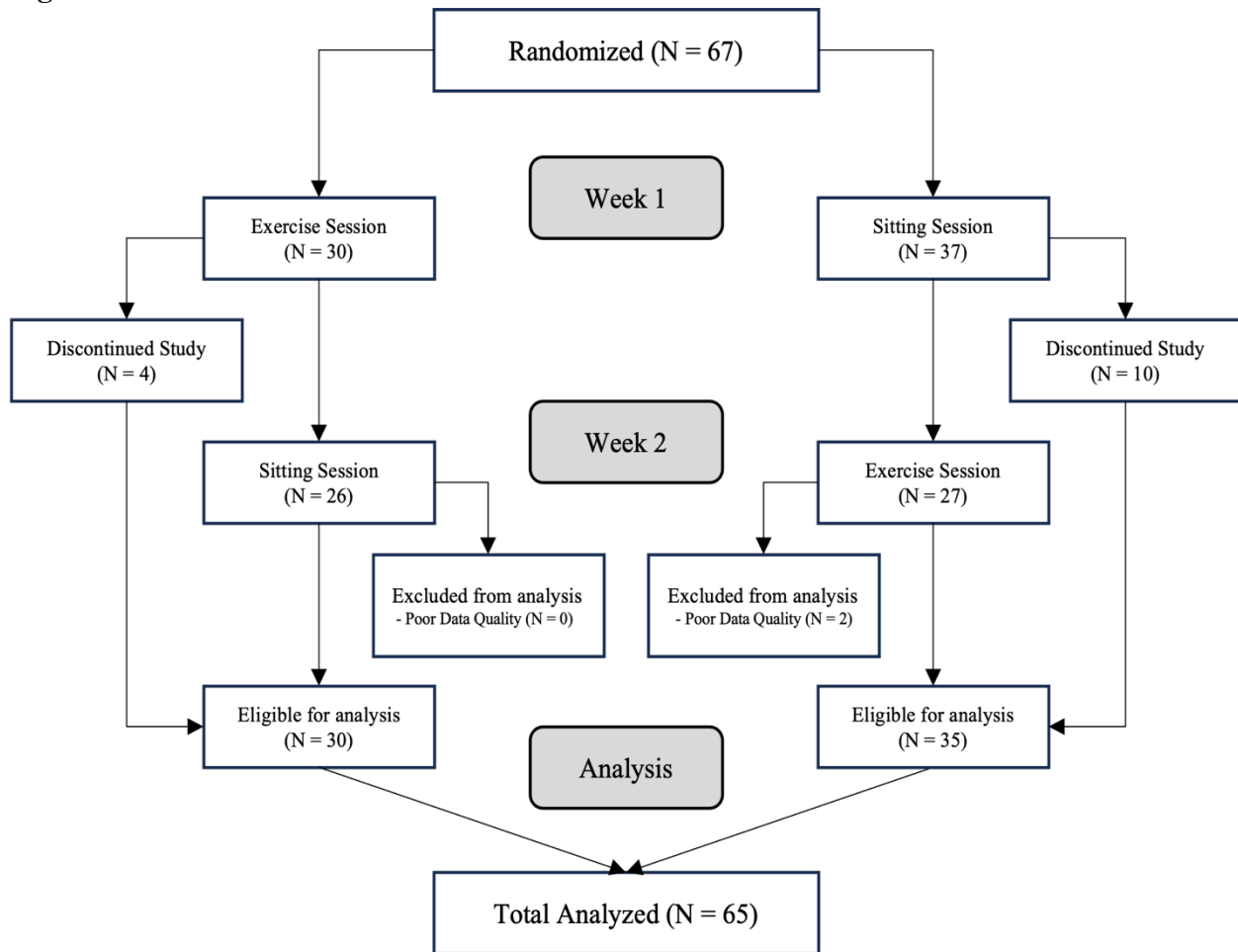


Figure 2. Study Design

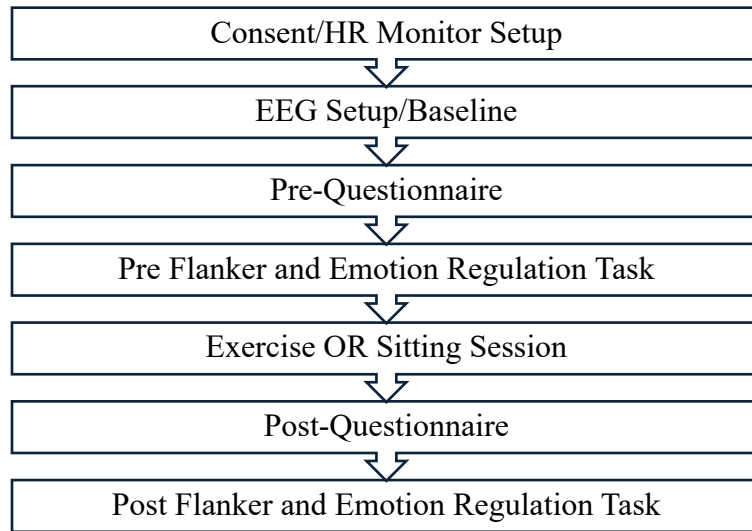


Figure 3. Emotion Regulation Picture-Viewing