EÖTVÖS LORÁND UNIVERSITY
FACULTY OF EDUCATION AND PSYCHOLOGY

Atakan Mustafa Akıl

AN INVESTIGATION OF SELF-REGULATION AND FRONTAL ALPHA ASYMMETRY

DOI: 10.15476/ELTE.2025.007

Doctoral School of Psychology
Head of the School: Prof. **Róbert Urbán**, Eötvös Loránd University

Clinical Psychology and Addiction Program
Head of the Program: Prof. **Zsolt Demetrovics**, Eötvös Loránd University

Supervisor
Assoc. Prof. **Adrienn Ujhelyi**, Eötvös Loránd University

Budapest, 2024

Table of Contents

CHAPTER III

Acknowledgements

First, I would like to express my deepest gratitude to my supervisor, Adrienn Ujhelyi, for the guidance, encouragement, and patience throughout this journey, as well as to H. N. Alexander Logemann for the support and insights. My heartfelt thanks extend to the members of the defense committee for their time, expertise, and thoughtful evaluation of this dissertation. I am also thankful to my colleagues and friends, your kindness and inspiration have been a great source of strength. I dedicate this dissertation to my mother, Dilek F. Gunduz, my father, Umit Akil, and my partner, Siyu Li. Their love, support, and belief in me have been my greatest motivation and source of resilience.

List of Abbreviations

AI: Artificial Intelligence

ANOVA: Analysis of Variance

BF: Bayes Factor

BMC: Bilateral Muscle Contraction

CEFR: Common European Framework of Reference for Languages

CI: Confidence Interval

cm: Centimeter

DERS: Difficulties in Emotion Regulation Scale

DERS-16: Difficulties in Emotion Regulation Scale-16

DLPFC: Dorsolateral Prefrontal Cortex

DSM-5: Diagnostic and Statistical Manual of Mental Disorders 5

EC: Eyes-closed

EEG: Electroencephalogram

ELTE: Eötvös Loránd University

EO: Eyes-open

ERP: Event-Related Potential

FAA: Frontal Alpha Asymmetry

FFT: Fast Fourier Transform

GAPED: Geneva Affective Picture Database

HEOG: Horizontal Electrooculography

Hz: Hertz

ICA: Independent Component Analysis

IRB: Institutional Review Board

M: Mean

MDD: Major Depressive Disorder

ms: Milliseconds

POMS: Profile of Mood States

POMS-SF: Profile of Mood States-Short Form

PSD: Power Spectral Density

SE: Standard Error

SD: Standard Deviation

SPSS-23: Statistical Package for Social Sciences 23

SOA: Stimulus Onset Asynchrony

SSRT: Stop-Signal Reaction Time

SST: Stop Signal Task

tDCS: Transcanial Direct Current Stimulation

tES: Transcranial Electrical Stimulation

UMC: Unilateral Muscle Contraction

VEOG: Vertical Electrooculography

List of Tables

List of Publications Included in the Dissertation

Akil, A. M., Ujhelyi, A., & Logemann, H. N. A. (2022). Exposure to Depression Memes on Social Media Increases Depressive Mood and It Is Moderated by Self-Regulation: Evidence From Self-Report and Resting EEG Assessments. *Frontiers in Psychology*, 13, 880065. https://doi.org/10.3389/fpsyg.2022.880065[1]

Akil, A. M., Watty, M., Cserjesi, R., & Logemann, H. N. A. (2024). The relationship between frontal alpha asymmetry and self-report measurements of depression, anxiety, stress, and self-regulation. *Applied Neuropsychology: Adult*, 1–7. https://doi.org/10.1080/23279095.2024.2425361

Akil, A. M., Cserjési, R., Nagy, T. Demetrovics, Z., & Logemann, H. N. A. (2024). The effect of unilateral hand muscle contraction on frontal alpha asymmetry and inhibitory control in intrinsic reward contexts, a randomized controlled trial. *Scientific Reports,* 14, 27289. https://doi.org/10.1038/s41598-024-74070-8

Akil, A. M., Cserjési, R., Nagy, T., Demetrovics, Z., Németh, D., & Logemann, H. N. A. (2024). The relationship between frontal alpha asymmetry and behavioral and brain activity indices of reactive inhibitory control. *Journal of Neurophysiology*, 132. https://doi.org/10.1152/jn.00046.2024

[1] Akil A.M., Ujhelyi, A. & Logemann, H.N.A. (2023) Corrigendum: Exposure to depression memes on social media increases depressive mood and it is moderated by self-regulation: Evidence from self-report and resting EEG assessments. *Frontiers in Psychology*. https://doi.org/10.3389/fpsyg.2023.1146810

CHAPTER I

1. General Introduction

This dissertation investigated the relationship between self-regulation and frontal alpha asymmetry. Through the execution of four studies, all of which have been published in peer-reviewed journals, we sought to deepen our understanding regarding these two phenomena. Each study will be discussed in its respective chapters.

All studies were approved by the Ethics Committee of ELTE Eötvös Loránd University, and all participants provided informed consent. We adhered to the ethical guidelines outlined in the Declaration of Helsinki and its subsequent amendments, ensuring the integrity of our methodology, and embraced open science principles, promoting transparency and reproducibility in our work. We declare that no scientific content has been generated by artificial intelligence (AI); rather, AI technology has solely assisted in enhancing the language and clarity of the material presented based on relevant regulations.

The first chapter was dedicated to providing an overview of the key concepts used in this dissertation. Our objective was to establish a foundational understanding of self-regulation and frontal alpha asymmetry. In the second chapter, we explored each of the four studies and provided summary information regarding them. In the final chapter of the dissertation, we synthesized the general findings collected from our research endeavors and discussed them together. We evaluated the results, implications, and limitations, which will hopefully guide further advancements in the field of psychology, specifically regarding the relationship between self-regulation and frontal alpha asymmetry.

1.1. Self-regulation

Self-regulation encompasses a wide range of processes and the precise definition of it remains a subject of ongoing scholarly debate, leading to varying and sometimes conflicting interpretations. One of the problems arises over whether self-regulation indicates the regulation by the self or of the self. For instance, the human body

constantly engages in various processes to maintain a stable internal temperature (i.e., homeostasis; (Cooper, 2008)), and this has been considered as a form of regulation of the self (Vohs & Baumeister, 2004). In contrast, self-regulation also signifies the connotation of regulation by the self. For example, while the psychological self may not actively participate in regulating bodily temperature, it often plays an essential role in regulating emotions, impulses, or thoughts (Vohs & Baumeister, 2004). Another critical challenge arises from the overlap with similar concepts, such as self-control and cognitive control. Self-control emphasizes resolving conflicts between goals that are temporally asymmetric (Inzlicht et al., 2021), while cognitive control focuses on attention processes and involves allocation of attention to support goal-directed behavior (Cohen, 2017).

Hence, when engaging with the literature, it is important to acknowledge the coexistence of various definitions and interpretations of self-regulation and appreciate their nuanced distinctions. Putting aside all indicated issues, the philosophical debate surrounding the nature of self-regulation falls outside the scope of this dissertation. We investigated self-regulation experimentally. An apt definition of self-regulation for the purpose of our research is simply the capacity to control thoughts, emotions, and behaviors (Baumeister et al., 1994).

As can be seen from the definitions, the term self-regulation is commonly used to describe any effort by an organism to modify or adjust its own responses (Tice & Bratslavsky, 2000). It is a broad and multifaceted concept situated at the crossroads of various academic disciplines and it encompasses a wide range of processes that involve cognitive, affective, behavioral, and environmental factors. In this dissertation, we mainly explored self-regulation through the lens of affective valence and motivational/behavioral responses, focusing on two contrasting emotional contexts: encountering negative valence stimuli (e.g., depression-related images) and positive valence stimuli (e.g., food-related images).

In daily life, individuals frequently encounter stimuli that can trigger emotional responses. Here, they can employ emotion-related self-regulation, also known as emotion regulation, which is a subconstruct of the broader concept of self-regulation and defined as the processes used to manage and modify when and how one experiences

emotions, along with the related motivational and physiological states, as well as the behavioral expression of those emotions (Eisenberg et al., 2007). This concept was further elucidated by distinguishing between intrinsic and extrinsic forms of emotion regulation (Gross, 2014). Intrinsic emotion regulation, which we investigated in this dissertation because our aim was to confirm a brain activity index regarding frontal hemispheric activity that could represent personal processes, involves individuals regulating their own emotions. Conversely, extrinsic emotion regulation entails individuals regulating the emotions of others, such as a caregiver soothing a crying baby (Gross, 2014).

During emotion regulation, individuals can increase, maintain, or decrease their positive and negative emotions via numerous strategies. Down-regulation processes strive for a rapid decrease in emotional intensity, facilitating a swift return to a neutral state (Gross, 1998a). Conversely, maintenance processes aim to sustain the emotional response over an extended period. Meanwhile, up-regulation processes can enhance the emotional response, sometimes by intensifying it (Schmeichel et al., 2006). Since emotion regulation influences emotions, its effects can be observed across all aspects of emotional responses, including feelings, physiology, behaviors, and thoughts.

The nature of emotions has been a long-standing topic of debate in the literature, with two primary theoretical approaches emerging. The first posits that emotions are organized along continuous dimensions, such as valence (positive to negative), arousal (low to high), and approach and avoidance motivations (Russell, 1980; Harmon-Jones et al., 2017). In contrast, the second approach conceptualizes emotions as discrete entities, such as anger, sadness, happiness, and fear (Ekman, 1992). Interestingly, research indicates that fundamental emotional processes, like approach and avoidance motivations, are present even in simple organisms such as worms (Harmon-Jones et al., 2017). This suggests that emotional responses could be fundamentally organized along these dimensions, then it becomes more complex after with cognitive reappraisal (Bradley et al., 2000), which is giving new meaning to an event or a stimulus (Garnefski et al., 2001). This perspective aligns with Koole's work, which highlights that emotion regulation may not necessarily focus on shifting people in or out of specific emotional states like anger, sadness, or joy, but rather on altering their emotional states along these

dimensions (Koole, 2009). Hence, in this dissertation, we interpret our results mainly based on dimensions of emotions.

Emotion regulation strategies were classified using various approaches, one of which focuses on cognitive aspects, known as cognitive emotion regulation strategies. These strategies involve the cognitive management of emotionally arousing information (Thompson, 1991). Research on cognitive emotion regulation has identified a functional distinction between these strategies based on their potential to promote adaptive or maladaptive responses to environmental challenges. For instance, strategies like reappraisal and problem-solving were typically linked to adaptive outcomes, such as a reduction in negative affect (Senqing et al., 2020; Vanderhasselt et al., 2014). Conversely, maladaptive strategies, including rumination, constantly thinking about the feelings and thoughts, catastrophizing, emphasizing the terror of an event, and self-blame, thoughts of blaming yourself for what you have experienced (Garnefski et al., 2001), were associated with negative outcomes like decreased autonomic flexibility (Hofmann et al., 2005).

Due to limited cognitive resources, there is a constant competition between emotion generation and emotion regulation processes for dominance over behavior (Gross et al., 2011a, 2011b) and effective emotion regulation requires the ability to recruit deliberate executive control processes to override conflicting associative emotional processes (Muraven & Baumeister, 2000) and achieve expected behavioral outcomes.

Inhibitory control is a foundational concept in understanding the behavioral component of self-regulation, often studied across different mental health disorders such as obesity (de Klerk et al., 2023), substance abuse (Feil et al., 2010), major depressive disorder (Han et al., 2020). It refers to the capacity to deliberately suppress or override a planned response in favor of a more goal-directed behavior (de Jong et al., 1990) and it is typically assessed using computer-based tasks such as the Stop Signal Task (Logan et al., 1984; Schmajuk et al., 2006), which measures action withdrawal in response to go stimuli (e.g., food images), which entails the ability to inhibit an action that has already been initiated (Mirabella, 2021).

Therefore, if self-regulation fails, it can result in unwanted emotions, thoughts, and behaviors despite the best efforts to avoid them. Specifically, previous studies have consistently found a significant link between self-regulatory dysfunction and various psychological disorders. For instance, anxiety disorders often involve difficulties in managing stress (Dubuc-Charbonneau & Durand-Bush, 2015) and controlling anxious thoughts (Kocovski & Endler, 2000), leading to persistent worry and fear. Depression is frequently associated with impaired self-regulation, resulting in an inability to down-regulate negative emotions (Strauman, 2002). Eating disorders are characterized by severe disruptions in self-regulatory processes, including the regulation of eating behaviors (Crino et al., 2019).

These findings highlight the importance of effective self-regulation in preventing and managing psychological disorders, underscoring its role as a foundational component of mental health. Hence, effective self-regulation can be crucial in maintaining a healthy lifestyle, encompassing physical, mental, and emotional well-being. The ability to manage one's thoughts, emotions, and behaviors in the face of challenges is essential for achieving long-term goals and sustaining overall health. These findings do not only underscore the urgency of understanding and addressing self-regulatory deficits further but also prompts contemplation on potential biomarker that could function as a target for therapeutic interventions directed at psychological health problems characterized by impaired self-regulation.

In this dissertation, we investigated frontal brain activity, with a particular focus on frontal asymmetry, as a candidate neural marker of self-regulation. Frontal asymmetry reflects the balance of activity between the left and right frontal hemispheres, which has been linked to approach and avoidance tendencies (Coan et al., 2006). However, it is still understudied whether it moderates and represents self-regulatory processes.

1.2. Mechanisms of self-regulation

Studies regarding brain lateralization have experienced fluctuations in research interest, marked by periods of heightened attention followed by periods of reduced focus. According to Vallortigara and Rogers, Broca’s discoveries regarding speech production in 1860s and Sperry’s discoveries with split-brain patients in the 1960s are important

time points in the history of hemispheric lateralization. Since then, numerous studies showed that each hemisphere is responsible for different emotional, behavioral, and cognitive processes (Vallortigara & Rogers, 2005).

In the 1970s, an intriguing discovery was made by Davidson et al. (Davidson, 1998; Davidson et al., 1990; Fingelkurts & Fingelkurts, 2015). It was found that there is a connection between the frontal electrophysiological activity of the brain and two primary motivational tendencies related to self-regulation: approach and withdrawal. One potential contributor to the functioning of approach and avoidance systems is the asymmetry in activity of the dorsolateral prefrontal cortex (DLPFC) (Kelley et al., 2007; Vallortigara & Rogers, 2005; Zheng et al., 2008). Specifically, an increase in right prefrontal activity, whether as a trait or a state, was associated with withdrawal motivation, while heightened left prefrontal activity was correlated with approach motivation.

Moreover, this asymmetry model was later found to be linked to valence-arousal model (Heller, 1993). Positive emotions were generally associated with left frontal brain activity and approach-related motivations, whereas negative emotions were typically correlated with right frontal brain activity and withdrawal-related motivations (Vohs & Baumeister, 2004).

Therefore, in contrast, the valence-arousal model (Heller, 1993) emphasizes that the emotional valence (positive or negative) is more critical than motivational tendencies in determining hemispheric activity. Despite these differences, both models agree that positive emotions are generally associated with approach-related motivation, and negative emotions with withdrawal-related motivation. However, research findings on anger, which has a negative valence but is also associated with an approach tendency (Berkowitz, 1999), indicate that frontal electrophysiological asymmetry may reflect the direction of motivation (approach versus withdrawal) rather than purely emotional valence.

This has led to more nuanced models such as the capability model proposed by Coan et al. (Coan et al., 2006). The capability model suggests that frontal electrophysiological asymmetry is not just a static marker of affective dispositions in a resting state but is

also influenced by situational factors and an individual's capacity to either approach towards or withdraw from emotionally salient contexts. Therefore, the electrophysiological asymmetry varies depending on specific emotional situations and the individual's regulatory capacity to either engage with or inhibit responses.

Lately, Grimshaw and Carmel introduced the asymmetric inhibition model, which reframes frontal asymmetries in terms of executive control mechanisms (Grimshaw & Carmel, 2014). According to this model, the left frontal cortex is responsible for inhibiting negative distractions, while the right frontal cortex suppresses positive distractions. This perspective explains why reduced left frontal activity is linked to an inability to disengage attention from negative stimuli, as seen in conditions like depression and anxiety (Cisler & Koster, 2010), whereas lower right frontal activity is associated with impaired inhibition of positive distractions, a pattern often observed in addiction (Goldstein & Volkow, 2011).

The overlap between these models makes it challenging to disentangle their unique contributions, as they often produce similar empirical predictions (Spielberg et al., 2008). Nevertheless, findings from numerous studies support the emerging consensus that relatively greater left frontal activity is associated with a general tendency toward appetitive or approach-related behaviors, while greater right frontal activity is linked to a predisposition toward avoidance or withdrawal responses (Coan & Allen, 2004; Davidson et al., 1990). Thus, while these models offer distinct perspectives, they converge on the idea that frontal electrophysiological asymmetry serves as an indicator of broader motivational and emotional processes, shaped by both trait dispositions and situational influences.

1.3. Frontal alpha asymmetry

One candidate index for asymmetric frontal cortical activity is frontal alpha asymmetry (Allen et al., 2018; Palmiero & Piccardi, 2017). Evidence indicates that activity within the alpha frequency range (typically 8–13 Hertz (Hz)) may reflect an inverse relationship with underlying cortical processing. This means that a decrease in alpha power is generally observed when cortical regions are actively engaged in processing information. In other words, lower alpha activity is often associated with heightened

neural engagement (Coan & Allen, 2004). The most widely used index for calculating frontal alpha asymmetry is obtained by subtracting the natural logarithm of left hemisphere alpha power from the natural logarithm of right hemisphere alpha power (ln[right alpha] − ln[left alpha]). This method produces a scale that reflects the balance of neural activity between the right and left hemispheres, with a midpoint of zero indicating symmetrical activation (Coan & Allen, 2004). When interpreting this measure, higher values correspond to relatively greater left frontal activity, while lower values suggest relatively greater right frontal activity. It is important to note, however, that because alpha power is thought to be inversely related to cortical activity, a higher asymmetry score results from relatively greater alpha power in the right hemisphere, which implies reduced activity on the right and, consequently, relatively increased activation on the left.

Asymmetry scores provide a conceptual simplification for specific analyses, particularly those examining correlations involving frontal asymmetries as difference scores. Metrics based on alpha power asymmetries generally exhibit high internal consistency and satisfactory test-retest reliability, alleviating concerns about potential reliability issues typically associated with difference scores. Moreover, asymmetry metrics enhance the sensitivity of statistical tests by decreasing the number of contrasts within a given model, thereby boosting statistical power. This reduction in complexity allows for clearer interpretations of hemispheric dominance and its association with behavioral or psychological outcomes.

Previous studies have observed that greater activation of the right frontal cortex is associated with an withdrawal tendency from negative stimuli, whereas greater activation of the left frontal cortex is linked to an approach tendency toward positive stimuli (Coan and Allen, 2004; Harmon-Jones et al., 2010). Similarly, previous research has also indicated that greater right frontal cortical activity relative to left is indicative of enhanced inhibitory control (Vallortigara & Rogers, 2005), crucial role in adaptive behavior (Braver, 2012; Mirabella, 2023) and is linked to various mental health conditions such as obesity (de Klerk et al., 2023), substance abuse (Feil et al., 2010), major depressive disorder (Han et al., 2020).

These studies suggested that frontal alpha asymmetry could possibly be an indicator of self-regulatory processes in the context of approach and withdrawal motivations. However, to the best of our knowledge, it remains unknown whether frontal alpha asymmetry moderates depressive mood in response to negatively valenced images on social media as well as whether whether it also influences inhibitory behavior in exposure to positively valenced reward-related images. We aimed to address these questions in our studies.

There is consensus regarding how to process the electrophysiological data regarding frontal alpha asymmetry. We followed this consensus paper throughout our four studies (Smith et al., 2017). First, we recorded resting-state electrophysiological data using electroencephalogram (EEG) for 10 minutes, divided into 5-minute eyes-open (EO) and 5-minute eyes-closed (EC) conditions. Collecting frontal alpha asymmetry data in both of these states provides valuable insights into how sensory inputs, internal cognitive processes, and intrinsic brain activity influence frontal alpha asymmetry, enhancing our understanding of the brain's functional organization (Barry et al., 2007).

Subsequently, the recorded data were re-referenced to linked mastoids, with a low cutoff filter set at 0.5 Hz, high cutoff filter at 40 Hz, and notch filter at 50 Hz. To eliminate artifacts, the first and last 10 seconds of the EEG data were excluded. The remaining data were then segmented into 2-second epochs, and ocular artifacts were corrected using independent component analysis (ICA) based on the vertical electrooculogram (VEOG) and vertical electrooculogram (HEOG) electrodes. Epochs that still contained artifacts, determined by a minimum-maximum amplitude criterion of 75 microvolts, were discarded. The remaining epochs underwent whole-segment baseline correction, and Power Spectral Density (PSD) was computed using Fast Fourier Transform (FFT) with a 10% Hanning window. The epochs were averaged, and mean alpha activity in the 8-13 Hz frequency band was calculated and exported for the relevant electrodes. Then, alpha power was adjusted for skewness through a natural log transformation (Smith et al., 2017). Finally, frontal alpha asymmetry was calculated by subtracting the log-transformed alpha values from the lateral left electrode sites from those at the right electrode sites (F4-F3/F8-F7).