Task Trial Sequence

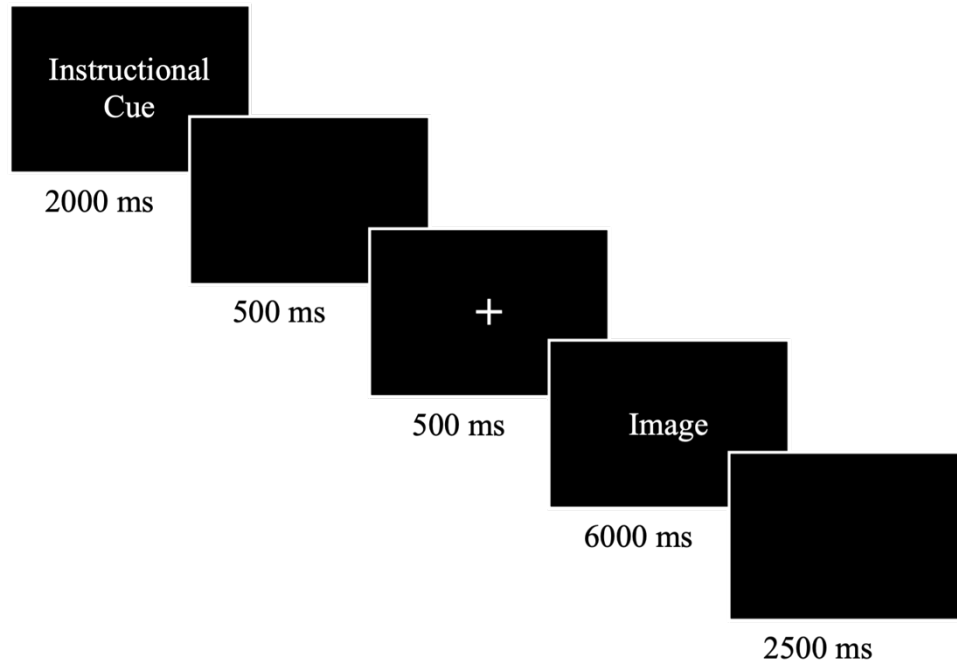
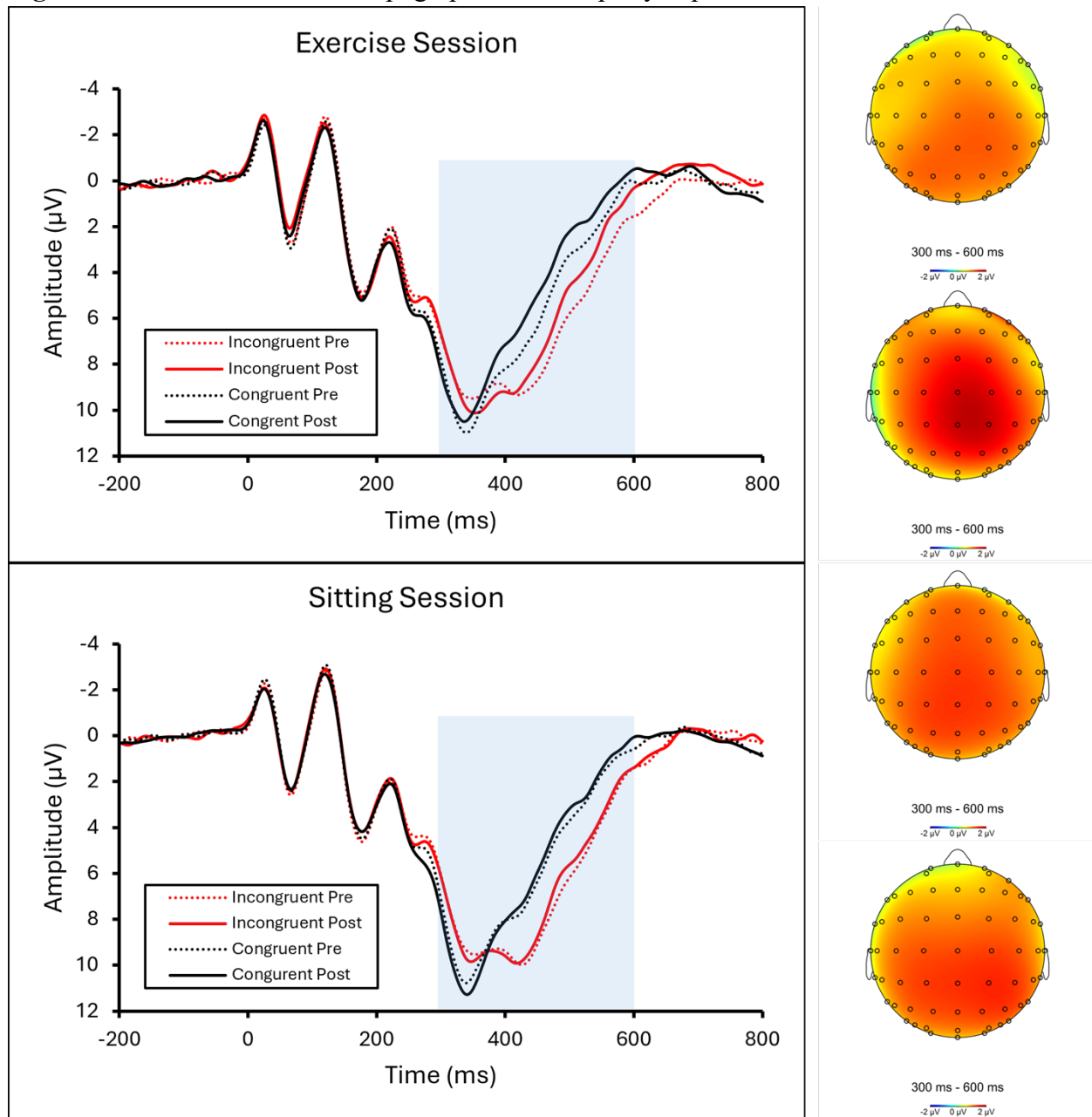
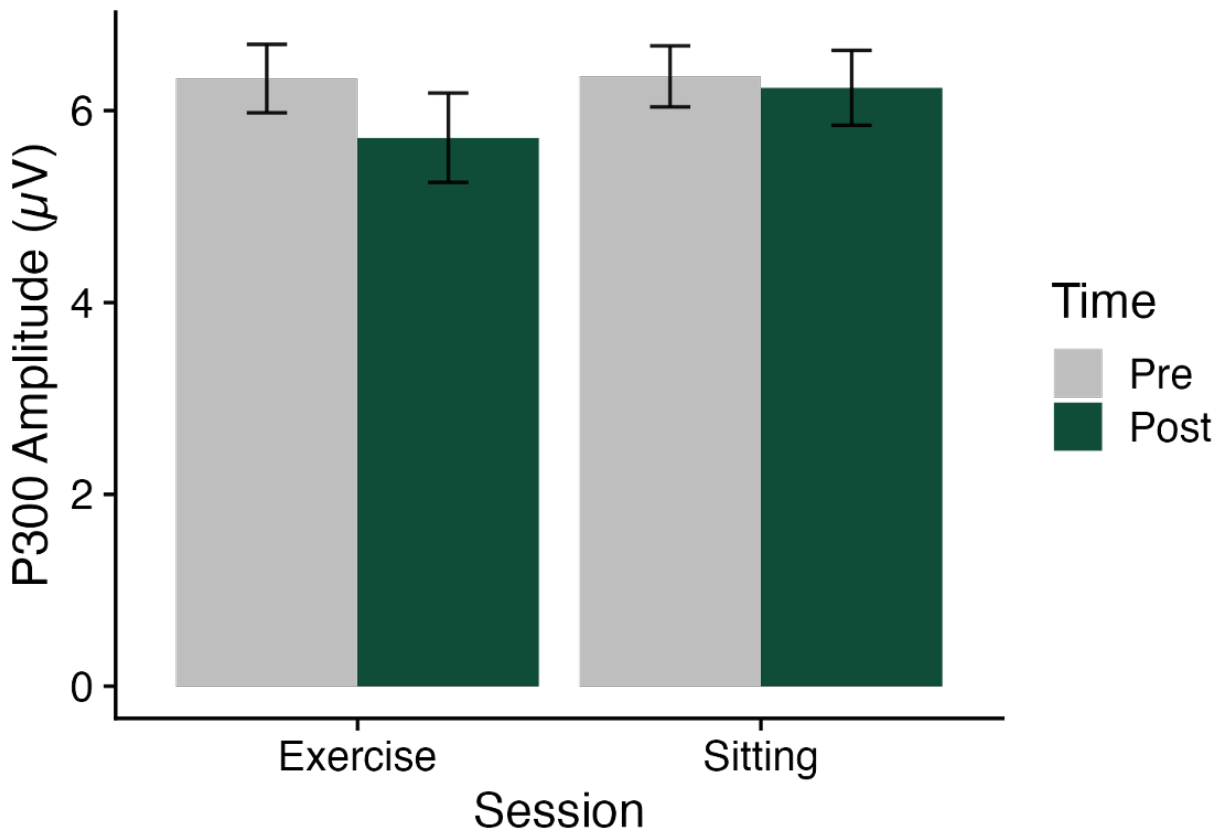


Figure 4. P300 waveforms and topographic head maps by experimental session and time



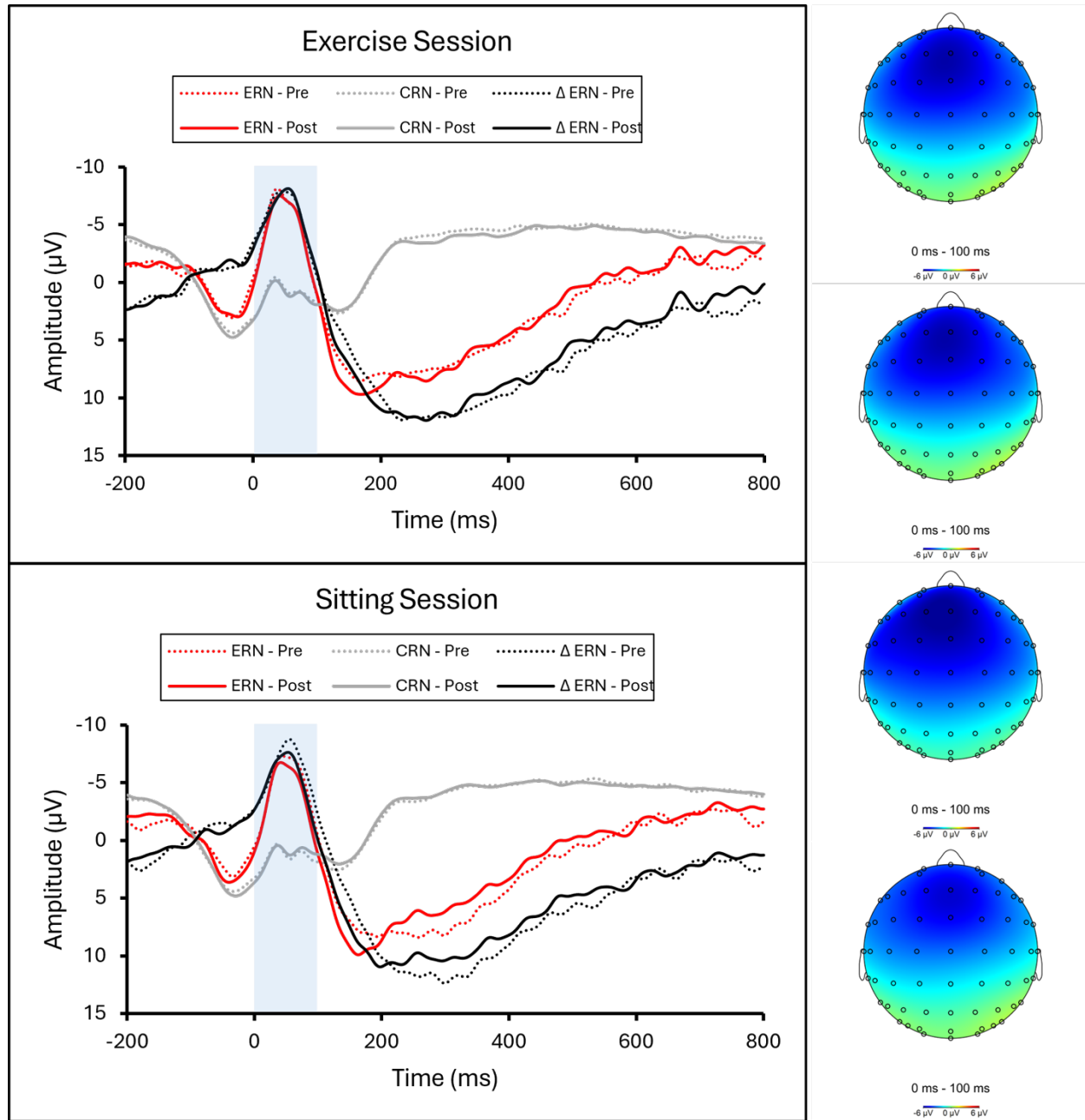
Note. Grand average stimulus-locked waveforms depicting the P300 during the flanker task before and after the exercise (top left) and sitting (bottom left) sessions. The P300 was extracted at the Pz electrode site, as the mean amplitude 300-600 ms post-stimulus onset. Topographic Head maps depicting the difference in amplitude between incongruent and congruent trials across the 300-600 ms time windows by session and time (right). Within each session, the top head map reflects the P300 at pre-test, and the bottom reflects post-test.

Figure 5. Bar Graph of P300 amplitude by experimental session and time



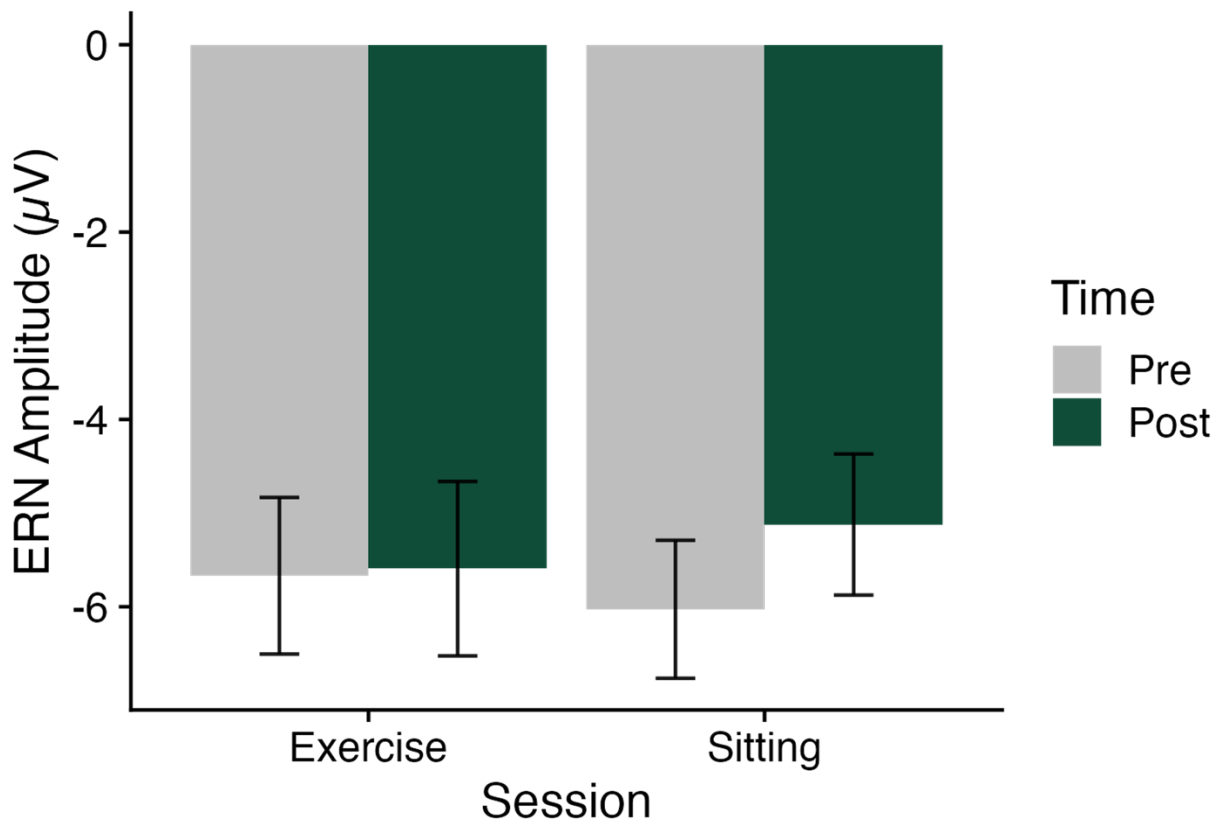
Note. Bar graph depicting the mean P300 amplitude (μV) within each session and time point. The P300 was extracted at the Pz electrode site, as the mean amplitude 300-600ms post-stimulus onset.