1.4. Event-related potentials: stop N2 and stop P3

In addition to frontal alpha asymmetry, event-related potentials could play a significant role in understanding the mechanisms of self-regulation. Event-related potentials reflect the coordinated activity of large groups of neurons, with their activation precisely aligned to the timing of a particular event (Kappenman & Luck, 2012). Specifically, two main event-related potentials associated with withdrawal motivation are the Stop N2 and Stop P3. The Stop N2 (Schmajuk et al., 2006), peaking at around 200 milliseconds (ms), shows larger negative amplitudes in stopping success compared to unsuccessful ones, and is linked to the right inferior frontal gyrus. The Stop P3, which exhibits larger amplitudes during successful stopping, is thought to originate primarily from the superior frontal gyrus (Kenemans & Kähkönen, 2010). These neural markers are closely associated with the inhibition of behavior, playing a critical role in the ability to suppress prepotent or ongoing responses, a fundamental aspect of the behavioral self-regulation.

Therefore, frontal alpha asymmetry provides a broad measure of hemispheric activation differences related to motivational and emotional states, while event-related potentials focus on the precise timing and neural mechanisms underlying specific event. In the context of self-regulation, frontal alpha asymmetry may offer insights into general motivational tendencies, whereas event-related potentials, such as Stop N2 and Stop P3, provide detailed information about specific inhibitory processes.

On the other hand, the relationship between approach and withdrawal motivations, frontal alpha asymmetry, and specific event-related potentials (Stop N2 and Stop P3) related to inhibitory control in food reward contexts remains underexplored. We investigated these relationships in this dissertation.

1. 3. Aim

The overarching aim of this dissertation was to explore the relationship between frontal alpha asymmetry and the cognitive, emotional, and behavioral components of self-regulatory processes. The research sought to address specific gaps in the literature by combining different methodologies. Table 1.1 shows the summary information regarding four studies.

Table 1.1.

Summary of Aims, Goals, and Methods from Studies

Summary	Aim	Hypothesis	Method
S1	To explore the relationship between frontal alpha asymmetry, self-report measurements of emotion regulation deficits and the effect of exposure to depression memes on depressive mood	Dispositional frontal alpha asymmetry would moderate the effect of depression memes on depressive mood. Higher self-report emotion regulation deficits would increase depressive mood after exposure to depression memes compared to neutral images.	A crossover study conducted in both in-lab and online settings
S2	To explore the relationship between self-reported measures of self-regulation and depression, and frontal alpha asymmetry	Dispositional frontal alpha asymmetry would be associated with the self-report measures of self-regulation and depression.	A correlational study conducted in-lab, incorporating both self-reported measures and electrophysiological recordings
S3	To explore the relationship between unilateral muscle contraction, frontal alpha asymmetry, and behavioral and brain activity indicators of inhibitory control	Compared to bilateral hand muscle contraction, unilateral left-hand muscle contraction would increase right frontal activity relative to the left (assessed via frontal alpha asymmetry), thereby enhancing inhibitory control (assessed via a computer task and event-related potentials), particularly in the reward condition.	A randomized controlled trial with unilateral left-hand muscle contraction or bilateral muscle contraction for 10 minutes
S4	To explore the relationship between transcranial direct current stimulation, frontal alpha asymmetry, and behavioral and brain activity indicators inhibitory control	Compared to sham condition, active anodal transcranial direct current stimulation would increase right frontal activity relative to the left (assessed via frontal alpha asymmetry), thereby enhancing inhibitory control (assessed via a computer task and event-related potentials), particularly in the reward condition.	A double-blind randomized sham-controlled trial with 2 milliampere anodal active or sham transcranial direct current stimulation for 20 minutes

Note. Abbreviations: S1 = Study 1; S2 = Study 2; S3 = Study 3; S4 = Study 4

Studies have shown that withdrawal motivation, reflected in heightened right-sided frontal brain activity (in contrast to approach motivation, which is associated with increased left-sided frontal brain activity), is linked to depressive mood (Coan & Allen, 2004; Harmon-Jones et al., 2010). However, the influence of depression memes on depressive mood, as well as the moderating role of frontal alpha asymmetry as a candidate index for emotion regulation in this relationship, remains unexplored. To address this gap, the first study examined the role of emotion regulation by investigating the relationship between frontal alpha asymmetry and the impact of depression memes

on depressive mood. Additionally, we also used self-report measurements regarding emotion regulation skills.

The findings of the first study prompted a deeper exploration of the relationship between self-reported measures and frontal alpha asymmetry. Specifically, they raised questions about how subjective reports of self-regulatory capacities and depressive symptoms align with neural markers like frontal alpha asymmetry. Given the importance of understanding these relationships, previous studies have begun to investigate them (Kemp et al., 2010). However, there remains a need for more extensive frontal alpha asymmetry data collected over longer periods and under varying conditions, such as EO and EC, to account for the influence of sensory input (Barry et al., 2007). This would allow for a more comprehensive evaluation of how frontal alpha asymmetry relates to individual differences in self-regulation and depression. Building on this need, our second study aimed to determine whether frontal alpha asymmetry, measured under these distinct conditions, could serve as a reliable indicator of self-regulatory capacities and depression.

In the final two studies, Study 3 and Study 4, we shifted our focus from examining dispositional frontal alpha asymmetry to directly manipulating frontal alpha asymmetry. This approach was designed to provide a more precise understanding of the relationship between self-regulation and frontal alpha asymmetry. Direct manipulation allowed for greater control over experimental conditions, offering a clearer insight into these phenomena.

We aimed to increase right-sided electrophysiological activity associated with withdrawal motivation, in contrast to left-sided activity linked to approach motivation, using non-invasive neuromodulation techniques. First, we employed unilateral muscle contraction (Harmon-Jones, 2006), a simple, cost-effective method that is easy to implement. In this technique, individuals simply squeezed a ball with their left hands to increase right frontal activity (Harmon-Jones, 2006). Second, we utilized transcranial direct current stimulation (Schestatsky et al., 2013), a technique that delivers low electric currents to the brain to activate specific regions. We expected that enhancing right-sided activity would decrease frontal alpha asymmetry and this would result in

improved inhibitory control (assessed via the Stop Signal Task and event-related potentials Stop N2 and Stop P3).

Overall, this dissertation aimed to advance the understanding of the relationship between frontal alpha asymmetry and self-regulation by exploring both dispositional and experimentally manipulated frontal alpha asymmetry. Through a combination of different approaches, it sought to clarify how frontal alpha asymmetry influences cognitive, behavioral, and emotional processes, including the regulation of depressive mood and inhibitory control. By addressing gaps in the literature and leveraging innovative neuromodulation techniques, this work contributes to a more comprehensive framework for understanding the neural underpinnings of self-regulatory processes and their potential applications for improving mental health.

CHAPTER II[2]

2. Study 1 - Exposure to Depression Memes on Social Media Increases Depressive Mood and It Is Moderated by Self-Regulation: Evidence From Self-Report and Resting EEG Assessments[3]

2.1. Introduction

The advancements in communication technology have made depression, a psychological condition mainly characterized by sadness, hopelessness, fatigue, loss of interest and pleasure (APA, n.d.), more apparent in daily life. For example, interactive online platforms like social media enable individuals to collaboratively create and moderate content without constraints of time or location (Aghaei, 2012). Consequently, these platforms offer a space for individuals to express their emotions openly.

Depression memes are a new form of communication used on the Internet. Figure 2.1 shows some examples. Internet memes are digital items with shared attributes in both form and content, distributed by numerous Internet users (Shifman, 2014). Milner and Miltner indicated that Internet memes have gained popularity due to their ability to evoke emotional resonance (Milner, 2016; Miltner, 2014). Depression memes are frequently circulated as a means to convey predominantly negative emotions. As a result, when individuals engage with these memes, they can be affected by the messages conveyed, warranting careful attention and consideration.

In one of the limited studies on the topic, Jadayel et al. observed in their case study that depression memes might prompt individuals towards physical harm and suicidal idealization (Jadayel et al., 2018). However, Akram et al. revealed in their exploratory study that humorous depression memes could potentially offer benefits to individuals if

[2] This chapter provides a concise summary of the published papers. For detailed information and supplementary materials, please refer to the original publications.

[3] Please refer to the published paper for further information and supplementary materials: Akil, A.M., Ujhelyi, A., & Logemann, H.N.A. (2022). Exposure to Depression Memes on Social Media Increases Depressive Mood and It Is Moderated by Self-Regulation: Evidence From Self-Report and Resting EEG Assessments. *Frontiers in Psychology*, 13, 880065. 10.3389/fpsyg.2022.880065.

adaptive emotion regulation strategies are employed (Akram et al., 2020). Clearly, depression memes exhibit a range of emotional valence. While certain depression memes adopt a humorous approach, eliciting laughter, others adopt a more negative tone, featuring sad quotes and monochromatic depictions of individuals experiencing negative mood.

Based on the existing literature, the effect of negative depression memes (hereafter referred to as depression memes) on mood and the moderating role of emotion regulation remain uncertain. As discussed previously in Chapter I, frontal alpha asymmetry could be a candidate index for emotion regulation. More specifically, previous research has suggested that heightened activation of the right frontal cortex is associated with an avoidance tendency in response to negative stimuli, while increased activation of the left frontal cortex is linked to an approach tendency toward positive stimuli (Coan & Allen, 2004; Harmon-Jones et al., 2010).

Therefore, in this study, we investigated that how dispositional frontal alpha asymmetry moderates the effects of exposure to depression memes on negative mood. We hypothesized that compared to neutral images, exposure to depression memes would elevate depressive mood, and frontal alpha asymmetry could moderate these effects. Additionally, we investigated that how self-reported emotion regulation skills affects the relationship between exposure to depression memes and depressive mood.

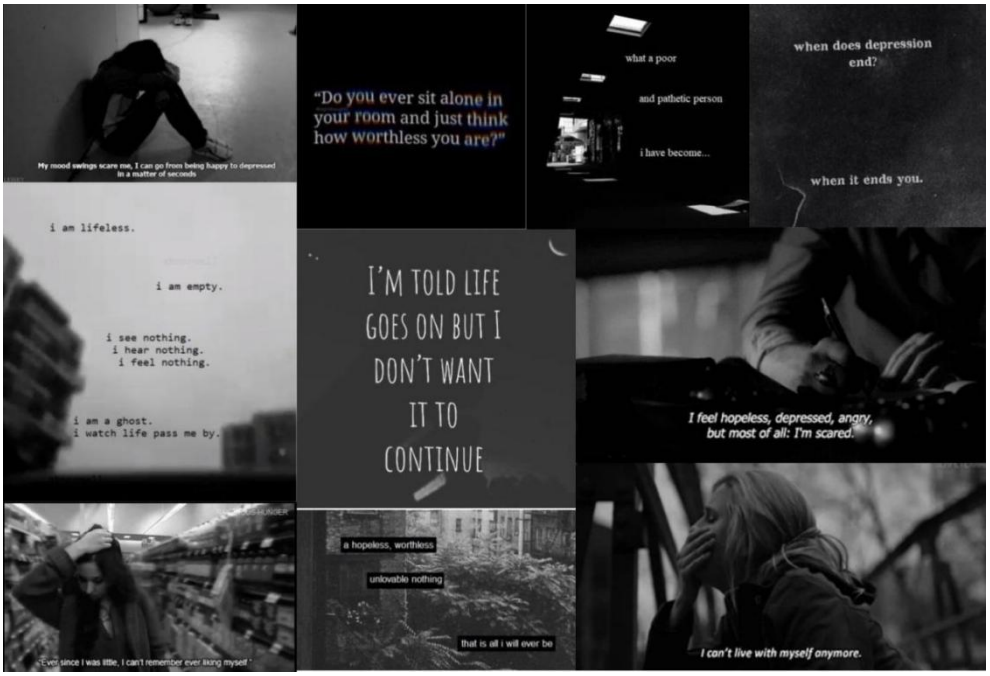


Figure 2.1. Examples of depression memes used in Study 1

2.2. Methods

2.2.1. Participants

Participants (n = 32 (20 females, 12 males, M_{age} = 29.4, SD_{age} = 9.5)) were recruited through social media ads, with eligibility criteria including a minimum age of 18. Exclusion criteria included psychological/psychiatric disorders, frequent headaches/migraines, epilepsy, significant or recent head trauma, and current drug use. At least two hours before the electrophysiological data collection, participants refrained from smoking, alcohol, or coffee. Participants were informed about the nature of the study. They gave informed consent and were provided with safety resources and support contacts in case of any discomfort. The study was approved by the Institutional Review Board (IRB) at Eötvös Loránd University.

2.2.2. Stimuli

We conducted a pilot study to develop and validate a new set of depression memes prior to the main study. We initially collected 1222 depression memes using TumblThreeApp,

a tool for downloading posts from Tumblr.com, a platform where depression memes are shared without restrictions. We excluded duplicates, advertisements, non-meme content, non-English memes, as well as those depicting mental health conditions other than depression, and those containing suicidal or sexual themes. Only memes that referenced at least one symptom of depression (e.g., hopelessness or worthlessness) as defined by the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) criteria for Major Depressive Disorder (MDD) were considered (APA, n.d.). A final set of 50 memes was chosen based on their potential to elicit either negative or humorous reactions, as determined by a voting process involving three researchers. All selected memes, preserving their original formatting and errors, were used in the pilot study. A total of 89 English-speaking individuals (43 females, 39 males, 6 non-binary/third gender, and 1 who preferred not to disclose; M_{age} = 29.5 years, SD_{age} = 9). Participants were then randomly shown 20 memes from the pool of 50 through the Qualtrics survey platform and asked to rate their emotional impact on a 9-point Likert scale from negative to positive. For the main study, 10 depression memes were selected based on low average scores and *SD*. Figure 2.1. shows the memes used in the study. To create a control condition, 10 neutral images from the Geneva Affective Picture Database (GAPED) were used instead of memes to avoid emotional priming (Dan-Glauser & Scherer, 2011). These neutral images depict non-emotive inanimate objects.

2.2.3. Questionnaires

Self-report assessments were conducted using PsyToolkit (Stoet, 2010, 2016). The Difficulties in Emotion Regulation Scale-16 (DERS-16) measured emotion regulation deficits. The DERS-16, developed by Bjureberg et al. (Bjureberg et al., 2015) from the original DERS (Gratz & Roemer, 2004), measures emotion regulation difficulties across five subscales: lack of emotional clarity, difficulties in goal-directed behaviors and impulse control, non-acceptance of emotional responses, and lastly, lack of adaptive emotion regulation strategies. Each subscale contains three items rated on a 5-point Likert scale ("almost never" to "almost always"). Higher scores indicate greater difficulties. Internal consistency (Cronbach's alpha) for subscales ranged from 0.82 to 0.94. The Profile of Mood States-Short Form (POMS-SF) was used to assess depressive mood pre- and post-intervention. The POMS-SF, initially developed by McNair et al. (McNair et al., 1971) and later adapted for various populations, is a 37-item checklist

rated on a 5-point Likert scale ("not at all" to "extremely"). For this study, only the depression subscale (eight items) was used, focusing on depressive mood such as sadness and hopelessness. Internal consistency was 0.89 pre-intervention and 0.95 post-intervention, indicating high reliability.

2.2.4. Frontal alpha asymmetry

Scalp voltage was recorded using a 21-channel EEG cap with Ag/AgCl electrodes, following the 10-20 system for electrode placement. The data was gathered via the NeXus-32 system from Mind Media (Nexus-32, n.d.). VEOG signals were recorded from electrodes placed above and below the left eye, while HEOG signals were captured from electrodes at the outer corners of both eyes. EEG data was re-referenced offline to linked mastoids. The data were recorded at a sampling rate of 512 Hz using a common average reference. Resting-state EEG data was collected for 10 minutes, divided into 5-minute EO and 5-minute EC conditions. Then, the data was preprocessed based on a previously established paradigm (Smith et al., 2017). Lastly, frontal alpha asymmetry scores were calculated by subtracting the log-transformed alpha values from the lateral left electrode sites from those at the right electrode sites (F4-F3/F8-F7). Please refer to Chapter I for more detail.

2.2.5. Procedure

Upon arrival at the laboratory, participants reviewed the information letter, confirmed exclusion criteria, and signed informed consent. They were then seated in a comfortable chair in a dimly lit room while an EEG cap and ocular electrodes were placed. These data were used to calculate frontal alpha asymmetry, and the online experiment followed in subsequent days. The online part consisted of two sessions separated by at least one day to prevent carry-over effects, with counterbalanced stimulus orders. Each session began with participants completing demographic questions, DERS-16, and POMS-SF. Then, either neutral images or depression memes were shown randomly on the screen, and participants rated each stimulus on a 9-point Likert scale (negative to positive) to maintain focus. Afterward, they completed the POMS-SF again to assess post-intervention depressive mood. Each session lasted around 20 minutes. The task is depicted in Figure 2.2.

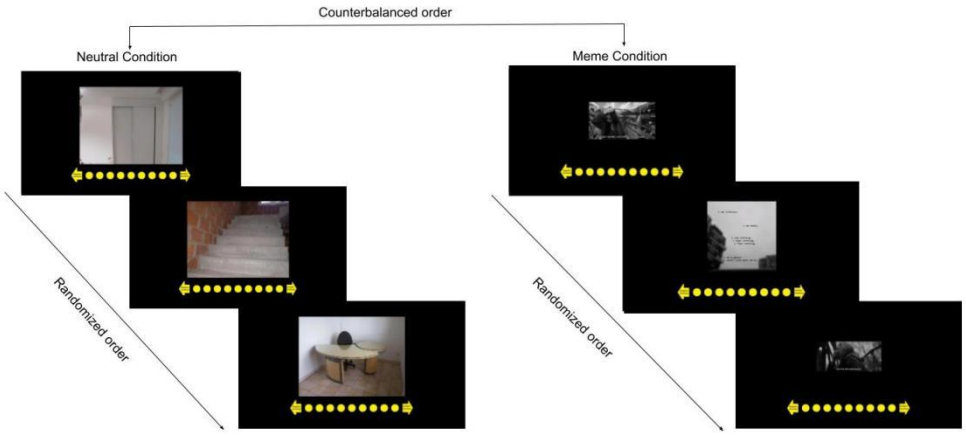


Figure 2.2. The figure illustrates the experimental procedure in Study 1. The left side displays the neutral condition, while the right side presents the meme condition. The order of conditions was counterbalanced, and the order of stimuli was randomized.

2.2.6. Statistical analysis

We used the Statistical Package for Social Sciences 23 (SPSS-23) (IBM Corporation, n.d.) to merge EEG and self-report data, and to create sum scores for each questionnaire subscale. For the main analysis, we used linear mixed effects models in R (R Software: A Tool Analysing Experimental Data, 2016), which handle repeated measures and nested data structures, minimizing Type 1 error (Baayen et al., 2008). This approach is robust to assumption violations and accommodates incomplete data (Schielzeth et al., 2020). Lastly, post hoc tests were performed for significant effects.

2.3. Results

The descriptive statistics for the main variables in the study are presented in Table 2.1.

Table 2.1.

Descriptive Statistics of the Main Variables in Study 1

Variables	*M*	Min	Max	*SD*
Neutral Condition				
Pre-depressive mood	13.68	8	40	7.17
Post-depressive mood	13.46	8	40	7.17
Meme Condition				
Pre-depressive mood	13.46	8	30	5.36
Post-depressive mood	15.18	8	38	7.83
Clarity	4.31	10	10	1.78
Goals	8.37	3	15	3.08
Impulse	6.15	3	15	3.35
Non-acceptance	6.59	3	15	3.46
Strategies	10.53	5	22	4.66
FAA EO	-0.04	-1.49	0.39	0.34
FAA EC	0.002	-0.21	0.37	0.10

Note. Abbreviations: FAA = Frontal alpha asymmetry; EO = Eyes-open; EC = Eyes-closed

Table 2.2 indicates that there was no carry-over effect between the sessions. Specifically, no significant difference was found between the pre-depressive mood levels in the neutral condition (M = 13.68, SD = 7.17) and the meme condition (M = 13.46, SD = 7.17); $t(31) = 0.19$, $p = .850$. This suggests that a washout period of at least one day between sessions was effective in returning participants to their baseline depressive mood levels. Furthermore, the table highlights a significant difference in the meme condition between pre-depressive mood (M = 13.46, SD = 5.36) and post-depressive mood (M = 15.18, SD = 7.83); $t(31) = -2.08$, $p = .045$, whereas no significant change was observed between the pre- (M = 13.68, SD = 7.17) and post-depressive mood (M = 13.46, SD = 7.17) levels in the neutral condition; $t(31) = 0.57$, $p = .567$, indicating successful manipulation.

Table 2.2.