Figure 6. ERN waveforms and topographic head maps by experimental session and time



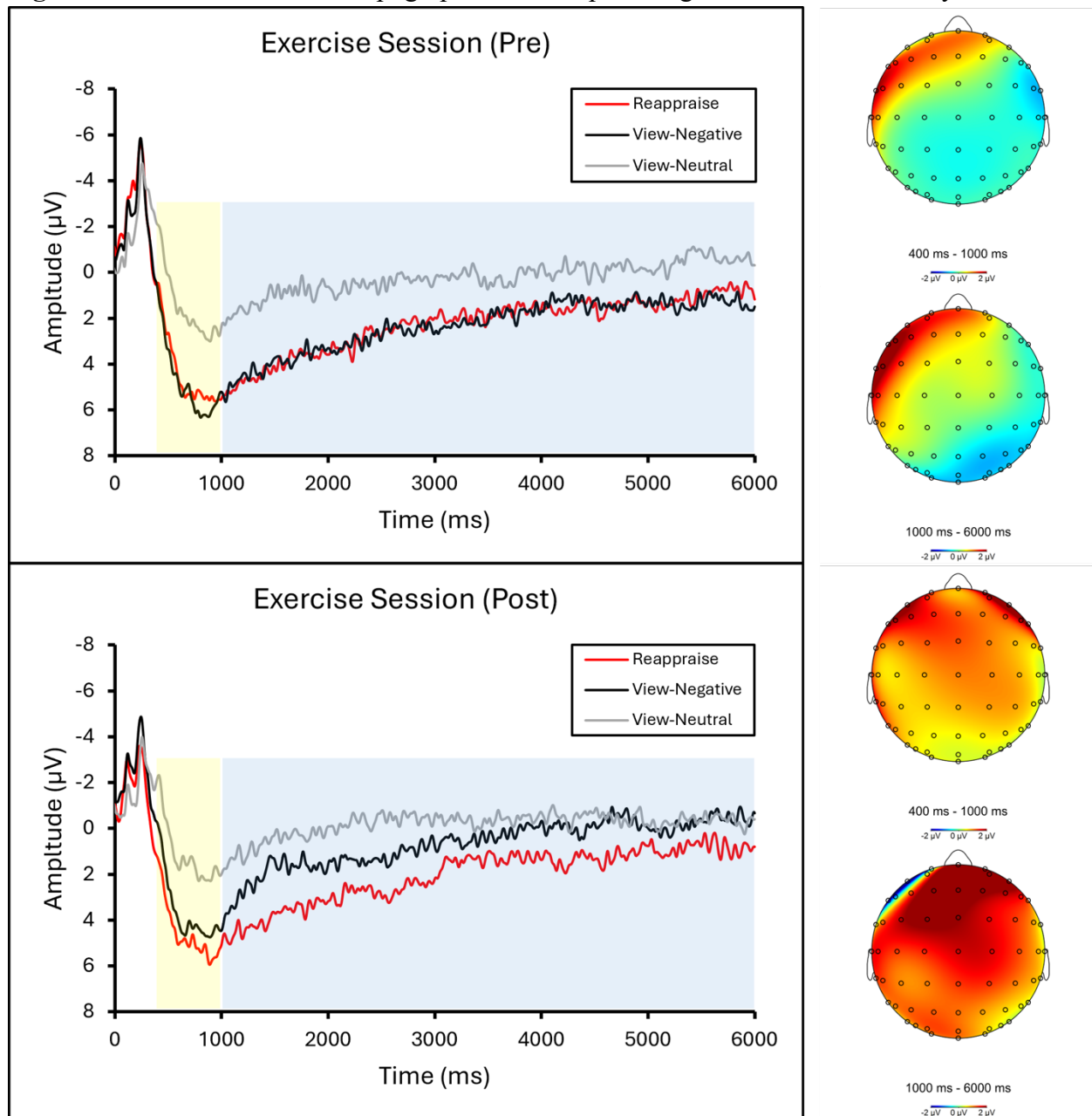
Note. Grand average response-locked waveforms depicting the ERN during the flanker task before and after the exercise (top left) and sitting (bottom left) sessions. The ERN was extracted at the FCz electrode site, as the mean amplitude 0-100 ms post-response. The ΔERN (black lines) was computed as the difference in amplitude between error (red lines) and correct (grey lines) trials. Topographic head maps depicting the ΔERN across the 0-100 ms time window by session and time (right). Within each session, the top head map reflects the ΔERN at pre-test, and the bottom reflects post-test.

Figure 7. Bar Graph of Δ ERN amplitude by experimental session and time



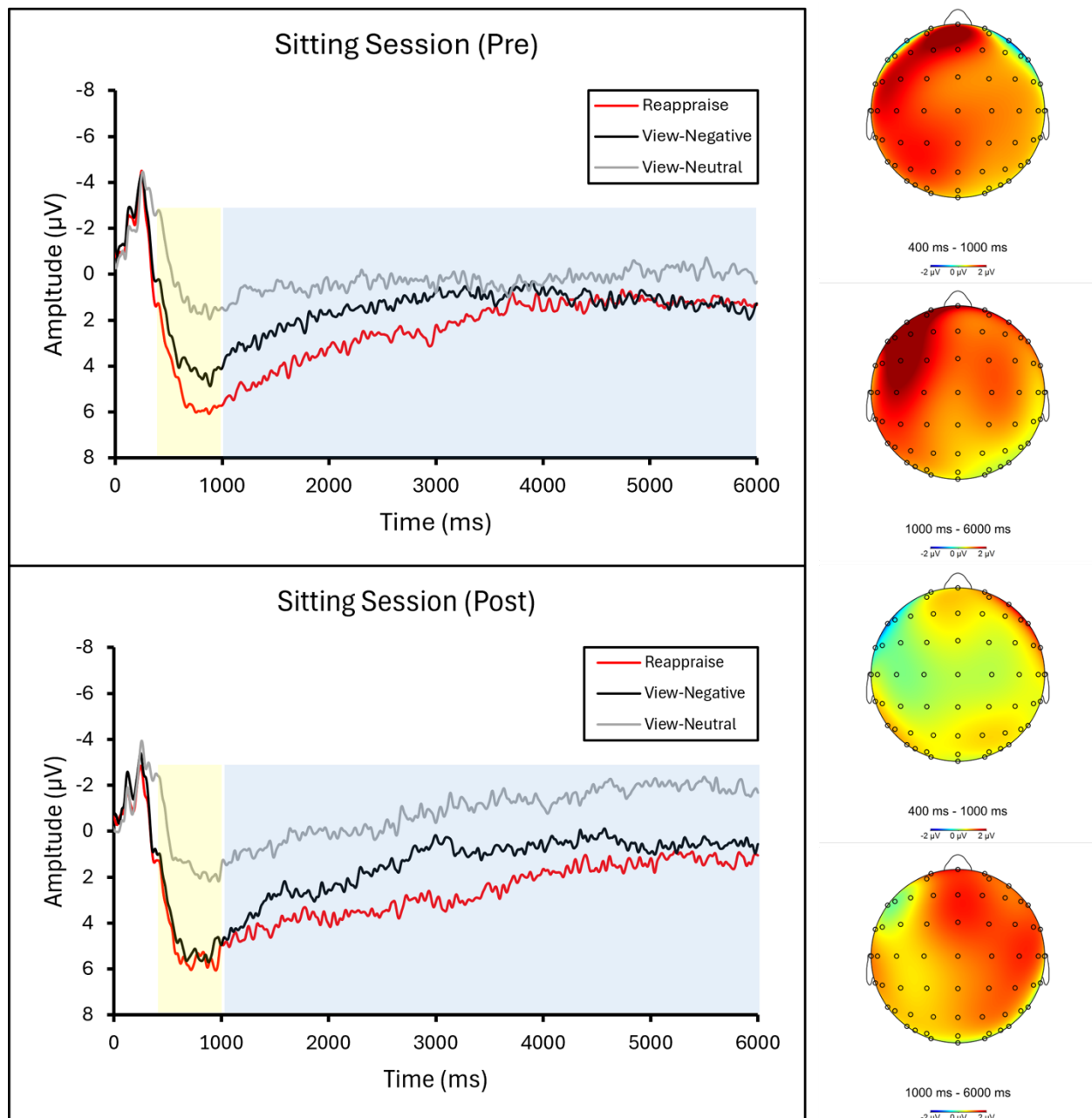
Note. Bar graph depicting the Δ ERN (error minus correct difference) amplitude (μV) within each session and time point. The Δ ERN was extracted at the FCz electrode site, as the mean amplitude 0-100ms post-response.

Figure 8. LPP waveforms and topographic head maps during the exercise session by time



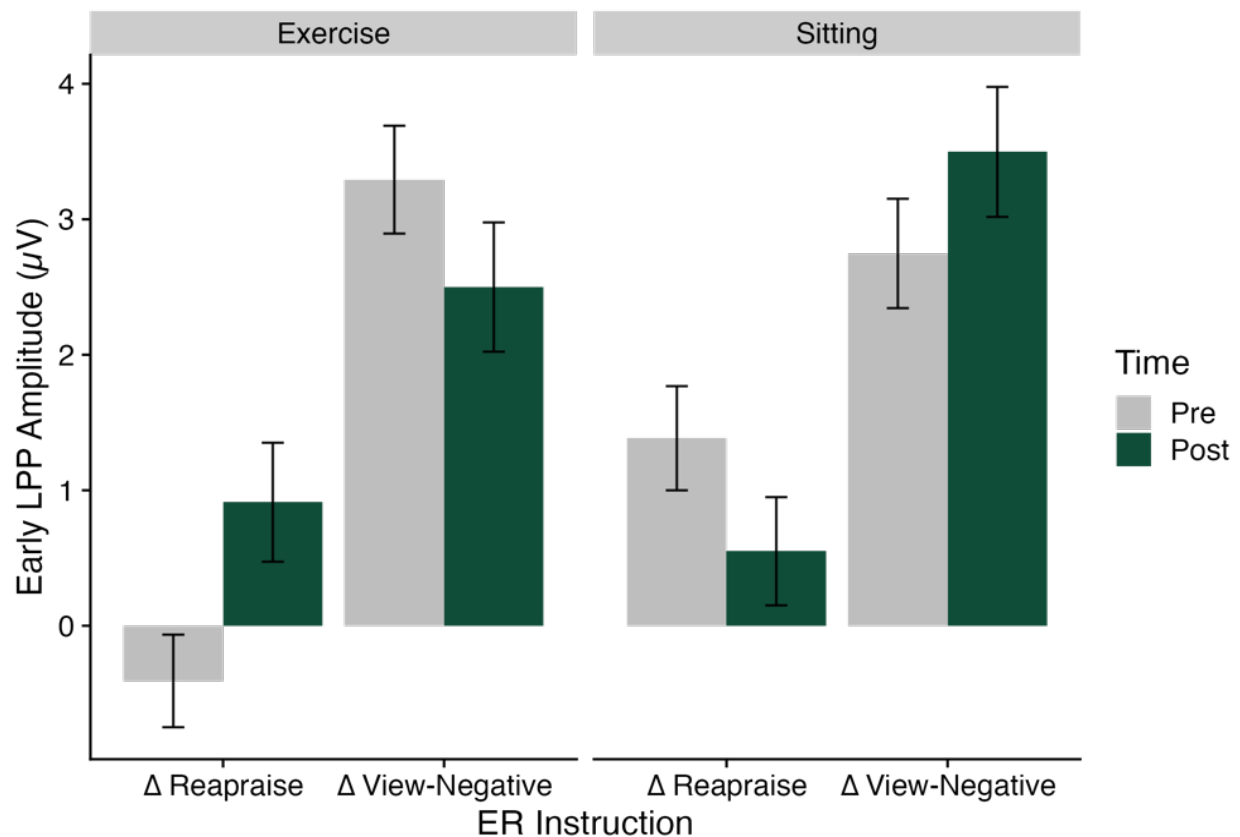
Note. Grand average stimulus-locked waveforms depicting the early and LPP amplitudes during the exercise session within each ER instruction at pre (top left) and post-test (bottom left). LPP amplitudes were extracted at the CPz electrode site. The yellow shaded area reflects the early LPP time window (400-1000 ms), and the blue shaded area reflects the late LPP time window (1s-6s). Topographic head maps depicting the reappraise minus view-negative difference score for the early and late LPP (right). Positive amplitude (µV) is reflective of a larger LPP amplitude during reappraisal relative to view-negative.