Paired Samples T-test Results For Intervention and Carry-Over Effects in Study 1

Variables	*M*	*SD*	Lower *CI*	Upper *CI*	*t*	*df*	*p*
Neutral Condition							
Pre- Post-depressive mood	0.21	2.13	[-0.55]	[0.98]	0.57	31	.567
Meme Condition							
Pre- Post-depressive mood	-1.71	4.66	[-3.40]	[-0.03]	-2.08	31	.045*
Inter-condition							
(Neutral) Pre - (Meme) Pre-depressive mood	0.21	6.47	[-2.11]	[2.55]	0.19	31	.850
(Neutral) Post - (Meme) Post-depressive mood	-1.5	9.23	[-4.83]	[1.83]	-0.91	31	.365

Note. Significance level used = .05; Confidence level used: 0.95

As previously mentioned, a series of mixed-effects model analyses were conducted to examine the moderating role of emotion regulation in the relationship between depression memes and depressive mood. The results are summarized in Table 2.3.

The interaction between depressive mood and condition was significant for several models, including clarity, goals, and non-acceptance, while the time interactions were not. Specifically, depressive mood varied significantly based on the condition and its interaction with lack of emotional clarity, $F(1,90) = 8.09$, $p = .005$; difficulties in goal-directed behavior during emotional distress, $F(1,90) = 16.15$, $p = .001$; and difficulties in impulse control, $F(1,90) = 6.47$, $p = .012$. These findings indicate that depressive mood is moderated by maladaptive emotion regulation strategies when exposed to depression memes compared to neutral images, as shown in Figure 2.3. Although the results for non-acceptance of emotional responses and limited access to adaptive emotion regulation strategies were not statistically significant, the patterns depicted in Figure 2.4 show a similar trend.

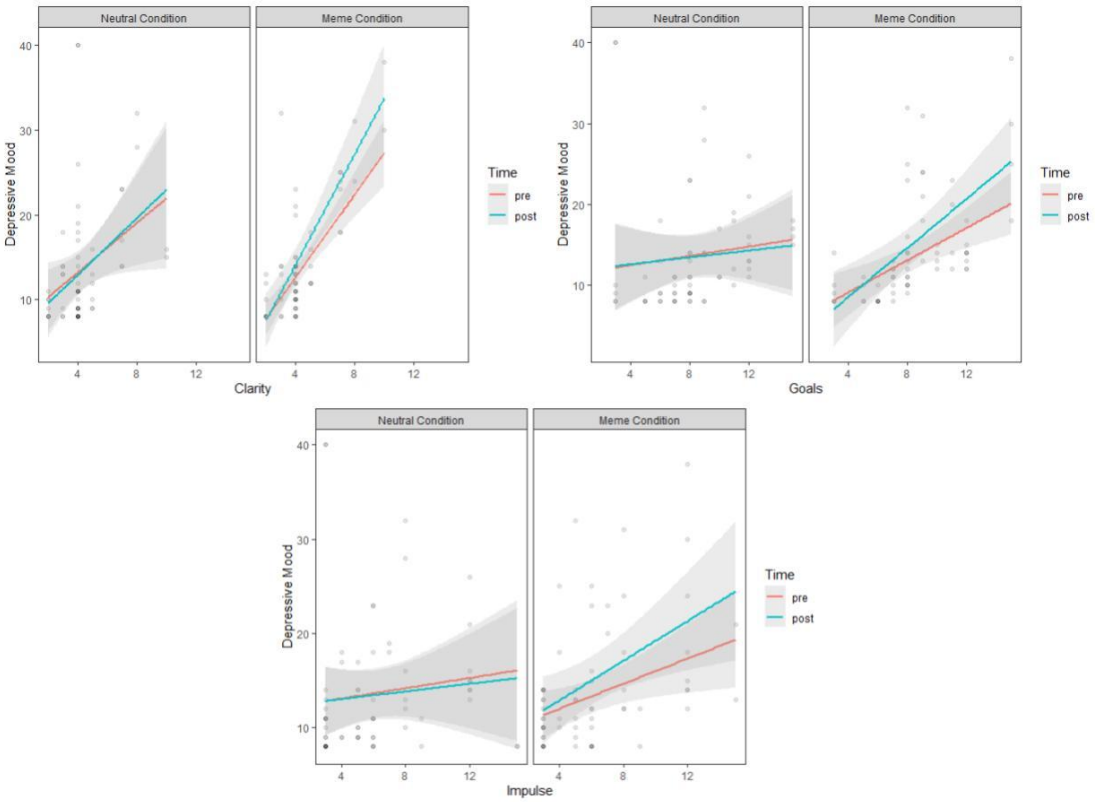


Figure 2.3. This figure illustrates the changes in depressive mood across the clarity, goals, and impulse subscales under both neutral and meme conditions.

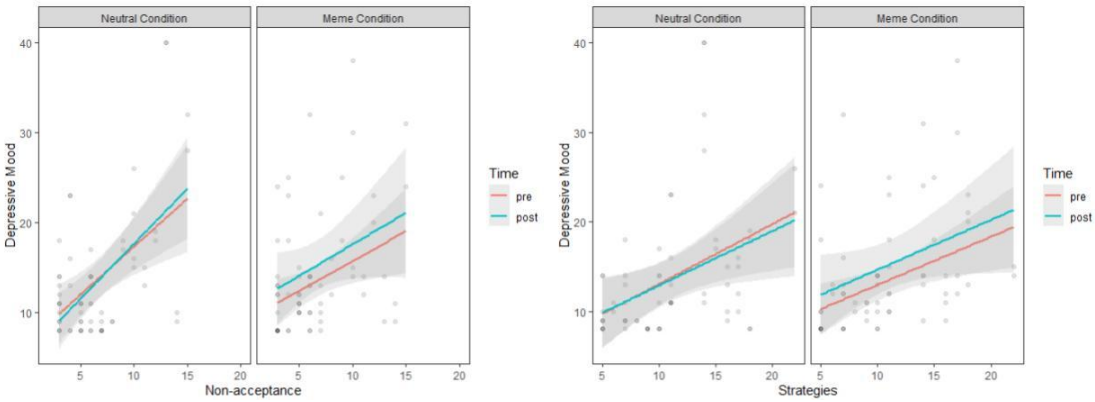


Figure 2.4. The figure depicts the changes in depressive mood across the non-acceptance and strategies subscales under both neutral and meme conditions.

Table 2.3.

Results of Linear Mixed-Effects Models for Self-Report Measurements in Study 1

Variables	Num *df*	Den *df*	*F*	*p*
Clarity	1	30	28.56	<.001*
Time	1	90	0.50	.48
Condition	1	90	5.19	.025*
Clarity x Time	1	90	1.32	.253
Clarity x Condition	1	90	8.09	.005*
Clarity x Time x Condition	1	90	0.45	.500
Goals	1	30	6.15	.018*
Time	1	90	0.24	.618
Condition	1	90	11.86	<.001*
Goals x Time	1	90	0.78	.377
Goals x Condition	1	90	16.15	<.001*
Goals x Time Condition	1	90	1.41	.237
Impulse	1	30	3.54	.069
Time	1	90	0.01	.894
Condition	1	90	3.24	.074
Impulse x Time	1	90	0.42	.517
Impulse x Condition	1	90	6.47	.012*
Impulse x Time Condition	1	90	0.86	.354
Non-acceptance	1	30	13.85	<.001*
Time	1	90	0	.956
Condition	1	90	4.56	.035*
Non-acceptance x Time	1	90	0.16	.682
Non-acceptance x Condition	1	90	3.73	.056
Non-acceptance x Time x Condition	1	90	0.06	.803
Strategies	1	30	9.47	.004*
Time	1	90	0.20	.652
Condition	1	90	0.20	.652
Strategies x Time	1	90	0.01	.909
Strategies x Condition	1	90	3.73	.056
Strategies x Time x Condition	1	90	0.04	.830

Note. Dependent variable = Depressive mood; Significance level used = .05

We conducted a series of Tukey-adjusted pairwise comparisons to further examine the significance of the slopes for clarity, goals, and impulse control. The results are presented in Table 2.4.

Table 2.4.

Pairwise Comparison Results for Clarity, Goals, and Impulse Slopes by Condition in Study 1

Variables	Trend	*SE*	Lower *CI*	Upper *CI*	Estimate	*t*	*p*
Clarity							
Neutral Condition	1.56	0.47	[0.61]	[2.51]			
Meme Condition	2.85	0.47	[1.90]	[3.80]			
Contrast					-1.29	-2.84	.005*
Goals							
Neutral Condition	0.24	0.32	[-0.41]	[0.91]			
Meme Condition	1.26	0.32	[0.59]	[1.92]			
Contrast					-1.01	-4.02	<.001*
Impulse							
Neutral Condition	0.23	0.31	[-0.40]	[0.87]			
Meme Condition	0.85	031	[0.22]	[1.49]			
Contrast					-0.62	-2.54	.012*

Note. Significance level used = .05 Confidence level used = 0.95

Table 2.5 displays the linear mixed-effects model results for the impact of depression memes, compared to neutral images, on depressive mood, and the moderating role of frontal alpha asymmetry. Similar to the results for non-acceptance and emotion regulation strategies, the effects were not significant but showed a comparable trend, as depicted in Figure 2.5, relative to the other self-reported covariates.

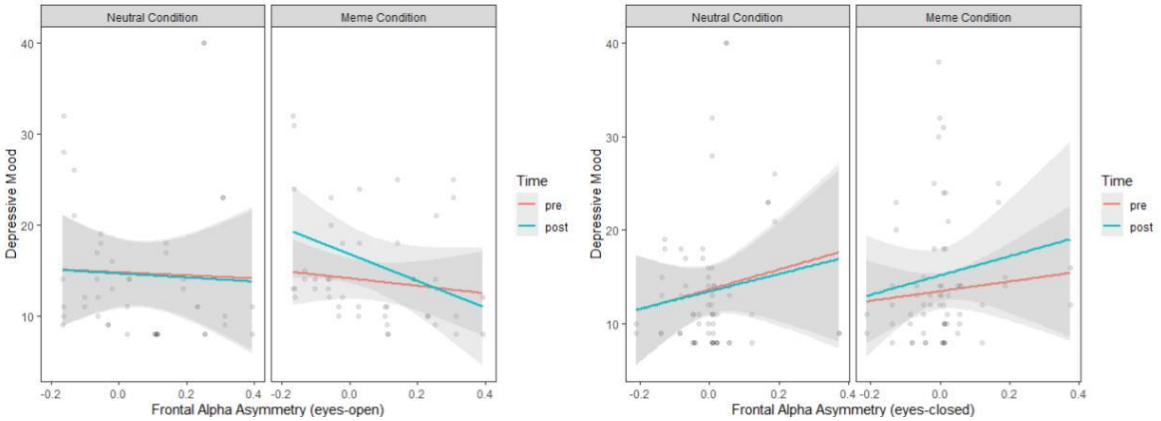


Figure 2.5. The figure shows the changes in depressive mood based on frontal alpha asymmetry scores under both neutral and meme conditions.

Table 2.5.

Results of Linear Mixed-Effects Models for Frontal Alpha Asymmetry in Study 1

	Num	**Den**		
Fixed Effects	***df***	***df***	***F***	***p***
FAA EO	1	26	0.27	.607
Time	1	78	0.46	.496
Condition	1	78	0	0.98
FAA EO x Time	1	78	0.07	.787
FAA EO x Condition	1	78	0.47	.490
FAA EO x Time x Condition	1	78	0.13	.716
FAA EC	1	30	0.86	.359
Time	1	90	0.78	.376
Condition	1	90	0.81	.369
FAA EC x Time	1	90	0.05	.811
FAA EC x Condition	1	90	0.07	.782
FAA EC x Time Condition	1	90	0.16	.685

Note. Abbreviations: FAA = Frontal alpha asymmetry; EO = Eyes-open; EC = Eyes-closed; Dependent Variable = Depressive mood; Significance level used = .05; 4 participants were excluded from the FAA EO model because of missing data.

2.4. Discussion

In the context of online media consumption, self-regulation is essential for managing emotions, whether to enhance positive states or diminish negative ones (Larose et al., 2001). This study explored the impact of exposure to depression memes on social media, specifically examining their effects on depression-related symptoms (e.g., sadness,

hopelessness, and worthlessness) and the moderating role of frontal alpha asymmetry and self-report measurements of emotion regulation.

Our findings revealed that exposure to depression memes, compared to neutral images, had a stronger negative effect on individuals with a higher maladaptive emotion regulation strategies, such as lack of emotional clarity, difficulties in goal-directed behavior under emotional stress, and impulse control issues. These results are consistent with previous research showing that behavioral components are central to various emotion regulation problems and related mental health disorders (Tice et al., 2001; Zhang et al., 2020). Further analysis indicated that better emotion regulation skills might mitigate the adverse effects of depression memes, suggesting that the impact on negative mood may be moderated by one's ability to apply adaptive strategies like positive reappraisal.

We also assessed frontal alpha asymmetry as a potential neural marker of emotion regulation and its role in the relationship between exposure to depression memes and negative mood. Although no significant effects were found, this may be due to a homogeneous sample of young, educated participants, which could have restricted variability in frontal alpha asymmetry patterns.

Despite the lack of statistical significance, our findings suggest that specific patterns in alpha asymmetry may still be relevant, aligning with previous studies indicating that higher asymmetry scores reflect lower withdrawal motivation and increased sensitivity to negative stimuli (Disner et al., 2011; Garcia-Martin et al., 2021; Gotlib & Joormann, 2010).

Frontal alpha asymmetry, particularly when the eyes were closed, showed a pattern similar to self-reported maladaptive strategies such as difficulties in goal-directed behavior and impulse control, indicating lower inhibitory control. However, higher frontal alpha asymmetry scores may also reflect reduced left frontal cortical alpha activity, suggesting a higher approach motivation to positive stimuli. Conversely, EO results revealed that lower asymmetry scores, indicative of higher withdrawal tendencies or inhibitory control, may be correlated with higher depressive mood after exposure to depression memes compared to neutral images, consistent with the

dominant research findings in the field (Coan & Allen, 2004; Harmon-Jones et al., 2010).

However, these results are not statistically significant and should be considered cautiously. Our findings highlight the complexity of relationships between frontal alpha asymmetry, emotion regulation, and mood changes, which may explain the inconsistencies reported in prior studies (Coan & Allen, 2004; Jesulola et al., 2015).

There are several limitations to our study. First, the sample consisted solely of young, healthy adults, limiting the generalization of the findings. Future research should include clinically depressed individuals or employ more comprehensive assessments of depressive symptoms. The short-term nature of our design (immediate pre- and post-exposure assessments) should be expanded to consider potential long-term effects of repeated exposure, given social media algorithms that can perpetuate such content. Addressing these limitations could enhance the validity and applicability of findings in this area.

The discrepancy between self-report measurements and frontal alpha asymmetry within the study led us to consider the relationship between them. Thus, in Study 2, we investigated this relationship specifically.

3. Study 2 - The relationship between frontal alpha asymmetry and self-report measurements of depression, anxiety, stress, and self-regulation[4]

3.1. Introduction

Self-report measures, such as questionnaires, are widely used in psychological research to capture subjective experiences such as emotions, thoughts, motivations, and attitudes. While these methods are important for accessing private mental states and capabilities, they are not without limitations. Self-report bias, arising from factors such as social desirability and response tendencies, can distort data and obscure true psychological processes (Bauhoff, 2011; Rosenman et al., 2011; Gorber & Tremblay, 2016). To address these limitations, researchers have turned to other direct measures, such as electrophysiological techniques, to complement self-reports. Previous research using both self-report and electrophysiological measures showed mixed results in different emotional processes (Elis et al., 2017; Kinney et al., 2019).

Building on the literature, we initiated our investigation into the relationship between self-regulation and frontal alpha asymmetry by comparing findings from these two complementary approaches. We utilized the Short Self-Regulation Questionnaire (SSRQ) (Carey et al., 2004), a well-established and widely used tool for assessing self-regulation. Despite its popularity and extensive application in psychological research, the compatibility of the SSRQ with electrophysiological measures such as frontal alpha asymmetry has not been thoroughly investigated. This gap in the literature provided a unique opportunity to explore whether frontal alpha asymmetry could serve as a reliable correlate or reflection of self-reported measures of self-regulation obtained through the SSRQ. Therefore, our main objective was to assess the extent to which the SSRQ captures self-regulatory processes that are also measurable through frontal alpha asymmetry, bridging the gap between subjective self-reports and neural indicators.

---

[4] Please refer to the published paper for further information and supplementary materials: Akil, A. M., Watty, M., Cserjesi, R., & Logemann, H. N. A. (2024). The relationship between frontal alpha asymmetry and self-report measurements of depression, anxiety, stress, and self-regulation. *Applied Neuropsychology: Adult*, 1–7. https://doi.org/10.1080/23279095.2024.2425361

Additionally, this research was conducted as a cohort study, allowing us to explore not only the primary focus on self-regulation but also the broader relationships between frontal alpha asymmetry and emotional states such as depression, anxiety, and stress. To achieve this, we employed the Depression, Anxiety and Stress Scale (DASS) (Lovibond and Lovibond, 1995), a validated and widely used instrument designed to measure the severity of these mental health conditions. To our knowledge, the relationship between frontal alpha asymmetry and the Depression Anxiety Stress Scales-21 (DASS-21) (Crawford & Henry, 2005) remains relatively underexplored, particularly across diverse conditions and extended time frames.

Previous studies provide some insights into this area: Kemp et al. (2010) reported reduced left-frontal activity in individuals with moderate to severe depression. Similarly, Mathersul et al. (2008) found increased right-lateralized activity in individuals with anxiety compared to those with depression, using the original version of the DASS during a 2-minute resting-state eyes-closed paradigm. Beaton et al. (2008) further demonstrated that self-reported shyness predicted heightened relative right-frontal EEG asymmetry, but this effect was only evident after controlling for depressive mood. Their protocol included one minute of EO and one minute of EC conditions. To address these gaps in the literature, we adopted a more comprehensive approach by collecting frontal alpha asymmetry data over longer duration under both EO and EC resting-state conditions.

We hypothesized that greater dispositional right-frontal activity would be associated with higher depression and anxiety scores on the DASS-21, while greater left-frontal activity would correlate with lower scores. Specifically, given the inverse relationship between alpha wave activity and cortical activity, we expected that lower dispositional frontal alpha asymmetry scores (indicating relatively higher right-frontal activity) would correspond to higher levels of depression and anxiety. Similarly, we expected a correlation between the SSRQ and dispositional frontal alpha asymmetry scores.

3.2. Methods

3.2.1. Participants

We used data from 130 participants. Of these, 83 (64%) were female. Ages ranged from 18 to 58, with a mean age of 25.2 years, SD_{age} = 6.8. Eligibility criteria required participants to be at least 18 years old and to pass a screening process excluding individuals with clinical diagnoses, frequent headaches or migraines, epilepsy, significant past head trauma, recent head injuries, chronic skin conditions, or current drug use. Participants were also instructed to abstain from smoking and consuming caffeine for at least two hours before the experiment. The study adhered to the ethical principles of the Declaration of Helsinki and its later amendments, with approval from the IRB of Eötvös Loránd University.

3.2.2. Questionnaires

The DASS is a self-report instrument developed by Lovibond and Lovibond (1995) to measure depression, anxiety, and stress independently. The shortened DASS-21 consists of 21 items distributed across the three scales (Henry & Crawford, 2005). Each item is scored on a 4-point Likert scale ranging from 0 ("Never") to 3 ("Almost Always"). For consistency with the original 42-item version, DASS-21 scores are multiplied by two. Higher scores indicate more severe symptoms in the respective domains. The internal consistency of the DASS-21 has been well-documented, with Cronbach's alpha coefficients of 0.88 for the Depression scale, 0.82 for the Anxiety scale, and 0.90 for the Stress scale (Henry & Crawford, 2005). Brown et al. (1999) originally developed the Self-Regulation Questionnaire (SRQ), a 63-item self-report tool designed to evaluate self-regulatory processes. Subsequently, the SRQ was refined into the Short Self-Regulation Questionnaire (SSRQ) (Carey et al., 2004). The SSRQ is a condensed, single-factor version consisting of 31 items, aimed at maintaining the validity of the original while increasing practicality. Participants rate each item on a 5-point Likert scale ranging from 1 ("Strongly Disagree") to 5 ("Strongly Agree"). Fourteen of the items are reverse-scored. The total score, calculated by summing the responses, serves as the outcome measure, with higher scores reflecting stronger self-regulatory abilities.

The SSRQ has demonstrated excellent internal consistency, with a Cronbach's alpha coefficient of 0.92 reported for the 31-item scale (Carey et al., 2004).

3.2.3. Frontal alpha asymmetry

The electrophysiological data collection, preprocessing procedures, and frontal alpha asymmetry calculations were conducted using the same methods and facilities as described in Study 1. In sum, scalp voltage was recorded using a 21-channel EEG cap with Ag/AgCl electrodes, adhering to the 10-20 international system for electrode placement. Data acquisition was performed with the NeXus-32 system (Mind Media; Nexus-32, n.d.), at a sampling rate of 512 Hz with a common average reference. VEOG signals were recorded using electrodes positioned above and below the left eye, while HEOG signals were captured from electrodes placed at the outer corners of both eyes. EEG data was re-referenced offline to linked mastoids for improved signal accuracy. Resting-state EEG data was collected over a 10-minute session, comprising five minutes in an EO condition and five minutes in an EC condition. The data was preprocessed following an established paradigm (Smith et al., 2017), as discussed in Chapter I, to ensure consistency and reliability. Frontal alpha asymmetry scores were computed by log-transforming alpha power values and subtracting those from the left-lateralized electrode sites from their right-lateralized counterparts (F4-F3/F8-F7).