Figure 9. LPP waveforms and topographic head maps during the sitting session by time



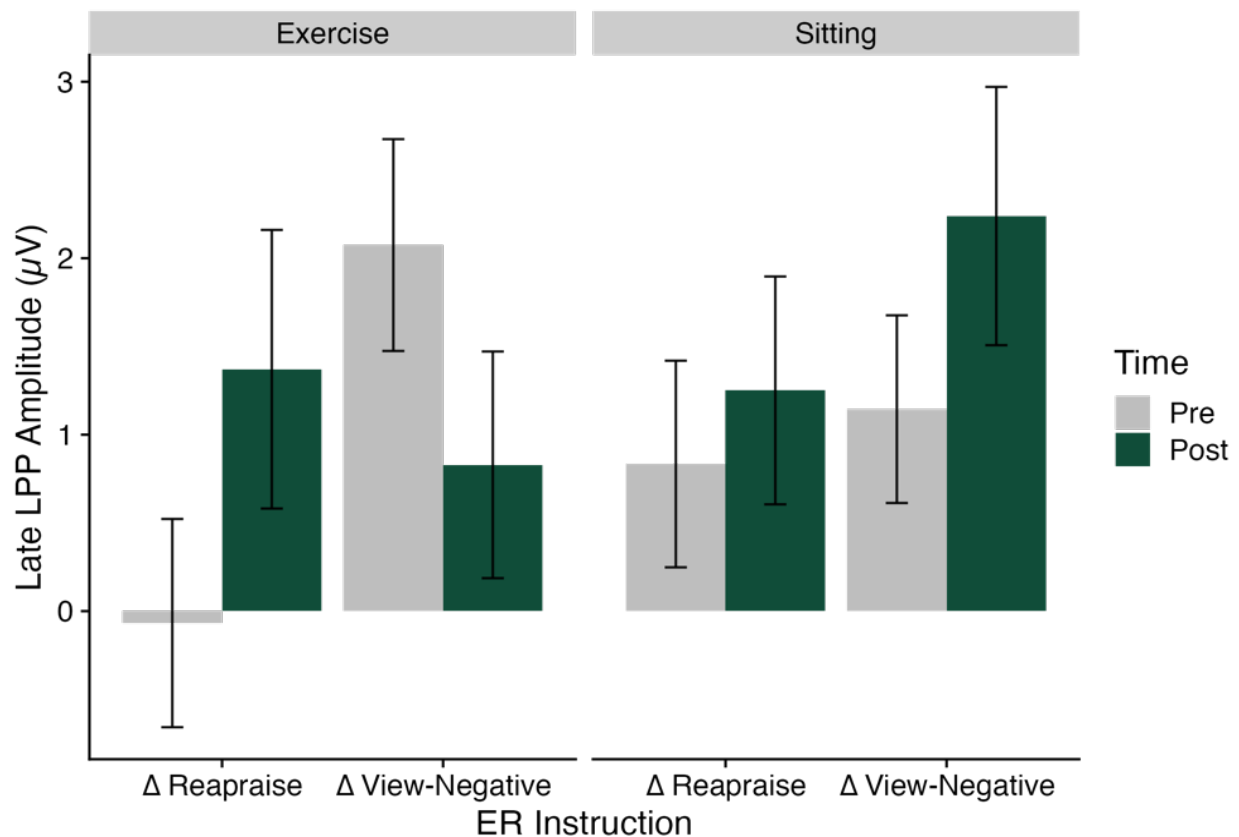
Note. Grand average stimulus-locked waveforms depicting the early and LPP amplitudes during the sitting session within each ER instruction at pre (top left) and post-test (bottom left). LPP amplitudes were extracted at the CPz electrode site. The yellow shaded area reflects the early LPP time window (400-1000 ms), and the blue shaded area reflects the late LPP time window (1s-6s). Topographic head maps depicting the reappraise minus view-negative difference score for the early and late LPP (right). Positive amplitude (μV) is reflective of a larger LPP amplitude during reappraisal relative to view-negative.

Figure 10. Early LPP Bar Graphs



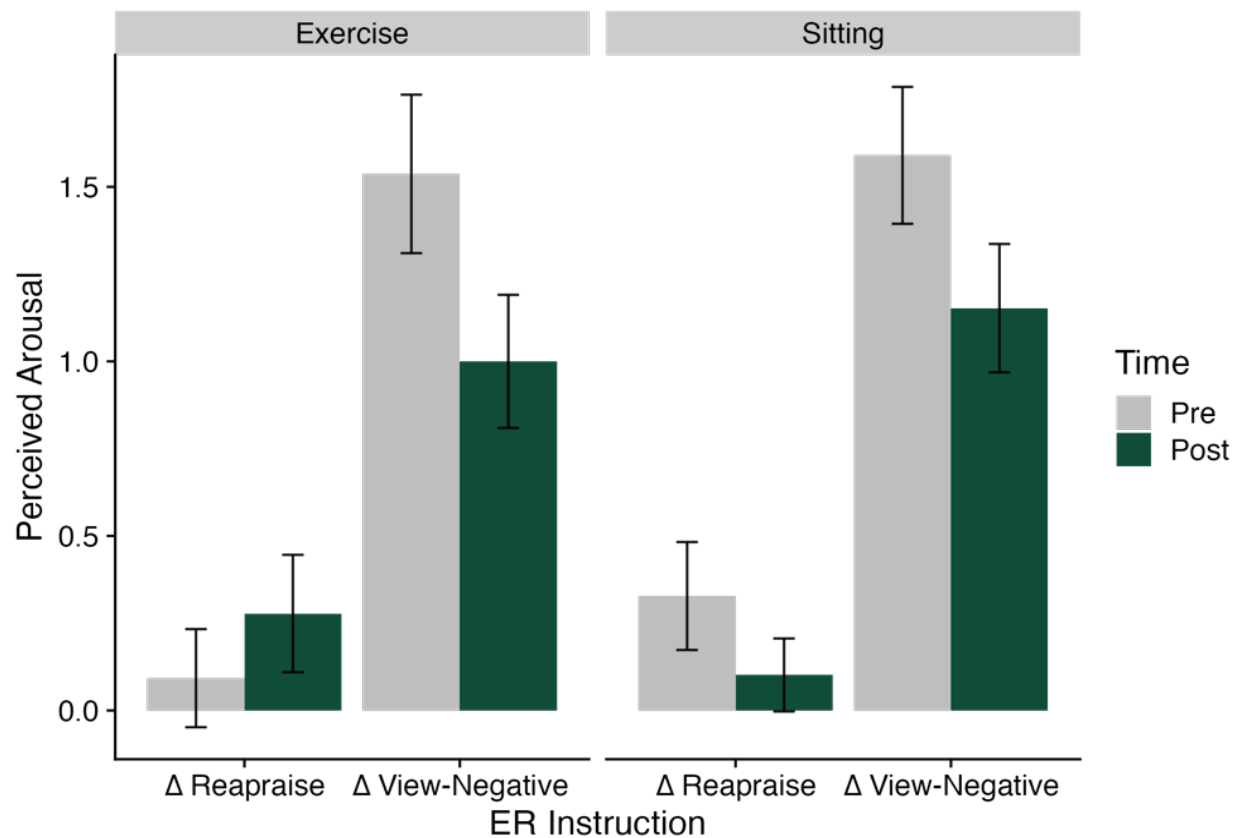
Note. Bar Graphs depicting the early LPP amplitude during the emotion regulation task by ER instruction, time, and session. The early LPP was extracted at the CPz electrode site, as the mean amplitude 400-1000 ms post-stimulus onset. Δ Reappraise was computed as the difference in amplitude (μ V) between reappraise and view-negative trials. Positive amplitudes reflect a larger early LPP amplitude during reappraise relative to view-negative trials. Δ View-Negative was computed as the difference in amplitude between view-negative and view-neutral. Positive amplitudes reflect a larger early LPP amplitude during view-negative relative to view-neutral trials.

Figure 11. Late LPP Bar Graphs



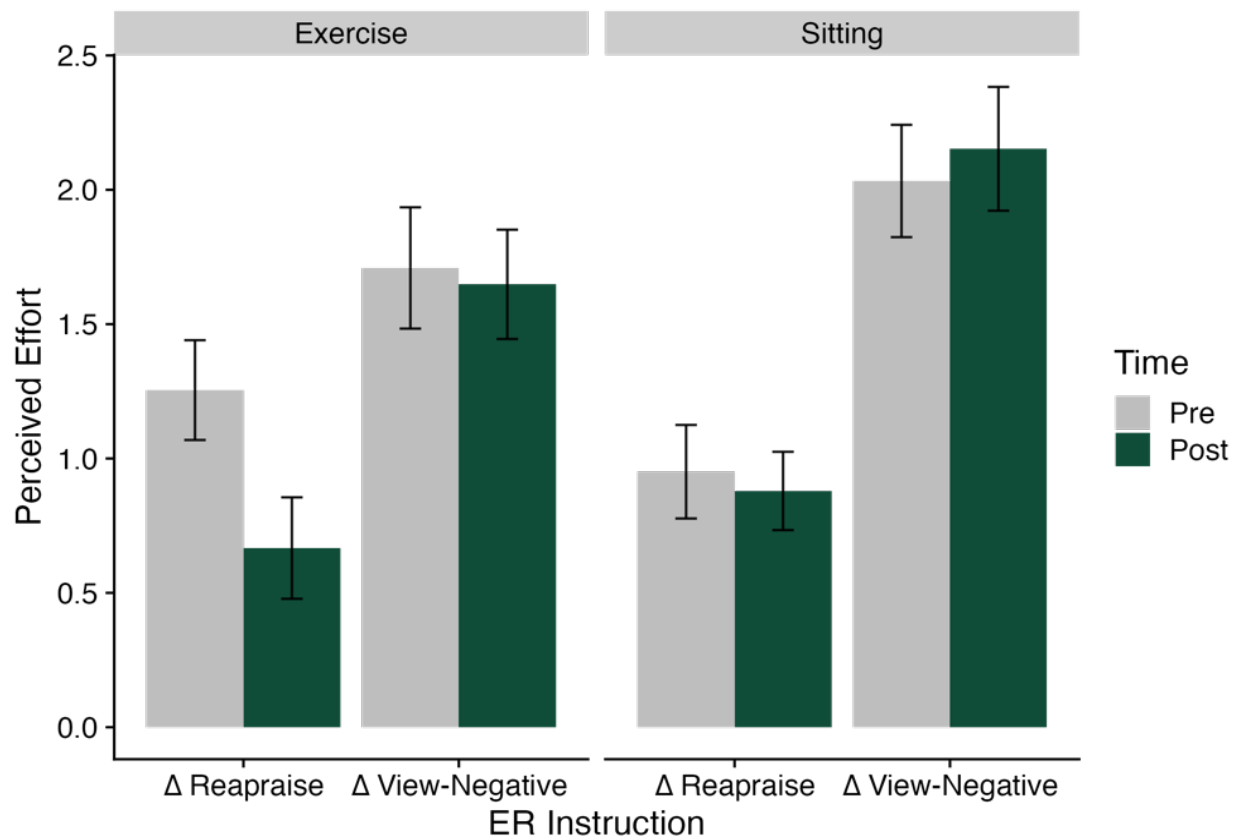
Note. Bar Graphs depicting the late LPP amplitude during the emotion regulation task by ER instruction, time, and session. The late LPP was extracted at the CPz electrode site, as the mean amplitude 1-6 s post-stimulus onset. ΔReappraise was computed as the difference in amplitude (μV) between reappraise and view-negative trials. Positive amplitudes reflect a larger late LPP amplitude during reappraise relative to view-negative trials. ΔView-Negative was computed as the difference in amplitude between view-negative and view-neutral. Positive amplitudes reflect a larger late LPP amplitude during view-negative relative to view-neutral trials.

Figure 12. Bar graphs of self-reported emotional arousal



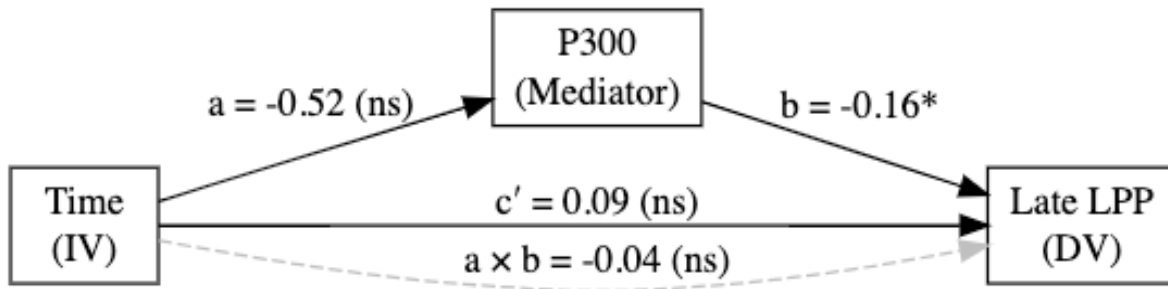
Note. Bar graphs depicting self-reported perceived emotional arousal during the emotion regulation task. Δ Reappraise was computed as the difference in perceived arousal between reappraise and view-negative trials. Positive values reflect greater emotional arousal during reappraisal relative to view-negative trials. Δ View-Negative was computed as the difference in perceived arousal between view-negative and view-neutral. Positive values reflect greater emotional arousal during view-negative relative to view-neutral trials.

Figure 13. Bar graphs of self-reported emotion regulation effort



Note. Bar graphs depicting self-reported perceived effort during the emotion regulation task. Δ Reappraise was computed as the difference in perceived effort between reappraise and view-negative trials. Positive values reflect greater effort during reappraisal relative to view-negative trials. Δ View-Negative was computed as the difference in perceived effort between view-negative and view-neutral. Positive values reflect greater effort during view-negative relative to view-neutral trials.

Figure 14. Mediation model



Note. Path diagram illustrating the hypothesized and tested mediation model. The independent variable, Time, reflects the effect of exercise from pre-test to post-test. The mediator is the P300 amplitude, and the dependent variable is the late LPP amplitude during reappraisal.