3.2.4. Procedures

Upon arrival at the laboratory, participants were provided with an information letter outlining the study procedures and eligibility criteria. After confirming eligibility based on these criteria, participants signed an informed consent form. EEG electrodes were then applied to the scalp, and resting-state EEG data were recorded in two 5-minute sessions: one with EO and the other with EC. After the EEG recording, participants completed a series of questionnaires. The entire procedure lasted approximately 30 minutes, although participants remained in the laboratory for additional assessments as part of the larger cohort study.

3.2.5. Statistical analysis

Data analyses were conducted using JASP (JASP Team, 2024). Key variables were computed first, followed by correlation analyses examining the relationships between frontal alpha asymmetry, depression, anxiety, stress, and self-regulation scores. To further explore these relationships, we applied the method used by Kemp et al. (2010), narrowing the analysis to participants with moderate to extreme levels of depression, anxiety, or stress. Specifically, individuals were included if they scored ≥14 on the depression subscale, ≥10 on the anxiety subscale, or ≥19 on the stress subscale. A second round of correlation analyses was performed within this subset. Both frequentist and Bayesian approaches were employed. For the frequentist analysis, a significance threshold of .05 was used, while Bayes factors (BF_{10}) were calculated to assess the strength of the evidence in the Bayesian framework.

3.3. Resutls

Table 3.1 provides an overview of the descriptive statistics for the main variables in the study.

Table 3.1.

Descriptive Statistics Regarding the Main Variables in Study 2

Variables (*n* = 130)	Min	Max	*M*	*SD*
FAA (F4-F3/EO)	-1.49	0.63	<0.01	0.22
FAA (F4-F3/EC)	-0.26	0.37	<0.01	0.09
FAA (F8-F7/EO)	-1.03	1.3	-0.02	0.35
FAA (F8-F7/EC)	-0.96	0.49	-0.03	0.17
Depression	0	28	8.76	6.49
Depressed participants (*n* = 26)	14	28	19.38	4.51
Anxiety	0	28	8.8	6.24
Anxious participants (*n* = 56)	10	28	14.82	4.08
Stress	0	30	13.72	6.69
Stressed participants (*n* = 30)	20	30	23.2	3.04
Self-regulation	64	147	114	14.45

Note. Abbreviations: FAA = Frontal alpha asymmetry, EO = Eyes-open, EC = Eyes-closed. Depressed, anxious, and stressed participants were identified using the method outlined by Kemp et al. (2010).

We conducted a correlation analysis using the full sample, with results summarized in Table 3.1. The analysis revealed several moderate positive correlations between

electrode sites and conditions. Notably, frontal alpha asymmetry during the EC condition exhibited significant correlations across electrode sites, particularly at F4-F3 and F8-F7 ($r = 0.554$, $p < .001$, $BF_{10} = 56580…$).

Table 3.1.

Correlations Analysis Results for Frontal Alpha Asymmetry, Depression, Anxiety, Stress, and Self-Reported Self-Regulation in Study 2

Variables (n = 130)									
FAA (F4-F3/EO)		1							
FAA (F4-F3/EC)	Pearson's r	0.31	1						
	p-value	< .001*							
	BF_{10}	47.088							
FAA (F8-F7/EO)	Pearson's r	0.201	0.168	1					
	p-value	.025*	.063						
	BF_{10}	1.355	0.619						
FAA (F8-F7/EC)	Pearson's r	0.124	0.554	0.296	1				
	p-value	.172	< .001*	< .001*					
	BF_{10}	0.284	5.658e+8	26.768					
Depression	Pearson's r	-0.036	-0.095	0.1	0.011	1			
	p-value	.69	.288	.267	.903				
	BF_{10}	0.121	0.195	0.205	0.112				
Anxiety	Pearson's r	-0.149	-0.013	0.08	0.026	0.372	1		
	p-value	.097	.889	.373	.775	< .001*			
	BF_{10}	0.435	0.113	0.165	0.116	1313.663			
Stress	Pearson's r	-0.025	0.06	0.009	0.069	0.469	0.526	1	
	p-value	.78	.507	.924	.442	< .001*	< .001*		
	BF_{10}	0.116	0.139	0.112	0.149	697835.224	8.369e+7		
Self-regulation	Pearson's r	-0.046	0.073	-0.04	0.026	-0.519	-0.115	-0.242	1
	p-value	.61	.417	.661	.776	< .001*	.192	0.006*	
	BF_{10}	0.127	0.154	0.123	0.116	4.356e +7	0.254	4.945	

Note. Abbreviations: FAA = Frontal alpha asymmetry, EO = Eyes-open, EC = Eyes-closed. Significance level used: .05.

Consistent with the study's expectations, moderate positive correlations were also detected among depression, anxiety, and stress scores. For instance, depression was positively associated with anxiety (r = 0.372, $p < .001$, $BF_{10} = 1313.66$), and anxiety showed a similar relationship with stress (r = 0.526, $p < .001$, $BF_{10} = 8369000…$). Additionally, the analysis revealed negative correlations between depression and self-regulation (r = -0.519, $p < .001$, $BF_{10} = 4356…$) and between stress and self-regulation (r = -0.242, $p = .006$, $BF_{10} = 4.945$). These findings highlight meaningful interactions between emotional states and self-regulatory capabilities.

Following the approach of Kemp et al. (2010), the analysis was refined to focus exclusively on individuals with moderate to extreme levels of depression, anxiety, or stress. These subsample results are detailed in Tables 3.2, 3.3, and 3.4, corresponding to the depression, anxiety, and stress groups, respectively.

Table 3.2.

Correlation Analysis Results for Individuals Experiencing Moderate to Extreme Levels of Depression

Variables (n = 26)							
FAA (F4-F3/EO)		1					
FAA (F4-F3/EC)	Pearson's r	0.224	1				
	p-value	.304					
	BF_{10}	0.425					
FAA (F8-F7/EO)	Pearson's r	-0.274	-0.002	1			
	p-value	.206	.992				
	BF_{10}	0.55	0.259				
FAA (F8-F7/EC)	Pearson's r	0.031	0.813	0.102	1		
	p-value	.887	< .001*	.642			
	BF_{10}	0.261	24239.545	0.286			
Depression	Pearson's r	0.261	0.044	0.05	0.095	1	
	p-value	.229	.834	.819	.653		
	BF_{10}	0.512	0.253	0.265	0.273		
Self-regulation	Pearson's r	-0.306	0.17	0.077	-0.022	-0.457	1
	p-value	.156	.417	.726	.916	.019*	
	BF_{10}	0.668	0.339	0.274	0.249	3.314	

Note. Pair-wise correlation was used in the analysis.

Table 3.3.

Correlation Analysis Results for Individuals Experiencing Moderate to Extreme Levels of Anxiety

Variables (n = 56)							
FAA (F4-F3/EO)		1					
FAA (F4-F3/EC)	Pearson's r	0.2	1				
	p-value	.147					
	BF_{10}	0.472					
FAA (F8-F7/EO)	Pearson's r	0.013	-0.094	1			
	p-value	.923	.498				
	BF_{10}	0.169	0.212				
FAA (F8-F7/EC)	Pearson's r	0.037	0.807	0.05	1		
	p-value	.79	< .001*	.718			
	BF_{10}	0.176	8.17E+10	0.181			
Anxiety	Pearson's r	-0.365	-0.068	0.196	0.039	1	
	p-value	.006*	.623	.151	.776		
	BF_{10}	6.551	0.189	0.459	0.175		
Self-regulation	Pearson's r	-0.108	0.174	-0.117	0.111	0.028	1
	p-value	.434	.203	.394	.42	.84	
	BF_{10}	0.227	0.37	0.24	0.231	0.17	

Note. Pair-wise correlation was used in the analysis.

Table 3.4.

Correlation Analysis Results for Individuals Experiencing Moderate to Extreme Levels of Stress

Variables (n = 30)							
FAA (F4-F3/EO)		1					
FAA (F4-F3/EC)	Pearson's r	0.401	1				
	p-value	.035*					
	BF_{10}	1.967					
FAA (F8-F7/EO)	Pearson's r	0.323	-0.133	1			
	p-value	.088	.501				
	BF_{10}	0.925	0.291				
FAA (F8-F7/EC)	Pearson's r	0.121	0.39	0.151	1		
	p-value	.539	.037*	.444			
	BF_{10}	0.281	1.853	0.31			
Stress	Pearson's r	-0.049	-0.231	-0.182	0.109	1	
	p-value	.799	.228	.344	.572		
	BF_{10}	0.238	0.462	0.353	0.269		
Self-regulation	Pearson's r	-0.066	0.31	-0.178	0.082	-0.421	1
	p-value	.733	.102	.355	.674	.021*	
	BF_{10}	0.244	0.827	0.347	0.251	2.936	

Note. Abbreviations: FAA = Frontal alpha asymmetry; EO = Eyes-open; EC = Eyes-closed, Pair-wise correlation was used in the analysis.

In the depression subsample, a strong positive correlation was observed between frontal alpha asymmetry during the EC condition at the F4-F3 and F8-F7 electrode sites (r = 0.813, $p < .001$, BF_{10} = 24239.545). Additionally, depression was moderately negatively correlated with self-regulation (r = -0.457, p = .019, BF_{10} = 3.314), further supporting the inverse relationship between depressive symptoms and self-regulatory abilities. For participants with moderate to extreme anxiety, a similarly strong positive correlation emerged between frontal alpha asymmetry during the EC condition at the F4-F3 and F8-F7 sites (r = 0.807, $p < .001$, BF_{10} = 817…). A noteworthy finding in this group was the moderate negative correlation between anxiety scores and frontal apha asymmetry during the EO condition at the F4-F3 site (r = -0.365, p = .006, BF_{10} = 6.551), indicating that anxiety may modulate frontal alpha asymmetry differently across conditions. In the stress subsample, a moderate positive correlation was identified between frontal alpha asymmetry during the EC condition at the F4-F3 and F8-F7 electrode sites (r = 0.39, p

= .037, BF_{10} = 1.853). Additionally, stress exhibited a moderate negative correlation with self-regulation (r = -0.421, p = .021, BF_{10} = 2.936), further emphasizing the potential impact of heightened stress on self-regulatory capabilities.

3.4. Discussion

In this study, we investigated the relationship between frontal alpha asymmetry, a potential electrophysiological marker for self-regulation and depression, and self-reported measures of depression, anxiety, stress, and self-regulation. To address gaps in the existing literature, we utilized a comprehensive resting-state frontal alpha asymmetry procedure that involved both EO and EC conditions for extended periods (five minutes each). We assessed participants' psychological states using the DASS-21 and the SSRQ.

Our findings were largely consistent with previous research, revealing several positive correlations among depression, anxiety, and stress as measured by the DASS-21. These results confirm the overlap in the concepts and mechanisms underlying these psychological phenomena, as noted by Eysenck & Fajkowska (2018) and Cole et al. (2001). Additionally, we observed a negative correlation between self-regulation and depression, suggesting that poor self-regulation may contribute to or exacerbate depression. This finding supports prior research indicating that compromised self-regulatory abilities are associated with poorer mental health outcomes (Johnstone et al., 2007).

We also identified a significant relationship between frontal alpha asymmetry at the F4-F3 and F8-F7 electrode sites during the eyes-closed condition, which suggests that this measure may be particularly reliable across these specific topographical sites. This highlights the importance of considering sensory input states when evaluating neural correlates of psychological states.

While some previous research has shown inconsistencies regarding the relationship between frontal alpha asymmetry and mood regulation, it is worth noting that these effects may vary based on the sample. For example, Kemp et al. (2010) refined their analysis to focus on individuals with moderate to extreme levels of depression, anxiety,

and stress, and our findings similarly benefit from focusing on participants with more pronounced psychological symptoms. In line with this approach, we observed that frontal alpha asymmetry during the EC condition was correlated with anxiety scores. Specifically, we observed a moderate negative correlation between frontal alpha asymmetry and anxiety scores. This suggests that lower frontal alpha asymmetry, indicative of greater right frontal activation, may be linked to higher levels of anxiety, which aligns with existing literature on the dominance of right frontal activity in anxious individuals (Mathersul et al., 2008).

However, while no definitive associations were found between frontal alpha asymmetry and depression in our study, prior research has produced mixed results. Some studies, like Van der Vinne et al. (2017), have found negligible effects, while others, such as Kaiser et al. (2018), have pointed to challenges in generalizing across different methodologies and sample characteristics. Moreover, while some studies suggest that depression is linked to right cortical asymmetry, it remains unclear whether this association is specific to the disorder or reflects a broader symptom profile (Fingelkurts & Fingelkurts, 2015).

Our findings may indicate that the observed effects were primarily related to behavioral self-regulation, with less emphasis on affective regulation. This suggests that future studies should examine different dimensions of self-regulation to better understand the complex relationships. Previous research has shown that depression and anxiety can affect different age groups, genders, and cultural backgrounds in distinct ways (Christensen et al., 1999; Salk et al., 2017). These variables, along with factors like childhood chronic diseases or low income in older populations, can influence mental health outcomes (Farhane-Medina et al., 2022; Schaakxs et al., 2017). This limits the generalizability of our findings, and future studies should aim to include more diverse samples to enhance the external validity of the results. Additionally, heterogeneity in research designs and gender-based differences in symptom expression (Cavanagh et al., 2017) should be considered in future investigations. Furthermore, the cross-sectional design of our study prevents us from drawing conclusions about causal relationships between the variables. Although correlations were observed, they do not imply causation, and longitudinal and experimental studies are needed to better understand the directionality of these relationships over time. Another limitation is that our study

focused primarily on frontal alpha asymmetry as an EEG index and did not consider other neurophysiological markers or brain regions that may be relevant to depression, anxiety, stress, and self-regulation. For example, a network study found coherence in the alpha frequency band among individuals with depression, which challenges studies showing differences in frontal alpha power (Leuchter et al., 2012). Conversely, a recent review found no robust relationship between brain structure abnormalities and depression (Scheepens et al., 2020). Exploring a broader range of neurobiological markers would provide a more comprehensive understanding of the mechanisms involved in these psychological processes. Finally, our study did not examine specific self-regulation strategies, which could be an important factor in understanding the relationship between frontal alpha asymmetry and mental health. Future research could explore how frontal alpha asymmetry correlates with specific self-regulation strategies, such as rumination, through self-report measures. While our study contributes to the existing literature on the relationship between frontal alpha asymmetry, depression, anxiety, stress, and self-regulation, these limitations highlight areas for future research that could further clarify these relationships and their implications for mental health interventions.

A potentially more effective approach for understanding the relationship between frontal alpha asymmetry and self-regulatory processes would be to directly modulate brain activity in experimental settings. By manipulating frontal alpha asymmetry through interventions such as muscle contraction or transcranial direct current stimulation, we could observe how changes in neural activity influence self-regulation, particularly in relation to approach and avoidance tendencies. This experimental modulation would allow for a clearer understanding of the causal direction between neural activity and self-regulation. Following such manipulation, self-regulatory processes could be evaluated through behavioral tasks that assess participants' approach-avoidance tendencies, cognitive control, and emotion regulation. In Study 3 and Study 4, we addressed these questions.

4. Study 3 - The effect of unilateral hand muscle contraction on frontal alpha asymmetry and inhibitory control in intrinsic reward contexts, a randomized controlled trial[5]

4.1. Introduction

Previous studies have suggested that heightened right frontal cortical activity relative to the left may reflect greater inhibitory control (Vallortigara & Rogers, 2005). Impairments in this capacity have been linked to various psychological conditions, including obesity (de Klerk et al., 2023) where challenges in regulating behavior in response to reward-related stimuli, often linked to increased approach motivation, are particularly evident.

Several neuromodulation techniques, such as transcranial electric stimulation (tES) and EEG-neurofeedback, have been employed to manipulate activity in the DLPFC and frontal brain asymmetry in both clinical and non-clinical populations (Kekic et al., 2017; Yadollahpour & Jalilifar, 2019). Unilateral muscle contraction (UMC), such as squeezing one hand for a prolonged period, is another method that has shown potential to modulate frontal asymmetry by activating the contralateral hemisphere, thereby impacting approach and avoidance tendencies (Dru & Cretenet, 2008; Harmon-Jones, 2006; Schiff & Lamon, 1994). However, its effects on frontal alpha asymmetry and the exact electrophysiological markers (Stop N2 and Stop P3) of inhibition remain unexplored in detail.

Therefore, this study investigated the effects of UMC on frontal alpha asymmetry and inhibitory control, particularly in the context of intrinsic reward, specifically food-reward contexts, which is known to activate left frontal cortical regions due to the implicit reward value associated with high-calorie and sweet food. Previous research in the topic of inhibitory control indicates that healthy individuals show a preference for food rewards over monetary rewards, as the latter are considered extrinsic and/or learned rewards (Tsegaye et al., 2022).

---

[5] Please refer to the published paper for further information and supplementary materials: Akil, A. M., Cserjési, R., Nagy, T. Demetrovics, Z., & Logemann, H. N. A. (2024). The effect of unilateral hand muscle contraction on frontal alpha asymmetry and inhibitory control in intrinsic reward contexts, a randomized controlled trial. *Scientific Reports,* 14, 27289. https://doi.org/10.1038/s41598-024-74070-8

We aimed to investigate the impact of left-hand UMC on frontal alpha asymmetry and inhibitory control using the Stop Signal Task, which facilitates the measurement of response inhibition through behavioral and event-related potential analyses (Kenemans et al., 2023; Logemann et al., 2013).

We hypothesized that active contraction of the left hand would increase right relative to left frontal cortical activity, resulting in a lower frontal alpha asymmetry score, indicating enhanced inhibitory control. We expected that left-hand UMC would result in enhanced inhibitory control, as evidenced by shorter stop-signal reaction times (SSRTs) and increased amplitudes of the Stop N2 and Stop P3 in the reward condition compared to a neutral condition. These effects would suggest that left-hand UMC, by increasing right frontal activity, could improve the ability to suppress responses to rewarding stimuli, potentially serving as a viable intervention for populations with self-regulation problems.

4.2. Methods

4.2.1. Participants

We initially conducted a pilot test with 10 participants to validate the experimental procedure and estimate the necessary sample size. Using G*Power software (Faul et al., 2007), a power analysis was performed to determine the sample size needed to detect changes in the primary outcome variable, frontal alpha asymmetry, before and after unilateral left-hand muscle contraction. The analysis considered an 80% statistical power, a 5% significance level, and a test-retest correlation of 0.6 for the time effect, predicting a detectable effect size of $f > 0.237$ ($\eta_p^2 > 0.053$). Based on these parameters, a minimum of 30 participants was required for the intervention group. A total of 65 individuals (37 females, 28 males, 61 right-handed participants, $M_{age} = 26.6$, $SD_{age} = 7.4$) were recruited for the study through social media and university courses. Handedness was not considered an exclusion criterion because prior research suggests it does not impact inhibitory control performance (Mancini & Mirabella, 2021). Participants needed to meet the following eligibility criteria: being at least 18 years old, and passing a screening for exclusion factors such as the presence of psychological or psychiatric disorders, frequent headaches or migraines, epilepsy, significant prior head trauma,

recent head injuries, chronic skin conditions, or current drug use. They were also required to avoid smoking and drinking coffee for at least two hours before the experiment. PsyToolkit (Stoet, 2010, 2017) was used for questions. The research received approval from the IRB of Eötvös Loránd University. Participants received either a voucher or course credit as compensation for their involvement in the study.

4.2.2. Stop signal task

The Stop Signal Task was designed using OpenSesame (Mathôt et al., 2012) and was adapted from its original version (Schmajuk et al., 2006) to include a food reward condition based on insights from prior studies (Houben et al., 2014; Tsegaye et al., 2022). The experimental design incorporated both neutral and reward conditions to assess inhibitory control in challenging situations. Figure 4.1.A. illustrates the task. The task began with a practice block followed by four experimental blocks, each containing 128 trials, 96 go trials (75%), and 32 stop trials (25%). Thus, each condition comprised a total of 512 trials, and across both the pre- and post-intervention phases, participants completed 1024 trials. This resulted in 2048 trials for the entire experiment, excluding practice blocks.

Participants were seated approximately 65 cm from the screen and received both written and oral instructions before starting. Each trial started with a fixation dot displayed at the center of the screen for 2000 ms to capture their attention. In the reward condition, images of palatable foods such as cookies, chips, chocolate, and nuts were presented as go stimuli. Each image measured 115 pixels in width (2.9°) and 200 pixels in height (5.1°) and appeared randomly in horizontal or vertical orientations for 150 ms. Participants were instructed to respond using their left or right index fingers according to the orientation of the food image. In the neutral condition, the go stimuli consisted of the letters "X" or "O," each measuring 200 pixels in both width and height (5.1°), presented for 150 ms, and requiring the same left or right index finger response based on the letter displayed.