APPENDIX C: SUPPLEMENTAL MATERIALS

Verbatim Emotion Regulation Instructions

View instructions

“When you see the phrase VIEW NEUTRAL OR VIEW NEGATIVE, you should let yourself respond naturally to the image. Don't change your natural emotional response; just respond to the image as you naturally would. Don't think about unrelated images when viewing these pictures. Also, make sure to view the image the whole time it's displayed on the screen; don't look away or close your eyes. Remember to respond to the image as you naturally would. It's important to note that the word “neutral” or “negative” DOES NOT mean you should respond to the image in a neutral or negative way; rather, it just means what kind of image is coming up next.”

Reappraise Instructions

“When you see the instruction phrase REAPPRAISE NEGATIVE, you should think about the following picture in such a way that you feel your negative emotions LESS strongly. Specifically, you should imagine that the pictured event gets better. For example, if you were viewing an image of a sick person, you could imagine that the person is only tired, strong-willed, or likely to recover quickly.

Do not simply think about something unrelated to the scene or try to just “think happy thoughts”. Don't think of the situation as fake or think of it like a movie. Also, please refrain from trying to distance yourself from the situation. Don't look away or close your eyes while the image is displayed; just try to think about the picture in the way I described by imagining the picture in a more positive light. Imagine a brighter future for each image.”

Self-Report Questionnaires

The following self-report questionnaires were also collected during the baseline questionnaire. These questionnaires were collected to further understand the characteristics of this sample and investigate potential individual differences. However, these questionnaires were not part of my hypotheses, and therefore, were not included in the analyses of this study.

The Life Events Checklist for the DSM-5 (LEC-5; (Weathers, Blake, et al., 2013) was used to assess for trauma history. The LEC-5 is a 17-item self-report measure designed to assess for exposure to various potentially traumatic experiences during an individual's lifetime. The items on this questionnaire assess for events that have the potential to result in PTSD (e.g., physical assault, combat, or exposure to a warzone). The LEC-5 also assesses the degree of exposure to a potentially traumatic event (e.g., happened to me, witnessed it, learned about it). Participants in this study were instructed to select multiple levels of exposure, if applicable.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) was used to assess individual differences in worry. The PSWQ is a 16-item questionnaire designed to assess worry symptoms. Responses on the PSWQ are rated on a 5-point Likert scale (1 = "not typical of me" to 5 = "very typical of me"). Total scores are determined by summing the scores for each of the 16 items, with higher scores indicating more significant worry symptoms. The PSWQ has been shown to be psychometrically sound with high validity and reliability (Meyer et al., 1990).

Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; (Radloff, 1977). The CESD is a 20-item self-report questionnaire designed to assess for depressive symptoms. Responses on the CES-D are rated on a 5-point Likert scale (1 = "Rarely or none of the time" to 5 = "Most or all of the time"). Total scores are determined by summing the scores for each of the 20 items, with higher scores indicating more

significant depressive symptoms. The CES-D has been shown to be psychometrically sound with high validity and reliability (Radloff, 1977).

Trait anxiety symptoms were assessed using the State-Trait Anxiety Inventory-Trait (Spielberger et al., 1983). The STAI-T is a 20-item questionnaire that measures anxiety symptoms on a 4-point Likert scale (1 = “Almost Never” to 4 = “Almost Always”). Total scores are determined by summing the scores for each of the 20 items, with higher scores indicating greater trait anxiety. The STAI-T has been shown to be psychometrically sound with high validity and reliability (Spielberger et al., 1983).

Appraisal of stressful life events was assessed using the Perceived Stress Scale (PSS; Cohen et al., 1983). The PSS is a 14-item self-report measure designed to assess “the degree to which individuals appraise situations in their lives as stressful” (Cohen et al., 1983, p. 385). The PSS measures perception of stress in the past month on a 5-point (0 = “Never” to 4 = “Very Often”) Likert scale. The PSS has been shown to be psychometrically sound with high validity and reliability (Cohen et al., 1983, Lee, 2012).

Affective experiences were measured using the Positive and Negative Effect Schedule (PANAS; Watson et al., 1988). The PANAS is a 20-item self-report measure designed to assess the degree to which individuals experience positive and negative emotions. The PANAS is measured using a 5-point (1 = “Very slightly or not at all” to 5 = “Extremely”) Likert scale. The PANAS has two subscales – Positive and Negative affect, with higher scores indicating greater positive and negative affect. The PANAS has been shown to be psychometrically sound with high validity and reliability (Watson et al., 1988).

The Profile of Mood States (POMS; Shacham, 1983) was used to measure positive and negative mood states. The POMS is a 40-item self-report measure designed to assess for current

mood states rated using a 5-point (0 = “Not at all” to 4 = “Extremely”) Likert scale. The POMS assesses mood states across seven subscales. The two positive mood subscales are 1) Esteem-related Affect (ERA) and 2) Vigor (VIG); and the five negative mood subscales are 1) Anger (ANG), 2) Confusion (CON), 3) Depression (DEP), 4) Fatigue (FAT), and 5) Tension (TEN). Total Mood Disturbance (TMD) is a composite scale that consists of the sum of the negative mood subscales minus the sum of the positive mood scales.

Emotion Regulation Questionnaires

Individual differences in emotion regulation were measured using the Emotion Regulation Questionnaire and the Difficulties in Emotion Regulation Scale. The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is a 10-item self-report measure designed to assess individual differences in the use of two common emotion regulation strategies – cognitive reappraisal and suppression. Cognitive reappraisal and suppression are measured using a 7-point (1 = “Strongly disagree” to 7 = “Strongly Agree”) Likert scale, with higher scores indicating greater use of each respective emotion regulation strategy.

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) was used to assess individual differences in one’s ability to regulate their emotions. The DERS is a 36-item self-report questionnaire that assesses emotion regulation difficulties across six subscales: 1) Nonacceptance of Emotional Responses, 2) Difficulties Engaging in Goal-Directed Behavior, 3) Impulse Control Difficulties, 4) Lack of Emotional Awareness, 5) Limited Access to Emotion Regulation Strategies, and 6) Lack of Emotional Clarity (Gratz and Roemer 2004). The DERS measures emotion regulation difficulties using a 5-point (1 = “Almost Never” to 5 = “Almost Always”) Likert scale with scores indicating greater difficulty regulating emotions. The DERS

has been shown to be psychometrically sound with strong internal consistency, reliability, and predictive validity (Gratz & Roemer, 2004).

Physical Activity Questionnaires

Individual differences in physical activity levels were measured using the International Physical Activity Questionnaire-Short Form (IPAQ-SF; Craig et al., 2003). The IPAQ-SF is a 7-item self-report questionnaire designed to measure physical activity levels in the past 7 days. The questionnaire assesses the number of minutes per day spent doing vigorous and moderate physical activity, along with the number of minutes spent walking and sitting. Physical activity was measured by converting the number of minutes engaged in physical activity to a metabolic equivalent task value (MET-minutes/week).