The stop trials featured the infrequent presentation of the letter "S," which appeared following a go stimulus and required participants to withhold their response. The initial stimulus onset asynchrony (SOA) was fixed at 350 ms, but it dynamically adjusted after each stop trial. If a participant failed to inhibit their response, the SOA decreased by 50

ms; if they succeeded, it increased by 50 ms. This tracking algorithm aimed to optimize the reliability of the stop-signal reaction time (Verbruggen et al., 2013), which is a primary measure of inhibitory control derived from the Stop Signal Task (de Jong et al., 1990; De Jong et al., 1995). In line with Verbruggen et al. omissions were replaced by the maximum reaction times (RTs) of 1500 ms (Verbruggen et al., 2013). SSRT is calculated by subtracting the mean stop-signal delay from the go reaction time, providing a quantifiable index of participants' inhibitory capacity.

4.2.3. Electrophysiological data acquisition

Scalp voltage measurements were obtained using a 21-channel EEG cap equipped with Ag/AgCl electrodes, which adhered to the 10-20 system for electrode placement. The data was collected using the NeXus-32 system from Mind Media, utilizing a hardware-based common average reference (Nexus-32, n.d.). The VEOG signals were recorded using electrodes placed above and below the left eye, while horizontal electrooculography The HEOG signals were captured by electrodes positioned at the outer canthi of both eyes. EEG data was continuously recorded throughout the experiment, including during the hand muscle contraction phase, and subsequently re-referenced offline to linked mastoids. The data was then filtered and down-sampled to 512 Hz for further analysis.

4.2.4. Frontal alpha asymmetry

Frontal alpha asymmetry scores were calculated based on EEG data collected across three distinct conditions: resting-state with EO, resting-state with EC, and during hand muscle contractions. The resting-state EEG was recorded in two separate 5-minute sessions, one with EO and one with EC, both before and after the intervention. The hand muscle contraction condition involved active muscle engagement, a manipulation hypothesized to influence frontal alpha asymmetry. EEG data preprocessing was conducted using the BrainVision Analyzer 2 software following standardized procedures (Smith et al., 2017), as discussed in Chapter I.

4.2.5. Event-related potentials

Event-related potentials were extracted to analyze the neural correlates of inhibitory control, focusing on two primary ERP components: the Stop N2 and Stop P3. Based on previous studies (Logemann et al., 2014a; Logemann et al., 2014b), the EEG signals were first re-referenced to the linked mastoid electrodes and filtered offline using a high-pass cutoff of 0.5 Hz, a low-pass cutoff of 30 Hz, and a 50 Hz notch filter to reduce noise. The data was then segmented into epochs ranging from -100 ms to 2600 ms, and ocular artifacts were addressed using ICA. After artifact correction, the epochs were baseline-corrected using a reference window of -100 to 0 ms and segmented relative to the presentation of the go-signal. Artifact rejection criteria included excluding epochs where amplitudes exceeded ±75 μV, with a 200 ms buffer window before and after identified artifacts. For the subsequent stop-signal analysis, epochs were segmented again relative to the onset of the stop-signal, followed by another round of baseline correction. Event-related potential waveforms were then averaged separately for successful and unsuccessful stop trials. The inhibition-related ERPs were computed by subtracting the average activity during unsuccessful stop trials from that of successful stop trials. Based on the grand average waveforms, specific latency intervals were selected for detailed analysis. The Stop N2 component was extracted from the F4 electrode in the time window of 172–292 ms, while the Stop P3 component was extracted from the Cz electrode between 191–241 ms. An exploratory analysis was also conducted for the early Stop N2 component from a shorter 160–180 ms time window.

4.2.6. Unilateral muscle contraction

The primary goal of incorporating UMC was to enhance relative right-sided brain activity in the DLPFC, a region associated with inhibitory control. Participants in the experimental group engaged in UMC by repeatedly squeezing a stress ball with their left hand for 45-second intervals, followed by 15-second rest periods. The UMC task was performed for a total of 10 minutes (Harmon-Jones, 2006). Participants in the control group performed simultaneous bilateral hand muscle contractions using both hands for the same duration. Without this approach, such as using right-hand muscle contraction, any observed effects could have been influenced by the right-hand-associated brain activity, making it challenging to isolate and accurately attribute the

changes in frontal alpha asymmetry and inhibitory control to left-hand muscle contraction.

4.2.7. Procedure

This study employed a controlled experimental design with both within-subject and between-subject factors. The within-subject factors included time (pre- and post-intervention) and condition (neutral and reward). The between-subject factor was the type of hand contraction (unilateral or bilateral). Upon arrival at the laboratory, participants reviewed an information letter, confirmed their eligibility based on the inclusion criteria, and provided informed consent. EEG electrodes were then placed on their scalp for data collection during the initial resting-state session, which involved recording EEG for a total of 10 minutes, 5 minutes each for the EO and EC conditions. After the EEG recording, participants completed questionnaires related to their demographic and cognitive profiles. Next, participants engaged in the initial phase of the Stop Signal Task to establish baseline inhibitory control metrics. Following this, participants were assigned to either the unilateral or bilateral muscle contraction group in a counterbalanced order. The UMC or bilateral hand contraction intervention was then administered, lasting 10 minutes. Immediately after the intervention, the same sequence of steps, resting-state EEG recording and the Stop Signal Task, was repeated. The entire experimental procedure, including the intervention and pre-/post-assessments, was completed in a single session lasting approximately five hours. Figure 4.1.B illustrates the procedure.

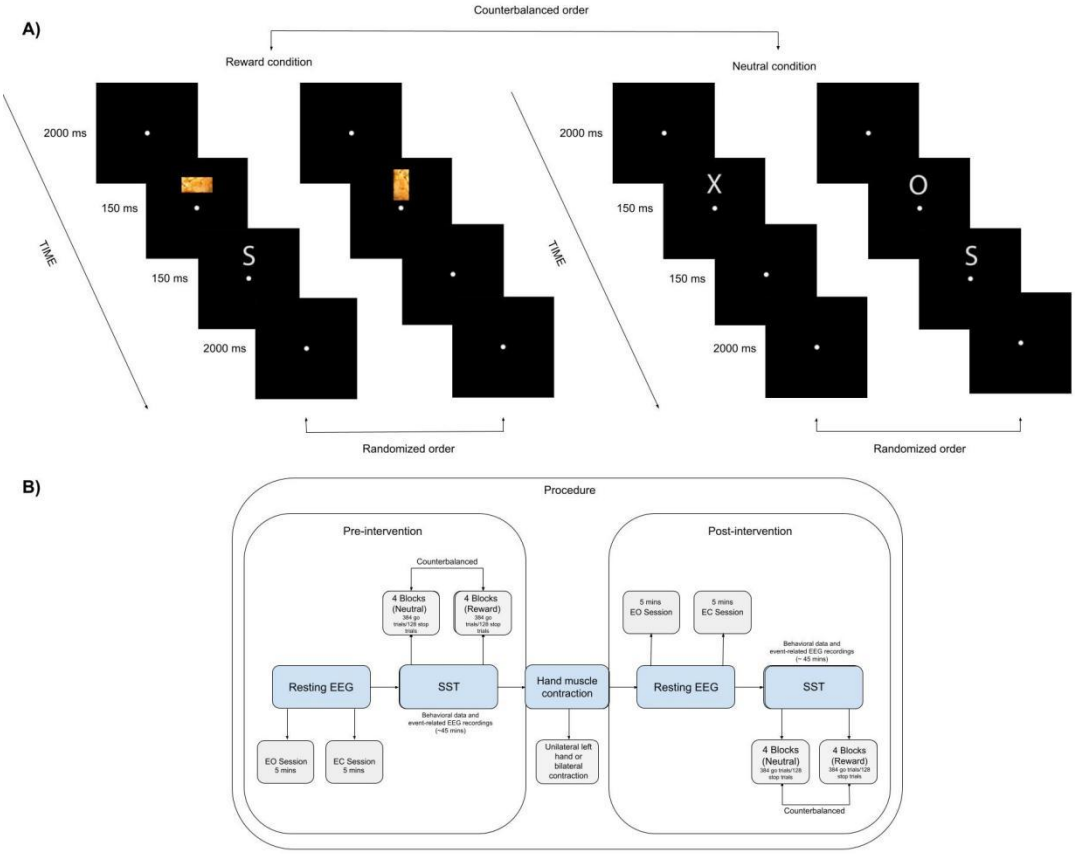


Figure 4.1. Figure 4.1.A outlines the task procedure. Participants started the Stop Signal Task in either the neutral or reward condition in a counterbalanced order. Each condition began with a practice block to determine the optimal go-stop interval, followed by four experimental blocks. In the reward condition, food pictures were shown, while in the neutral condition, the letters "X" and "O" were displayed. Some trials included a stop signal, requiring participants to withhold responses. Figure 4.1.B describes the process of collecting pre-intervention resting-state EEG to measure frontal alpha asymmetry, with 5-minute sessions of eyes open and closed. Afterward, participants completed the Stop Signal Task. This was followed by either bilateral or unilateral left hand muscle contractions for 10 minutes. Frontal alpha asymmetry was recorded during the contractions. The post-intervention assessment repeated the resting-state EEG and Stop Signal Task.

4.2.8. Statistical analysis

All statistical analyses were conducted using R (R Software: A Tool Analysing Experimental Data, 2016). Participants with missing data or extreme outliers exceeding 3 SD from the mean were excluded from the relevant analyses. For frontal alpha asymmetry, a repeated-measures ANOVA with a 2 × 2 design was employed to evaluate the effects of UMC. For the electrophysiological and behavioral indices of inhibitory control, a repeated-measures ANOVA with a 2 × 2 × 2 design was used to assess the interaction effects of condition, time, and intervention type. To supplement these analyses, Bayesian statistics were employed, utilizing a Bayes factor of 10 (BF_{10}). We used the "BayesFactor" package in R. Results from the exploratory analyses and Bayesian factor analyses are presented in the supplementary materials, offering a nuanced interpretation of the intervention's impact on frontal alpha asymmetry and self-regulatory processes.

4.3. Results

The results regarding the effect of UMC on frontal alpha asymmetry are detailed in Table 4.1. The main effect of group on frontal alpha asymmetry F8-F7 scores during EO conditions was significant: $F(1, 110) = 7.14$, $p = .008$, $\eta_p^2 = 0.061$. This finding was further corroborated by Bayesian statistics ($BF_{10} = 4.94$; Supplementary Table 4.1), indicating that the UMC group is linked with higher right frontal, inhibitory, activity, independent of temporal effects (Figure 4.2). The observed effect likely reflects inherent differences between the groups, which may stem from factors such as initial group characteristics. Conversely, the immediate (online) effect of unilateral left-hand muscle contraction was not statistically significant: $F(1, 59) = 0.07$, $p = .787$, $\eta_p^2 = 0.001$. We also examined the time and group interaction by including three time points (pre-, during, and post-intervention) within the same model for frontal alpha asymmetry measurements. The interaction effects were not significant (Supplementary Table 4.2). Complete results are available in the supplementary materials.

Table 4.1.

Repeated Measures ANOVA Results for Frontal Alpha Asymmetry in Study 2

Variables	*df*	*F*	*p*	η_p^2
FAA F4-F3 (EO) (*n* = 57)				
Time	1	0.01	.952	<0.001
Group	1	0.06	.796	<0.001
Time x Group	1	0.20	.651	0.001
Residuals	110			
FAA F4-F3 (EC) (*n* = 58)				
Time	1	<0.01	.982	<0.001
Group	1	0.39	.532	0.003
Time x Group	1	<0.01	.936	<0.001
Residuals	112			
FAA F4-F3 (int) (*n* = 61)				
Group	1	0.07	.787	0.001
Residuals	59			
FAA F8-F7 (EO) (*n* = 57)				
Time	1	<0.01	.926	<0.001
Group	1	7.14	.008*	0.061
Time x Group	1	0.02	.871	<0.001
Residuals	110			
FAA F8-F7 (EC) (*n* = 58)				
Time	1	0.42	.516	0.003
Group	1	0.39	.532	0.003
Time x Group	1	0.52	.471	0.004
Residuals	112			
FAA F8-F7 (int) (*n* = 61)				
Group	1	0.01	.921	<0.001
Residuals	59			

Note. Abbreviations: FAA = Frontal alpha asymmetry; EO = Eyes-open; EC = Eyes-closed; Int = Intervention. Significance level used = .05. Participants with missing values were excluded from the analyses. “Int” refers to the frontal alpha asymmetry during the bilateral and unilateral hand muscle contraction interventions.

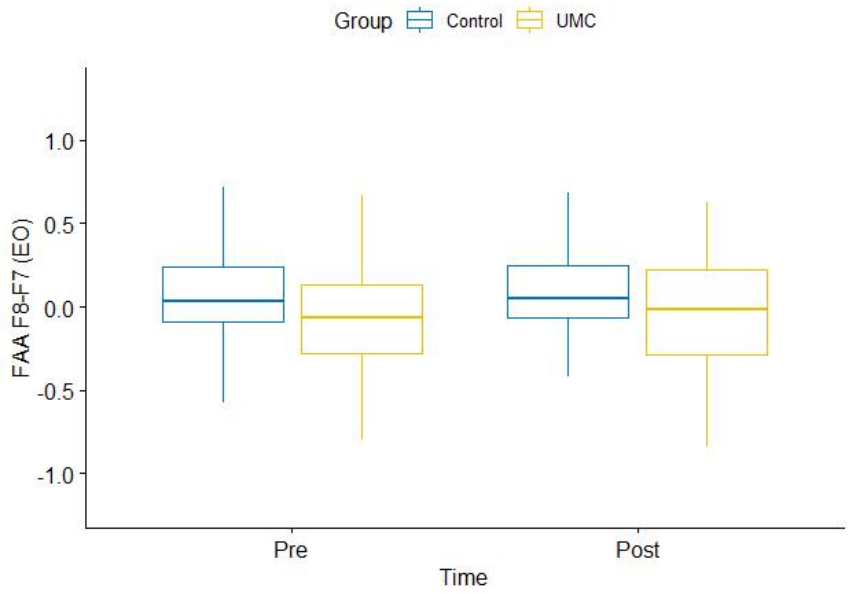


Figure 4.2. The figure shows the main effect of group on eyes open frontal alpha asymmetry at F8-F7 electrodes.

To analyze the impact of UMC on inhibitory control, first, we excluded the participants with missing values and <10% inhibition rates for the calculation of SSRTs. The details are in Table 4.2 and 4.3. Notably, we did not observe any significant influence of UMC on SSRT scores, specifically: $F(1, 220) = 0.21$, $p = .645$, $\eta_p^2 < 0.001$. Bayesian factor results can be found in Supplementary Table 4.3.

Table 4.2.

Descriptive Statistics for Stop-Signal Task Performance in Study 2

Variables (n = 57)	Min	Max	*M*	*SD*
Pre-intervention				
Neutral				
SSRT (ms)	3.26	576.93	183.13	89.60
Inhibition Rate (%)	17.97	52.34	46.53	7.55
Omission Rate (%)	0	33.59	6.08	3.36
Reward				
SSRT (ms)	36.28	703.02	188.15	97.54
Inhibition Rate (%)	17.19	54.69	48.13	5.38
Omission Rate (%)	0	43.22	6.87	8.55
Post-intervention				

Neutral				
SSRT (ms)	53.7	1109.6	207	139.73
Inhibition Rate (%)	5.46	53.12	46.36	8.89
Omission Rate (%)	0	41.92	9.19	9.65
Reward				
SSRT (ms)	52.52	602.58	200.86	84.07
Inhibition Rate (%)	7.03	52.34	46.72	8.02
Omission Rate (%)	0.26	39.05	8.47	10.05

Note. Abbreviations: SSRT = Stop-signal reaction times. Participants with missing data and <10% inhibition rates were excluded from the analysis for the calculation of stop-signal reaction times.

Table 4.3.

Repeated Measures ANOVA Results for Stop Signal Task Performance in Study 2

Variables (n = 57)	*df*	*F*	*p*	η_p^2
SSRT				
Time	1	0.9	.342	0.004
Condition	1	0.54	.461	0.002
Group	1	0.49	.484	0.002
Time x Condition	1	0.23	.629	0.001
Time x Group	1	0.21	.645	<0.001
Condition x Group	1	<0.01	.949	<0.001
Time x Condition x Group	1	0.15	.629	<0.001
Residuals	220			

Note. Abbreviations: SSRT = Stop-signal reaction time. Significance level used: .05. Participants with missing data and <10% inhibition rates were excluded from the analysis.

We presented the results regarding event-related potentials in Table 4.4. There was no significant effect of unilateral left hand muscle contraction on the brain activity indices of inhibitory control. However, the main effect of time on the stop N2 and the stop P3 was statistically significant: $F(1, 204) = 4.79$, $p = .029$, $\eta_p^2 = 0.022$, $BF_{10} = 1.37$ in Supplementary Table 4.4 and $F(1, 196) = 4.19$, $p = .041$, $\eta_p^2 = 0.020$, $BF_{10} = 1.10$ in Supplementary Table 4.5, respectively. Figure 4.3 and Figure 4.4 show the results of the Stop N2 and the Stop P3, respectively. Surprisingly, while the Stop N2 suggested an increased negativity over time, the stop P3 showed a decreased positivity. Our

exploratory analysis regarding the Stop N2 at 160-180 ms showed a similar effect of time: $F(1, 204) = 4.40$, $p = .037$, $\eta_p^2 = 0.021$. For details, please refer to Supplementary Tables 4.6.

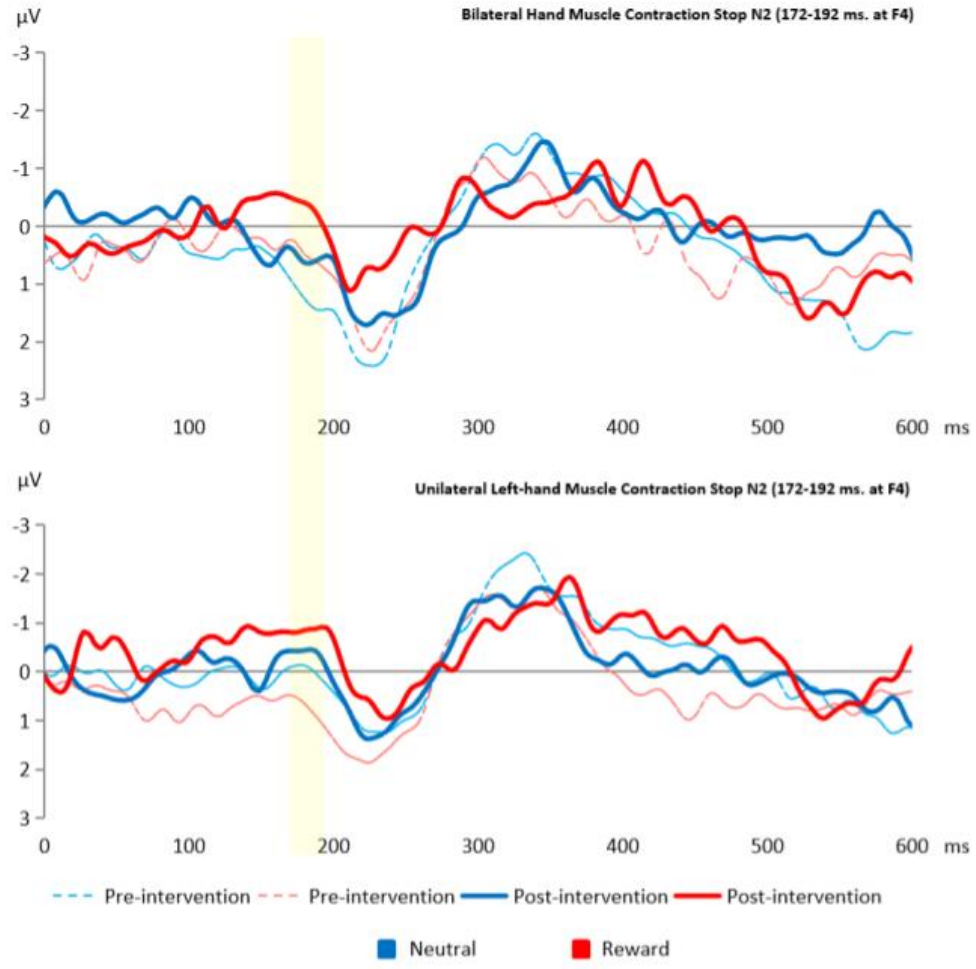


Figure 4.3. This figure illustrates the effect of time on the Stop N2 (172-192 ms) at F4 electrode. The bar represents the Stop N2 peaks.

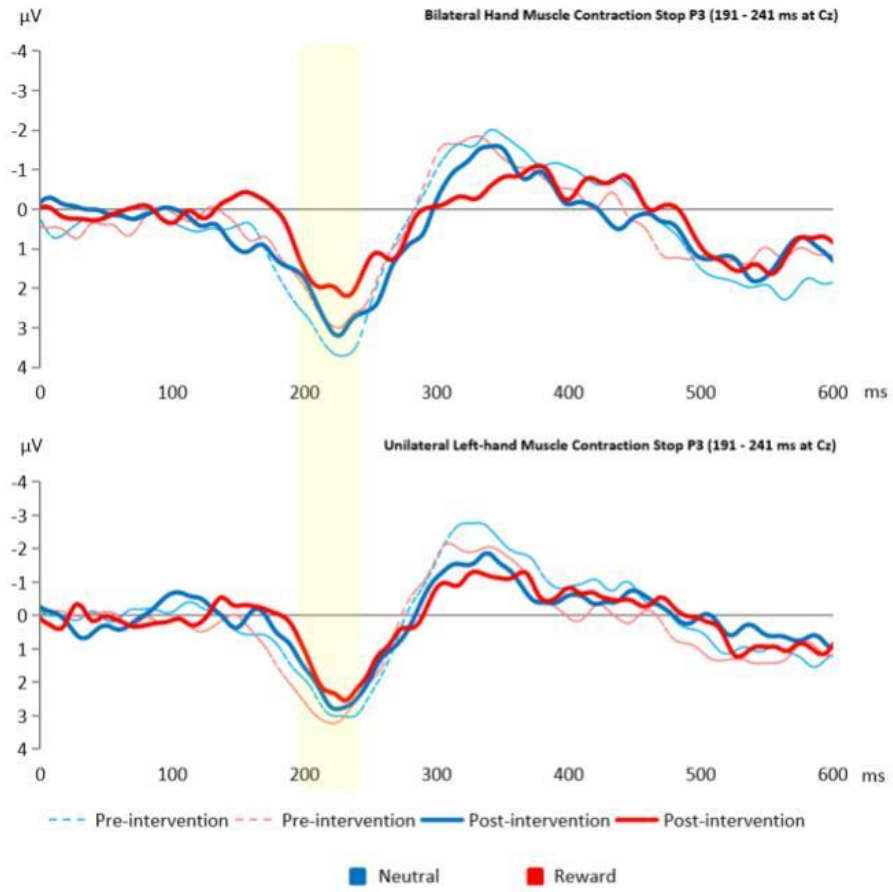


Figure 4.4. This figure illustrates the effect of time on the Stop P3 (191-241) at Cz electrode. The bar represents the Stop P3 peaks.

Table 4.4.

Repeated Measures ANOVA Results for Event-Related Potentials in Study 2

Variables	*df*	*F*	*p*	η_p^2
Stop N2 at 172-192 ms. (F4) (*n* = 53)				
Intercept	1	1.17	.283	0.022
Time	1	4.79	.029*	0.022
Condition	1	1.14	.285	0.005
Group	1	2.12	.146	0.010
Time x Condition	1	1.07	.301	0.005
Time x Group	1	0.07	.792	<0.001
Condition x Group	1	3.18	.076	0.015
Time x Condition x Group	1	0.60	.437	0.002
Residuals	204			
Stop P3 at 191-241 ms. (Cz) (*n* = 51)				
Intercept	1	87.24	<.001*	0.640
Time	1	4.19	.041*	0.020
Condition	1	0.89	.345	0.004
Group	1	0.02	.872	<0.001
Time x Condition	1	0.27	.601	0.001
Time x Group	1	<0.01	.954	<0.001
Condition x Group	1	1.37	0.241	0.006
Time x Condition x Group	1	0.29	0.587	0.001
Residuals	196			

Note. Significance level used = .05. Participants with missing data and low segments (≤ 2) were excluded from the analyses.

We conducted further analyses regarding the effect of unilateral left-hand muscle contraction on stop signal reaction times. We created two models, one of which included participants starting with the neutral condition, while the other consisted of participants starting with the reward condition. Therefore, we divided the task to investigate the immediate effects of the interventions. We found no effect of UMC on the first condition, both neutral and reward ($F(1, 28) = 2.07$, $p = .161$, $\eta_p^2 = 0.068$ and $F(1, 26) = 0.06$, $p = .809$, $\eta_p^2 = 0.002$, respectively). Supplementary Tables 4.7 and 4.8 contain detailed results.

4.4. Discussion

This study investigated the impact of UMC on frontal alpha asymmetry, potential marker of self-regulation, as well as behavioral and neural indices associated with inhibitory control in the context of food rewards. Our primary aim was to determine whether UMC influenced frontal alpha asymmetry and inhibitory control processes.

The results revealed a significant main effect of group on frontal alpha asymmetry, indicating that the UMC group exhibited higher right relative to left frontal cortical activity compared to the bilateral hand muscle contraction group. This finding aligns with prior research suggesting that unilateral motor actions can modulate cortical asymmetry and potentially impact inhibitory processes (Harmon-Jones, 2006; Hellige, 1993; Schiff & Lamon, 1994). However, the lack of a significant time-by-group interaction indicates that this observed group effect on frontal alpha asymmetry may not be driven by the intervention itself but might reflect a pre-existing difference in frontal alpha asymmetry between groups.

The electrophysiological findings provided further insight into the temporal dynamics of inhibitory control. Specifically, we observed a significant increase in the Stop N2 amplitude over time, suggesting increased inhibitory brain activity as the task progressed. In contrast, the Stop P3 amplitude exhibited a significant reduction in positivity, which may indicate reduced inhibitory brain activity or altered task engagement over time. These distinct changes in the Stop N2 and P3 components suggest that different aspects of inhibitory control, such as conflict detection and the allocation of cognitive resources, may be modulated independently by time and experience during task performance.

The Stop N2 is associated with response conflict and the engagement of early inhibitory processes, while the Stop P3 is often linked to the successful inhibition of a response (Groom & Cragg, 2015) and the evaluation of task outcomes (Jollans et al., 2017). Our results showed a complex temporal pattern where the initial increase in inhibitory activity, as reflected by the Stop N2, was not sustained, as indicated by the reduction in the Stop P3 amplitude. This pattern may reflect an heightened conflict early in the task but subsequently experienced reduced engagement or altered task expectations, potentially due to fatigue or decreased motivation. Such an interpretation is consistent

with findings from previous research, which has linked the Stop N2 to conflict monitoring (Groom & Cragg, 2015) and the Stop P3 to the subjective evaluation of task success and error monitoring (Jollans et al., 2017).

Despite these neural changes, we did not observe corresponding behavioral changes in response inhibition performance, as indicated by the absence of significant differences in the SSRT across conditions. Therefore, these effects did not translate into overt behavioral alterations in inhibitory control. One possible explanation is that the dynamic tracking algorithm used in the Stop Signal Task, which adjusts go-stop intervals to maintain a 50% inhibition rate, may have masked subtle behavioral differences that would otherwise manifest under fixed-interval conditions.

The pattern of findings regarding the Stop N2 and Stop P3 components also has broader implications for understanding the roles of these event-related potentials in inhibitory control. The ongoing debate concerning whether the Stop N2 reflects early response conflict or a preparatory inhibitory signal, and whether the Stop P3 is more closely related to the evaluation of response inhibition success or cognitive control, remains unresolved. Our results align with studies that have shown the Stop N2 to be sensitive to conflict monitoring, while the Stop P3 appears to be influenced by expectations and task outcome evaluations. This interpretation is supported by evidence that the P3 component increases with the subjective unexpectedness of task outcomes, suggesting that a reduction in the P3 over time may reflect a decrease in outcome expectancy as participants become more accustomed to the Stop Signal Task.

The absence of a clear UMC effect on behavioral indices of inhibition could also be due to the influence of the control condition, which, despite its design to be neutral, might still modulate brain activity and performance due to simultaneous bilateral hand contractions. This pattern resembles findings in tES research, where bilateral stimulation can induce complex effects depending on the balance of activation across hemispheres (Monai et al., 2016; Monai & Hirase, 2017). In this context, the apparent lack of behavioral differences between the UMC and control groups could reflect an interaction between the interventions' direct impact on brain activity and the participants' evolving task engagement or motivation levels across sessions.

In conclusion, while UMC induced alterations in frontal alpha asymmetry, it did not produce significant changes in the behavioral indices of inhibitory control. The significant time effects observed for the Stop N2 and Stop P3 components, however, underscore the complex temporal modulation of inhibitory brain activity and suggest that the dynamics of response conflict and post-response evaluation evolve throughout task performance. These findings highlight the importance of considering temporal changes when assessing neural markers of inhibitory control and suggest that UMC may modulate specific aspects of cortical asymmetry without necessarily translating into immediate behavioral changes. Future research should further investigate the role of temporal dynamics in inhibitory control and explore how different muscle contraction protocols might interact with other cognitive and motivational factors to refine the assessment and potential therapeutic application of UMC for enhancing inhibitory processes.

5. Study 4 - The relationship between frontal alpha asymmetry and behavioral and brain activity indices of reactive inhibitory control[6]

5.1. Introduction

Building on the approach of Study 3, we explored inhibitory control, the critical component of adaptive behavior (Braver, 2012; Mirabella, 2023) within the framework of self-regulatory processes. Given that the DLPFC is thought to play a central role in generating approach-withdrawal system asymmetries (Kelley et al., 2017), this study aimed to modulate activity in the right DLPFC, targeting improved withdrawal motivation.

To achieve this, in contrast to Study 3, which utilized behavioral neuromodulation, this study uniquely employed transcranial direct current stimulation (tDCS) as its intervention technique. TDCS is a noninvasive method that delivers low electrical

[6] Please refer to the published paper for further information and supplementary materials: Akil, A.M., Cserjési, R., Nagy, T., Demetrovics, Z., Németh, D., & Logemann, H. N. A. (2024). The relationship between frontal alpha asymmetry and behavioral and brain activity indices of reactive inhibitory control. *Journal of Neurophysiology*, 132. https://doi.org/10.1152/jn.00046.2024.

currents via scalp electrodes to modulate brain activity (Schestatsky et al., 2013). Previous studies have demonstrated that tDCS targeting the right DLPFC (anode over the right DLPFC/cathode over the left DLPFC) can effectively reduce food cravings and consumption in both clinical and nonclinical populations (Burgess et al., 2016; Kekic et al., 2017; Lapenta et al., 2014).

Although the effects of tDCS on inhibitory control have been documented, it remains uncertain whether these effects are mediated by changes in frontal alpha asymmetry and how such changes influence specific markers of inhibitory abilities. As in the third study, we employed the Stop Signal Task (Logan et al., 1984; Schmajuk et al., 2006) in food-within food-reward contexts and examined related neural markers, including the Stop N2 and Stop P3 event-related potentials (Schmajuk et al., 2006; Kenemans & Kähkönen, 2010).

Our hypotheses remained consistent. We anticipated that active tDCS would modulate frontal alpha asymmetry by increasing activity in the right DLPFC, which in turn would enhance both behavioral performance in inhibitory control and inhibitory brain activity, specifically the Stop N2 and Stop P3.

5.2. Methods

5.2.1. Participants

A pilot study (n = 10) guided the sample size determination for the main study using G*Power (Faul et al., 2007), targeting a power of 80% and alpha of .05, with an estimated FAA test-retest correlation of 0.6. An effect size of $f > 0.237$ was expected to be detectable with a sample of 30 in the active intervention group. Ultimately, 65 healthy participants (46 females, 19 males, 57 right-handed, M_{age} = 23.93 years, SD_{age} = 6.08) were recruited via social media and university courses. They had to be at least 18 years old. Participants were excluded if they declared a history of psychiatric disorders, frequent headaches, metallic implants, epilepsy, head trauma, pacemaker usage, chronic skin conditions, or recent drug use. Smoking and caffeine intake were prohibited at least two hours prior to the study. Handedness was not considered as an exclusion criterion because previous studies indicated that it does not have impact on inhibitory control

(Mancini & Mirabella, 2021). Participants provided informed consent, and approval was granted by the Ethics Committee of Eötvös Loránd University.

5.2.2. Stop signal task

As in Study 3, the task was adapted from the original version (Schmajuk et al., 2006) using OpenSesame (Mathôt et al., 2012) and incorporated a food-reward condition based on previous studies (Houben et al., 2014; Tsegaye et al., 2022). The task was illustrated in Figure 5.1. For each condition, a practice block was used to determine the optimal go-stop delay. Each condition included four experimental blocks with 96 go trials and 32 stop trials each, totaling 512 trials per condition and 1,024 trials per phase (pre-modulation and post-modulation), resulting in 2,048 trials in total.

The task began with a fixation dot for 2000 ms. In the food-reward condition, images of palatable foods (e.g., cookies, chips, chocolate, nuts) were presented in either horizontal or vertical orientations for 150 ms, requiring a key response. Each image was 115 pixels in width (2.9°) and 200 pixels in height (5.1°). In the neutral condition, instead of food images, the letters “X” or “O” were shown, and participants responded based on the letter. They were 200 pixels in both width and height (5.1°). During stop trials, the letter “S” appeared 150 ms after the go stimulus, signaling participants to withhold their response. Participants used their right and left index fingers. The stop-signal delay was initially set at 350 ms and adjusted using a staircase algorithm to maintain a 50% inhibition rate. The primary outcome measure was SSRT, calculated by replacing omissions with maximum reaction times (1500 ms) (Verbruggen et al., 2013).

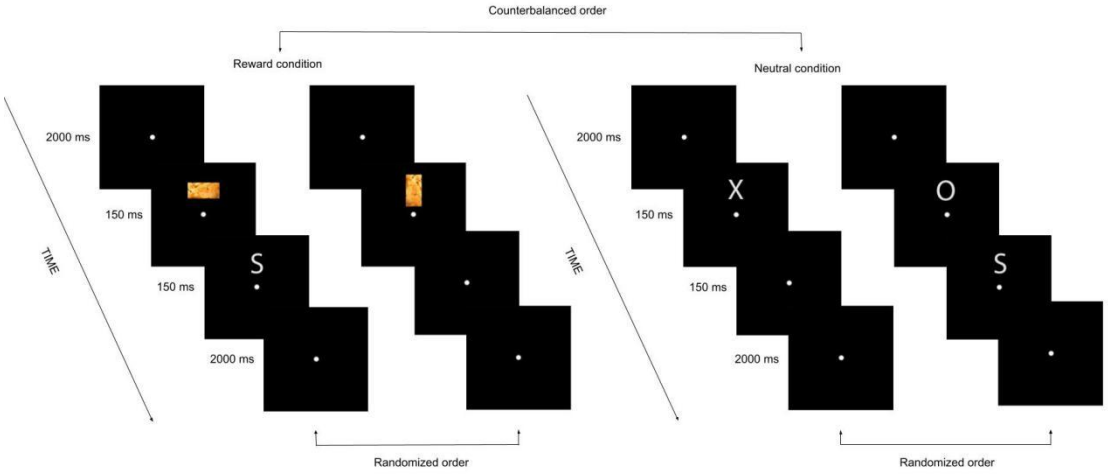


Figure 5.1. Participants began the Stop Signal Task in either the neutral or food-reward condition, based on a counterbalanced order. In the food-reward condition (left), targets (horizontal/vertical) and food images (e.g., chips, chocolate) were presented. In the neutral condition (right), letters "X" and "O" were displayed. Some trials included a stop signal ("S"), requiring participants to withhold their response.

5.2.3. Electrophysiological data acquisition

Scalp voltages were captured with a 21-channel cap Ag/AgCl electrode set according to the 10-20 system. The brand was Mind Media NeXus-32 (Nexus-32, n.d.). The VEOG was recorded above and below the left eye, and the HEOG was recorded bipolarly from the outer canthi of both eyes. Sampling rate was set at 512 Hz. EEG was continually recorded except when tDCS was applied.

5.2.4. Frontal alpha asymmetry

Frontal alpha asymmetry scores were derived from EEG data recorded during two separate 5-min resting-state sessions, one with EO and another with EC, both before and after the intervention. Frontal alpha asymmetry was preprocessed and calculated using BrainVision Analyzer 2, following the methodology outlined in previous studies (Smith et al., 2017) and discussed in Chapter I.

5.2.5. Event-related potentials

BrainVision Analyzer 2 was used for preprocessing. First, signals were referenced to linked mastoids. Subsequently, following previous studies (Logemann et al, 2014a; Logemann et al., 2014b), EEG data were filtered (offline) with a high cutoff of 30 Hz, a low cutoff of 0.5 Hz, and notch filter of 50 Hz. Data were segmented into epochs ranging from −100 ms to 2,600 ms. Ocular corrections were conducted by independent component analysis. After that, epochs were segmented and they were baseline corrected with the baseline set at −100 to 0 ms. We conducted a go-signal locked segmentation, then baseline correction followed by artifact rejection (using minimal/maximal allowed amplitude −75 μV/75 μV and marking 200 ms before and after events as bad). We employed a stop-signal locked segmentation and baseline correction. Next, we computed separate averages for segments corresponding to failed stops and successful stops. The inhibitory Event-related potentials were computed by subtracting the average stop-signal locked activity for failed stops from successful ones. Following a thorough examination of the grand average waveforms, we have identified specific latency intervals for further analysis. Previous studies have shown that the N2 modulation by stopping success is most pronounced at right frontal sites and the P3 modulation at frontocentral sites (Logemann et al., 2013; Schmajuk et al., 2006). For the Stop N2 component, we extracted data from the time window of 166–286 ms at F4, a nearby site, since our EEG setup did not include FC4/FC2 electrode sites. For the Stop P3 component, data from the 211–271 ms time window at Cz were used for export.

5.2.6. Transcranial direct current stimulation

The aim of brain modulation was to modulate frontal alpha asymmetry. A pair of circular sponges (25 cm2) soaked in saline solution were used to deliver direct electrical current with STARSTIM-8 (Neuroelectrics; www.neuroelectrics.com). According to the 10-20 system, the anode was positioned on the right DLPFC (F4) and the cathode on the left DLPFC (F3). Unless it was a sham condition, a steady current of 2 milliampere (mA) was applied for 20 min (Kelley et al., 2017). Figure. 5.2 illustrates the protocol. The safety of this parameter has been shown in healthy subjects (Iyer et al., 2005). In the sham condition, a brief current was also applied to make it comparable sensation-wise to the active condition. Upon completion of the experiment, participants were asked to

identify the condition they had experienced. There was no statistically significant association between the actual group assignment and the correctness of participants' guesses: $\chi^2 = 0.08$; $df = 1$; $p = .772$.

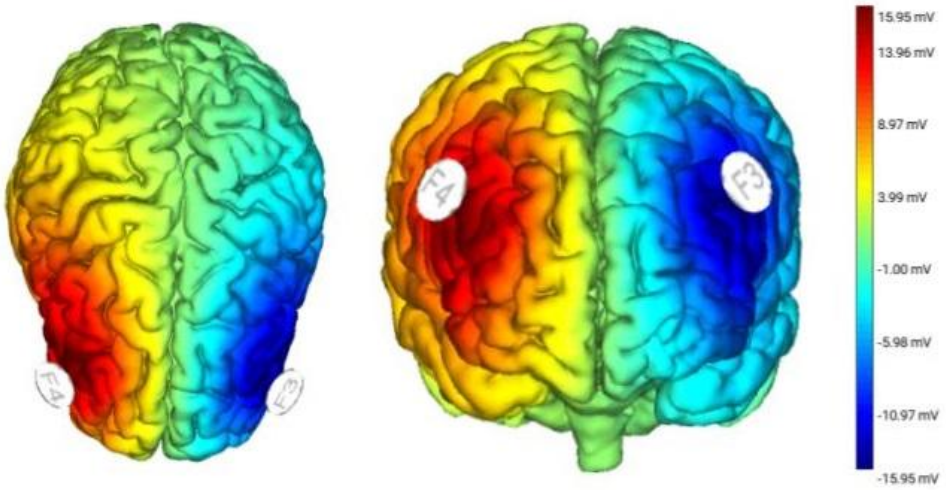


Figure 5.2. This illustration shows the placement of the tDCS electrodes and the protocol used. A constant 2 mA current was delivered through two circular sponge electrodes (25 cm² each), placed on the scalp at F4 (anode) and F3 (cathode) using a saline solution. The stimulation lasted 20 minutes, with a maximum electric field strength of 15.95 μV at the anodal electrode.

5.2.7. Procedure

The study was preregistered on Open Science Framework (OSF)[7]. A randomized, triple-blind, sham-controlled design was used in this research, with within-subject (time: pre-/post-assessment, condition: neutral/food-reward) and between-subject (group: active/sham tDCS) factors (see Figure 5.3). Before and after the neural modulation session, the Stop Signal Task was completed. Participants were seated in a comfortable chair in a dimly lit testing room for the placement of EEG electrodes on the scalp sites after reading the information letter, verifying the inclusion and exclusion criteria, and completing the informed consent form when they arrived at our laboratory. Subsequently, resting-state EEG data were collected, recording for 10 minutes in total

[7] https://osf.io/9y548/

with two blocks of 5-min sessions (EO/EC). They finished the questionnaires after recording of the resting-state EEG. They then completed the first phase of the Stop Signal Task before the tDCS. EEG was recorded throughout the resting states and computer tasks but not during the intervention as the cap was changed. After that, individuals were assigned to either a 2-mA active or a sham tDCS for 20 minutes. It was double-blind. After the intervention, participants immediately started the second phase of the experiment. During the postmodulation evaluation, the same steps, resting-state EEG and the Stop Signal Task, were repeated. The stimulation and pre-/post-assessments were conducted on the same day and lasted approximately five hours.

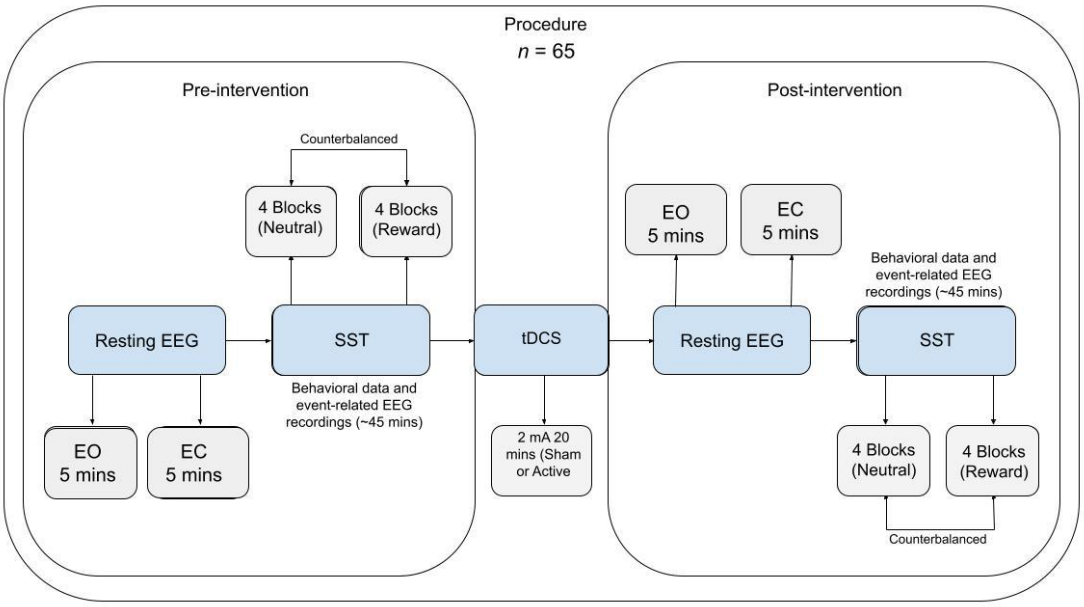


Figure 5.3. The experiment started with pre-intervention resting-state EEG to measure frontal alpha asymmetry in two 5-minute sessions: one with EO and EC. Participants then completed the Stop Signal Task. Afterward, they received either sham or 2 mA active tDCS. In the sham condition, a brief current was applied to mimic the sensation of active tDCS. The procedure was repeated post-stimulation, including the resting-state EEG and Stop Signal Task.

5.2.8. Statistical analyses

The analyses were conducted with SPSS 22 (IBM Corporation, n.d.) and R (R Software: A Tool Analysing Experimental Data, 2016). In addition to employing frequentist

statistics, we also conducted a series of Bayesian analyses using JASP (Love et al., 2019). Upon completing the calculations of the main variables, we excluded participants with missing values and outliers beyond 3 *SD*s from the mean. Each model has a different number of participants excluded, and they are indicated in the tables. A mixed ANOVA with a 2 × 2 design was utilized to investigate hypotheses regarding frontal alpha asymmetry, whereas a 2 × 2 × 2 design was used for analyzing the behavioral and brain activity indices of inhibitory control. We also conducted a correlation analysis to examine the relationship between baseline (pre-tDCS) frontal alpha asymmetry and behavioral and brain activity indices of inhibitory control. Although previous research found that handedness does not affect inhibitory control (Mancini & Mirabella, 2021), as part of the exploratory analyses we excluded left-handed participants and examined the relationships. The electrophysiological variables investigated included frontal alpha asymmetry, the Stop N2, the Stop P3, in addition to the behavioral indices of inhibitory control, SSRTs. For all frequentist statistical analyses the significance level was set at 0.05, and for the null results a Bayesian approach was used with Bayes factor 01 (BF_{01}), which is in favor of null hypotheses (H_0) over alternative hypotheses (H_1). More specifically, BF_{01} values ranging from 1 to 3 are indicative of anecdotal evidence, whereas values falling between 3 and 10 suggest substantial evidence in favor of the null hypothesis (H_0). BF_{01} values exceeding 10 provide strong evidence for H_0. Conversely, values ranging from 1 to 1/3 suggest anecdotal evidence against H_0, whereas values between 1/3 and 1/10 indicate substantial evidence against H_0. Values below 1/10 provide strong evidence against H_0. Values around 1 do not support either hypothesis (Wagenmakers et al., 2011, 2018). The results of Bayesian and exploratory analyses can be found in the Supplemental Materials.

5.3. Results

The ANOVA results revealed no significant interaction between the time and group factors concerning frontal alpha asymmetry. Table 5.1 shows the details. These results were also supported by Bayesian statistics. We found substantial evidence in favor of the null hypothesis ($BF_{01} > 3$) (Supplementary Table 5.1). Our exploratory analysis, limited to right-handed participants, similarly failed to yield any novel insights. For further information, please see Supplementary Table 5.2. On the other hand, in the food-reward condition, we observed a significant negative correlation between baseline

frontal alpha asymmetry F4-F3 (EO) and two key brain activity indices of inhibitory control, namely the Stop N2 (r = -0.48, p < .001) and P3 (r = -0.34, p = .010). For further information regarding the correlation analysis, refer to Supplementary Table 5.3.

Table 5.1.

Repeated Measures ANOVA Results for Frontal Alpha Asymmetry in Study 4

Variables	*df*	*F*	*p*	η_p^2	BF_{01}
FAA F4-F3 (EO) (*n* = 59)					
Group	1	3.64	.058	0.030	1.7
Time	1	0.39	.530	0.003	5.53
Group x Time	1	0.28	0.594	0.002	8.85
Error	114				
FAA F4-F3 (EC) (*n* = 59)					
Group	1	1.22	.271	0.010	4.08
Time	1	1.27	.261	0.010	3.64
Group x Time	1	0.49	.484	0.004	11.61
Error	114				
FAA F8-F7 (EO) (*n* = 62)					
Group	1	<0.01	.951	<0.001	6.30
Time	1	<0.01	.933	<0.001	7.31
Group x Time	1	0.29	.588	0.002	27.49
Error	120				
FAA F8-F7 (EC) (*n* = 59)					
Group	1	0.03	.849	<0.001	5.57
Time	1	3.58	.060	0.030	0.98
Group x Time	1	0.08	.769	<0.001	8.25
Error	114				

Note. Significance level used = .05. Participants with missing values and outliers (based on 3 standard deviations from the mean) were excluded from the analyses.

The results regarding inhibitory control are illustrated in Figure 5.4, and descriptive and inferential statistics are shown in Table 5.2 and 5.3, respectively. We found no significant effect of tDCS on the SSRT. However, there was a significant main effect of time on the SSRT and it was supported by Bayesian statistics as well: $F(1, 220) = 6.33$, $p = 0.012$, $\eta_p^2 = 0.027$, $BF_{01} < 0.1$ (Supplementary Table 5.4). These findings indicate

that inhibitory control decreases as time progresses, irrespective of the condition or group. As part of our exploratory analysis, left-handed participants were excluded. However, the new model did not reveal any new insights. Further details can be found in Supplementary Table 5.5.

Table 5.2.

Descriptive Statistics for Stop-Signal Task Performance in Study 4

Variables (n = 57)	Min	Max	*M*	*SD*
Pre-intervention				
Neutral				
SSRT (ms)	1	331.4	185.6	80.4
Go Trials RT	339.7	926	647.2	125.6
Stop Trials RT	237.7	442.9	307.7	42
Inhibition Rate (%)	10.9	57	46.3	8.1
Omission Rate (%)	0	44	8.7	9.6
Food-reward				
SSRT (ms)	78.8	330.0	201.6	68.6
Go Trials RT	377.7	973	666.8	132.1
Stop Trials RT	441.9	436.6	308.7	
Inhibition Rate (%)	25.7	51.5	46.6	5.8
Omission Rate (%)	0	34.1	6.9	7.3
Post-intervention				
Neutral				
SSRT (ms)	48.3	389.2	215.8	75.9
Go Trials RT	299.6	957.7	612	162.4
Stop Trials RT	206.2	418.5	298.4	55.6
Inhibition Rate (%)	15.6	53.1	45.1	8.6
Omission Rate (%)	0	32.2	9	8
Food-reward				
SSRT (ms)	82.6	430.9	220.6	68.5
Go Trials RT	317.6	1015	616.6	155.9
Stop Trials RT	224.5	473.6	301.2	57.3
Inhibition Rate (%)	13.2	54.6	45.6	7.8
Omission Rate (%)	0.2	36.7	9	8.4

Note. The participant exclusion criteria included missing values, outliers, negative values, and inhibition rates under 10% for the calculation of stop-signal reaction times.

Table 5.3.

Repeated Measures ANOVA Results for Stop Signal Task Performance in Study 4

Variables (n = 57)	*df*	*F*	*p*	η_p^2	BF_{01}
SSRT					
Time	1	6.33	.012*	0.027	0.01
Condition	1	1.13	.287	0.005	3.24
Group	1	1.49	.222	0.006	4.28
Time x Condition	1	0.33	.565	0.001	3.94
Time x Group	1	<0.01	.956	<0.001	6.66
Condition x Group	1	1.28	.257	0.005	4.17
Time x Condition x Group	1	0.03	.862	<0.001	5591
Error	220				

Note. Participants with missing values, outliers, negative values, and inhibition rates under 10% were excluded from the analysis.

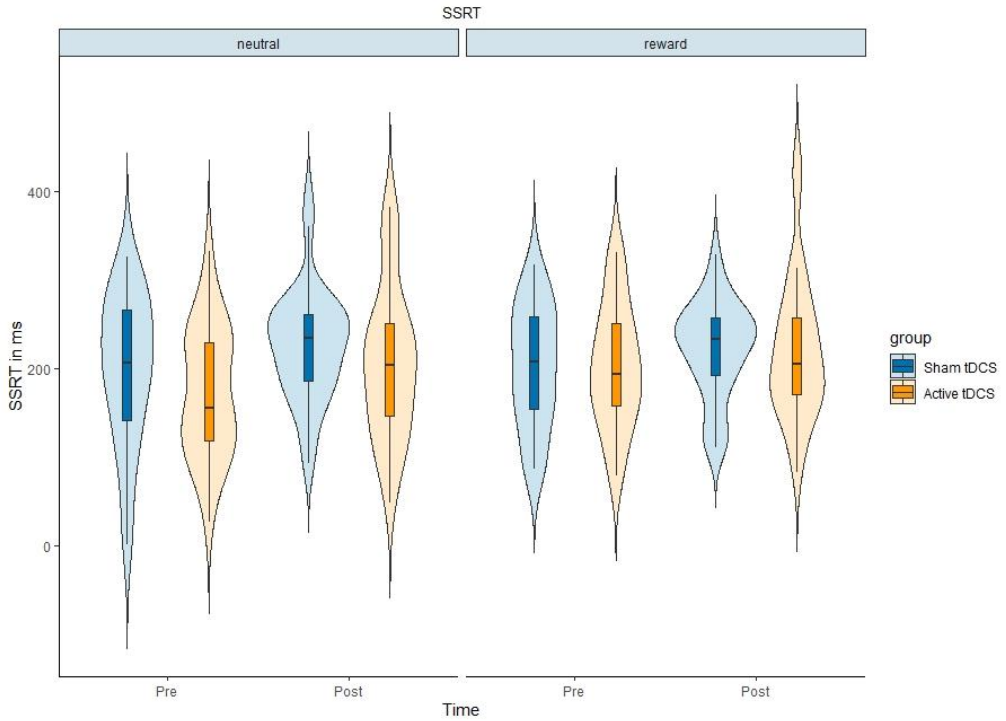


Figure 5.4. This figure displays the average stop-signal reaction times, considering the factors of time (pre-/post-intervention), condition (neutral/food-reward), and group (sham/active transcranial direct current stimulation). The error bars indicate standard errors. It illustrates that inhibitory control decreased from pre-assessment to post-assessment, regardless of condition and group factors. It is important to note that longer stop-signal reaction times represent decreased inhibitory control.

The results regarding the effect of tDCS on the Stop N2 and the Stop P3 are displayed in Figure 5.5 and Figure 5.6, respectively. The interaction effect of time, condition, and group on the indices was found to be insignificant. There was a statistically significant effect of the interaction between time and condition on the Stop P3: $F(1, 228) = 4.21$, $p = .041$, $\eta_p^2 = 0.018$, $BF_{01} = 2.96$. Please see Table 5.4 and Figure 5.7. In other instances, Bayesian factors supported H_0 ($BF_{01} > 3$) (Supplementary Tables 5.6 and 5.7).

As an exploratory analysis, we excluded the left-handed participants. Similarly, we found a significant effect of time and condition interaction on the Stop N2: $F(1, 204) = 4.46$, $p = .035$, $\eta_p^2 = 0.021$ (Supplementary Table 5.8). Supplementary Figure 5.1 indicated that over time, there was a decrease in the early-onset inhibitory brain activity, particularly in the reward condition.

Table 5.4.

Repeated Measures ANOVA Results for Event-Related Potentials in Study 4

Variables	*df*	*F*	*p*	η_p^2
Stop N2 at 166-188 ms. (F4) (*n* = 59)				
Time	1	1.25	.264	0.005
Condition	1	0.29	.591	0.001
Group	1	1.91	.168	0.008
Time x Condition	1	1.54	.216	0.006
Time x Group	1	0.20	.649	<0.001
Condition x Group	1	0.15	.698	0.001
Time x Condition x Group	1	0.36	.545	0.001
Error	228			
Stop P3 at 211-271 ms. (Cz) (*n* = 59)				
Time	1	0.80	.329	0.003
Condition	1	0.97	.323	0.004
Group	1	5.87	.016*	0.025
Time x Condition	1	4.21	.041*	0.018
Time x Group	1	0.10	.750	<0.001
Condition x Group	1	0.03	.850	<0.001
Time x Condition x Group	1	0.04	0.825	<0.001
Error	228			

Note. Participants with missing values, erroneous values, and outliers (based on 3 standard deviations from the mean) were excluded from the analyses. Significance level used: .05.

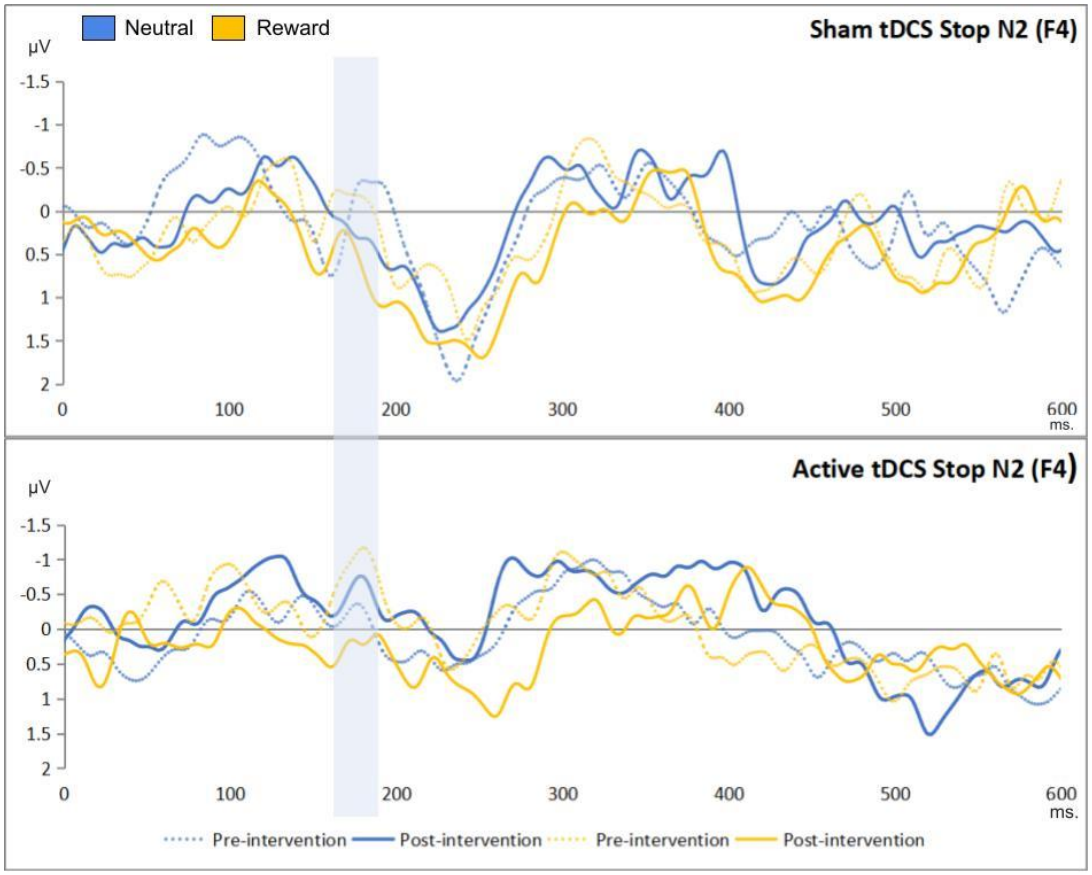


Figure 5.5. This figure shows the stop-signal locked event-related potentials during the stop signal task, effects of time, condition, and group on the Stop N2 (166-286 ms), based on successful inhibitions minus failed ones. The x-axes represent the time in milliseconds; the y-axes represent the Stop N2 scores in microvolts. The blue bar highlights the Stop N2 peaks.

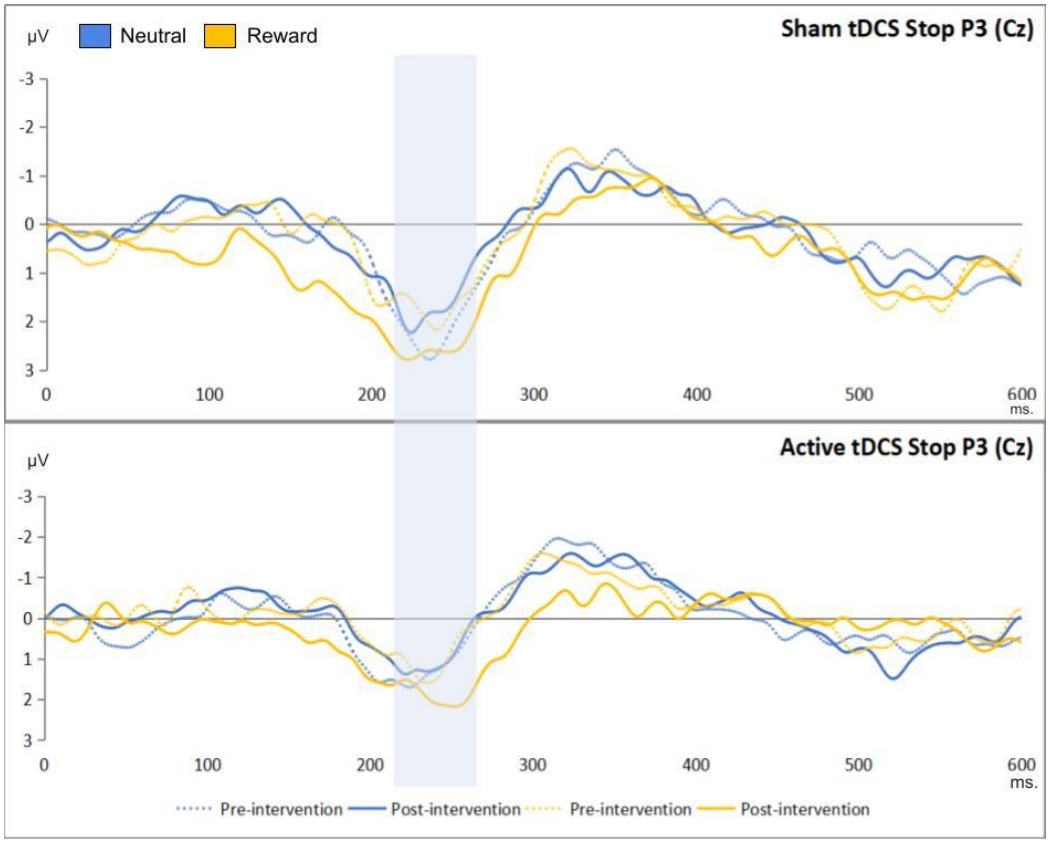


Figure 5.6. This figure shows the stop-signal locked event-related potentials during the stop signal task, the effects of time, condition, and group on the Stop P3 (211-271 ms), based on successful inhibitions minus failed ones. The x-axes represent the time in milliseconds; the y-axes represents the Stop P3 scores in microvolts. The blue bar highlights the Stop P3 peaks.

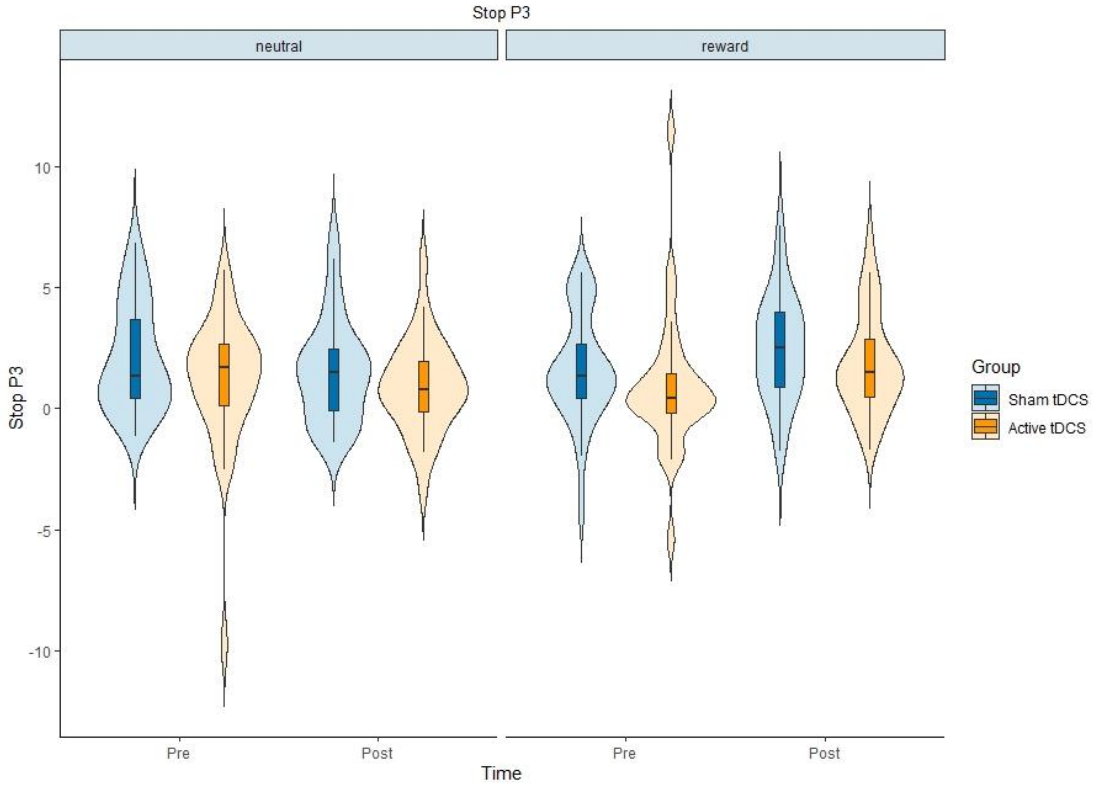


Figure 5.7. The figure shows the exact effect of time and condition interaction on the Stop P3. The x-axes represent the time factor; the y-axes represent the Stop P3 in microvolts.

5.4. Discussion

Our study aimed to explore the association between frontal alpha asymmetry and behavioral and brain activity indices of inhibitory control. Contrary to our initial hypothesis, we did not observe any significant effect between tDCS and FAA, and there were also no noticeable impacts of tDCS on the behavioral and brain activity indicators of inhibitory control. A noteworthy trend emerged where stop-signal reaction times showed a decline across all conditions and groups over time, accompanied by a concurrent decrease in early-onset inhibitory brain activity and an increase in late-onset inhibitory brain activity in the intrinsic reward (food) condition.

In our correlation analysis on the baseline (pre-intervention) frontal alpha asymmetry, greater right frontal brain activity compared to the left (indicating lower frontal alpha asymmetry) was found to be associated with reduced early-onset inhibition (as

evidenced by Stop N2), yet it was associated with heightened late-onset inhibitory activity (as indicated by Stop P3).

There was also no effect of tDCS and measures of inhibitory control. The potential impact of tDCS on cognition and behavior is notably variable (Jacobson et al., 2012), challenging the notion of a polarity-specific influence. Although tDCS is theoretically expected to increase excitability under the anode and decrease it under the cathode, the actual cognitive and behavioral effects are far more complicated. Interestingly, some studies have even reported facilitatory effects associated with stimulation under the cathode, possibly attributed to noise reduction in specific networks, leading to improved performance (Jacobson et al., 2012). Alternatively, cathodal tDCS might inhibit a particular function as well, leading to enhanced performance in specific tasks, like faster reaction times (Tremblay et al., 2014).

The effect of prefrontal tDCS heavily depend on the state of the targeted neural network as well (Janacsek et al., 2015). In the online paradigm, tDCS influences networks already engaged in the task, whereas in the offline paradigm, it modifies neural activity beyond the stimulation period. Understanding these state-dependent effects is crucial for cognitive and behavioral studies, as factors like fatigue, task knowledge, and network connectivity can significantly influence the baseline neural state.

It should be noted that the exact electrode placement may also affect results. Specifically, results from a recent meta-analysis suggest that the effect on inhibitory control performance measures obtained in the Stop Signal Task and Go/No-go task may vary as a function of electrode placement, with more consistent results when the active electrode is placed over the right inferior frontal gyrus (Schroeder et al., 2020). Specifically, tDCS targeting the right inferior frontal gyrus demonstrated a medium effect size, whereas a stimulation site over the DLPFC region showed an overall null effect. Variation in results was attributed to the positioning of the return electrode, with extracephalic placement differing from various positions across the head. Factors related to electrode properties may have also played a role in shaping the outcomes. The size, shape, and conductivity of the electrodes, as well as the use of gels and saline solutions, may have influenced the distribution and intensity of the electric field

(Saturnino et al., 2015). However, further studies are needed to further assess the potential moderating role of electrode placement and features.

Another plausible explanation could be attributed to the sample characteristics. Each participant's unique brain structure, including factors like skull thickness and sulcal depth, could have resulted in divergent responses to tDCS (Laakso et al., 2015; Opitz et al., 2015; Truong et al., 2013). Research on binge eating (22–24) suggests that tDCS primarily affects inhibitory control in samples with considerable room for improvement. This hypothesis proposes that individuals with relatively weaker inhibitory control at baseline may experience more pronounced enhancements following tDCS intervention.

Conversely, in healthy individuals the influence of tDCS may not predominantly target frontal alpha asymmetry and inhibitory activity but rather attention control (Akil et al., 2023). Furthermore, frontal tDCS is known to produce more variable electric fields compared to other types of tDCS (Laakso et al., 2016), adding further complexity to the neural adjustment processes. Our findings underscore the importance of considering individual differences and optimizing stimulation protocols in future research.

Our correlation analysis revealed a significant connection between inhibitory brain activity during food-reward conditions and baseline frontal brain asymmetry, as indexed by resting-state frontal alpha asymmetry before the neurostimulation. Precisely, greater right-sided frontal brain activity compared to the left side was linked to reduced initial inhibitory activity (Stop N2), likely emanating from the inferior frontal gyrus (Schmajuk et al., 2006). However, it was also associated with heightened subsequent inhibitory control (Stop P3), which is thought to originate from the superior frontal gyrus (Kenemans & Kähkönen, 2010). To the best of our knowledge, this is the first study showing the dissociation between baseline frontal EEG alpha asymmetry and the timing differentials of early- and late-onset inhibitory brain activities within intrinsic reward contexts. However, some other research found simultaneous increase in N2 and P3 (Liu & Zhou, 2020).

The interaction between different regions is crucial for complex processes. The observed correlation might suggest a dynamic relationship between the inferior and superior frontal gyri in managing inhibitory control. Higher activity in one region may

trigger or facilitate inhibitory control processes in another region. Furthermore, the higher right frontal brain activity might signify a compensatory mechanism. When early-onset inhibitory activity originating from the inferior frontal gyrus is compromised, the brain might engage the superior frontal gyrus to enhance inhibitory control at a later stage.

Alternatively, the nature and demands of the task being performed could influence how inhibitory control is exerted. Based on this, the brain's inhibitory processes might operate differently at different stages of a task such as at early-onset inhibitory activity, associated with the inferior frontal gyrus, and late-onset inhibitory control involving the superior frontal gyrus to achieve optimal inhibitory control. It is important to note that the interpretation provided is speculative and would need to be validated through empirical research and neuroimaging studies.

Based on the Stop Signal Task results, a notable reduction in inhibitory control performance (i.e., increased SSRT) was observed as time progressed, which aligns with expectations due to factors like tiredness and fatigue. However, the event-related potential results revealed both reduced early-onset and enhanced late-onset inhibitory activity in the brain (indexed by the Stop N2 and the Stop P3, respectively) as time progressed in the food-reward context relative to the neutral context. These results suggest that despite the absence of a significant time and condition interaction concerning SSRTs, the post-test food-reward block posed a stronger inhibitory challenge. As a consequence, participants displayed an adapted response marked by increased inhibition-related activity in the brain. This adaptive neurophysiological response may reflect the brain's capacity to dynamically adjust and allocate cognitive resources in response to varying levels of inhibitory demand.

Prior studies have provided conflicting results such as increased N2 and P3 amplitudes during food-specific trials (Chami et al., 2019) and decreased P3 but not N2 in obese participants across all Go/No-go task conditions compared to normal-weight control participants. This suggests that P3 might serve as a more critical biomarker of inhibitory control deficits (Wang et al., 2022). However, it is important to note that variations in stimuli, paradigms, component timescales, and event-related potential analyses present challenges in synthesizing results across the existing literature.

The diversity in methodologies utilized calls for caution in drawing definitive conclusions from the available evidence. Future research could benefit from standardized protocols and methodologies to address these complexities, enabling more robust comparisons and a deeper understanding of the neurophysiological underpinnings of inhibitory control in various populations. Despite the valuable insights obtained from this research, several limitations require careful consideration. First of all, it is still controversial whether the DLPFC is a key region of this network (Mirabella, 2014). In addition, the right lateralization of the inhibitory network is not a ubiquitous accepted notion (Hampshire & Sharp, 2015; Swick et al., 2008). Potentially, neither the right nor the left lateralization alone affects inhibitory control (Mancini et al., 2019). Future studies should consider adjusting experimental settings for target regions (Mattia et al., 2012). tDCS involves generating an electric field in the brain tissue, modulating neuronal activity locally and in connected regions, presumably influencing cognitive functions, and ultimately, resulting in behavioral changes. This complex process involves many mediating and confounding variables (Bergmann & Hartwigsen, 2021). Additionally, the effectiveness of tDCS depends on numerous stimulation parameters such as duration, intensity, and electrode placement. Although the chosen intervention parameters were based on existing literature and logical reasoning, indeed, alternative stimulation settings might have yielded different outcomes, for example, placing the cathode on extracranial areas rather than the left DLPFC to minimize its impact on the right DLPFC. Our Bayesian analyses indicated that there is support for H_1 for SSRT. Using a relatively small-sample (n = 10) pilot study to identify the effect size of interest can yield extreme results. This can lead to an overestimation of the effect size, which, in turn, can result in an underpowered study. Furthermore, the effect size was calculated for the main effect of tDCS on the primary outcome, frontal alpha asymmetry, and effects may not translate to the inhibitory indices as captured in the more complex models in the study.

CHAPTER III

6. General Discussion

The four studies offer an in-depth exploration of the relationship between frontal alpha asymmetry and self-regulation, considering different emotional contexts and employing diverse interventions to thoroughly examine these dynamics. Despite methodological differences, these studies collectively demonstrate that the interpretation of frontal alpha asymmetry and self-regulation goes beyond a straightforward left-right hemispheric activation balance, revealing complicated neural processes that fluctuate based on individual characteristics and temporal dynamics. Table 6.1 summarizes the results and conclusion.

Table 6.1.

Summary of Aims, Results, and Conclusions from Studies

Summary	Aim	Result	Conclusion
S1	To explore the relationship between frontal alpha asymmetry and the effect of exposure to depression memes on depressive mood	Deficits in emotion regulation (assessed via a questionnaire) result in a higher depressive mood following exposure to depression memes compared to neutral images. Dispositional frontal alpha asymmetry does not influence the effects of depression memes on depressive mood.	Individuals with deficits in emotion regulation are more vulnerable to the effects of depression memes, while improvements in emotion regulation may eliminate it. Dispositional frontal alpha asymmetry should be interpreted cautiously as an index of emotion regulation.
S2	To explore the relationship between self-reported measures of self-regulation and depression, and frontal alpha asymmetry	There is overlap between the depression, anxiety, and stress scales. Self-reported self-regulation is negatively correlated with depression. Dispositional frontal alpha asymmetry is only associated with participants exhibiting moderate to extreme levels of anxiety. Higher right-frontal activity compared to the left, in other words, lower frontal alpha asymmetry, is linked with higher anxiety.	Problems with self-regulation may contribute to depression. Dispositional frontal alpha asymmetry may serve as a marker for anxiety, specific symptoms, or comorbid depression with anxiety. Higher right frontal activity compared to the left (lower frontal alpha asymmetry) in this context suggests the potential role of dispositional frontal alpha asymmetry in self-regulation even though no relation was observed with self-report measurements.
S3	To explore the relationship between unlilateral muscle contraction, frontal alpha asymmetry, and behavioral and brain	Unilateral left-hand muscle contraction is linked to higher right frontal activity, in other words, lower frontal alpha asymmetry, with no time or interaction effects. The Stop N2	The association between unilateral left-hand muscle contraction and higher right frontal activity may be attributed to a coincidental baseline frontal alpha

	activity indicators of inhibitory control	amplitude increased over time, suggesting greater inhibitory brain activity as the task progressed. In contrast, the Stop P3 amplitude decreased, indicating reduced inhibitory brain activity.	asymmetry, as no interaction effect was observed. The dissociation between Stop N2 and Stop P3 may reflect heightened conflict early in the task, followed by reduced engagement or altered task expectations later on, potentially driven by fatigue or decreased motivation.
S4	To explore the relationship between transcranial direct current stimulation, frontal alpha asymmetry, and behavioral and brain activity indicators of inhibitory control	We found no effect of transcranial direct current stimulation on frontal alpha asymmetry or on behavioral and electrophysiological indices of inhibitory control. Greater baseline right-sided frontal brain activity, in other words, lower frontal alpha asymmetry, compared to the left is linked to reduced initial inhibitory control (Stop N2), while also associated with increased late-onset inhibitory control (Stop P3). We also found a reduced early-onset and enhanced late-onset inhibitory brain activity (Stop N2 and Stop P3, respectively) over time in the food-reward context compared to the neutral condition.	The observed dissociation between frontal alpha asymmetry, decreased early-onset inhibitory activity, and increased late-onset inhibitory activity suggests a dynamic relationship between the inferior and superior frontal gyri in managing inhibitory control. Higher activity in one region may trigger or facilitate inhibitory processes in another. The higher right frontal brain activity may represent a compensatory mechanism. When early-onset inhibitory activity originating from the inferior frontal gyrus is compromised, the brain may recruit the superior frontal gyrus to enhance inhibitory control at a later stage.

Note. Abbreviations: S1 = Study 1, S2 = Study 2, S3 = Study 3, S4 = Study 4

The first study investigated whether dispositional frontal alpha asymmetry, as a potential indicator of emotion regulation ability, could predict the impact of depression memes on depressive mood. Memes were chosen for their relevance to real-life scenarios, particularly for social media users, enhancing the ecological validity of the findings. The study also allowed for a comparison between frontal alpha asymmetry and a self-report measure of emotion regulation, the DERS-16 (Bjureberg et al., 2015).

From a broader perspective, the results suggest that dispositional frontal alpha asymmetry may not reliably reflect situational changes, aligning with Coan's capability model (Coan et al., 2006), which posits that frontal alpha asymmetry is not a static marker in the resting state but instead dynamically shifts depending on context. While the self-report measure showed statistically significant role of emotion regulation in moderating the effects of exposure to depression memes on mood changes, frontal alpha

asymmetry did not, supporting the idea that frontal alpha asymmetry is situation-dependent.

Although our study did not yield statistically significant results with respect to frontal alpha asymmetry, the patterns observed under different conditions may provide valuable insights into its association with mood regulation and motivational processes. Specifically, the findings suggest that a higher frontal alpha asymmetry under eyes-closed conditions may be linked to reduced inhibitory control, potentially contributing to negative mood. In contrast, a lower frontal alpha asymmetry under eyes-open conditions might be indicative of heightened withdrawal motivation and reduced approach motivation. The dual roles of frontal alpha asymmetry in self-regulation suggest that not just the direction, but also magnitude of asymmetry may affect self-regulatory processes and mood. This remains unexplored.

The study demonstrated that emotion dysregulation leads to increased negative mood and depressive symptoms, aligning with previous research showing that individuals with depression often exhibit altered frontal alpha asymmetry, particularly reduced left-hemisphere activation/increased right-hemisphere activation (Coan & Allen, 2004; Harmon-Jones et al., 2010). Frontal alpha asymmetry may still be a candidate as an index of emotion regulation, particularly in cases of severe emotional problems or when frontal alpha asymmetry is highly sensitive to situational changes in healthy individuals. Recognizing these nuances could be important in research design.

The results of the first study led us to consider the relationship between dispositional frontal alpha asymmetry and self-report measurements of self-regulation and depression. Therefore, we investigated whether there is a correlation between frontal alpha asymmetry and self-report measurements of depression-related symptoms and self-regulation in the second study. Unsurprisingly, there were positive correlations between depression, anxiety, and stress, and a negative correlation between depression and self-regulation in self-report measurements. There was also a negative correlation between frontal alpha asymmetry and anxiety, indicating that greater withdrawal motivation is associated with higher levels of anxiety. Therefore, frontal alpha asymmetry scores may be more linked to anxiety than depression. Previous research similarly found a right

lateralization in anxiety and comorbid conditions (Methersul et al., 2008), but not in depression (van Der Vinne et al., 2017).

In the third and fourth studies, we aimed to manipulate frontal alpha asymmetry using non-invasive neuromodulation techniques, unilateral muscle contraction (UMC) and transcranial direct current stimulation (tDCS). We expected that this approach would yield more comprehensive insights into the causal relationship between frontal alpha asymmetry and self-regulatory processes.

We utilized intrinsic reward conditions, specifically food, distinguishing them from extrinsic rewards, such as money (Tsegaye et al., 2020). This approach enabled us to evaluate inhibitory control within the framework of both approach and avoidance motivations. Furthermore, this framework allowed us to investigate the effectiveness of the neuromodulation methods in modulating frontal alpha asymmetry and self-regulatory processes.

Food-reward contexts are presumed to activate the left DLPFC (Kelley et al., 2017). We expected that left-hand muscle contraction would reduce frontal alpha asymmetry, indicating increased withdrawal motivation and inhibitory activity, particularly in challenging contexts. We used bilateral muscle contraction (BMC) in the control group, allowing us to isolate the effects specifically attributed to left-hand muscle contraction. Without this approach, such as with right-hand muscle contraction, any observed effects could have been influenced by right-hand muscle contraction, making it difficult to pinpoint the source of the changes in frontal alpha asymmetry and inhibitory control.

The results suggested that UMC is correlated with lower frontal alpha asymmetry (higher right frontal activation relative to left activation) and higher withdrawal motivation. The finding suggests that UMC still holds promise for frontal alpha asymmetry. On the other hand, this could potentially be attributed to inherent group differences or the possibility that the effect of time was not sufficiently robust given the current sample size. More importantly, we observed significant temporal changes in the neural indices of inhibition, with an initial increase in the Stop N2 amplitude (associated with early inhibitory processing) and a subsequent decrease in Stop P3 amplitude (probably linked to response evaluation and conflict). The lack of group effect on

behavioral changes underscores the sensitivity of frontal alpha asymmetry as a neural marker.

The study's most significant finding was that, although UMC is a convenient method (e.g., cost-effective, easy to apply) for modulating frontal alpha asymmetry, it proved insufficient, even during the online (i.e., intervention) phase. Additionally, we demonstrated a potential dissociation between early- and late-onset inhibitory brain activity, both of which are influenced by temporal factors, though not specifically linked to frontal alpha asymmetry. Therefore, an immediate evaluation of inhibitory activity (e.g., in conflict processing) may potentially impair the ability to effectively regulate behavior. Here, it should be emphasized that a decrease in Stop P3 amplitude could also suggest a reduction in the efficiency of this later stage of inhibition, possibly due to cognitive overload, task difficulty, or fatigue. In other words, while individuals may become better at detecting the need for inhibition, they might struggle more with fully implementing the inhibitory response.

The fourth study utilized tDCS to directly manipulate frontal alpha asymmetry, aiming to enhance inhibitory control in a food-reward context. Contrary to expectations, no significant effects of tDCS on frontal alpha asymmetry or inhibitory control performance were observed. This outcome highlights the complex and often inconsistent nature of tDCS effects on cognition and behavior, which are influenced by numerous factors such as tDCS protocols and individual differences (Laakso et al., 2015; Opitz et al., 2015; Truong et al., 2013).

However, the study revealed a significant correlation between baseline frontal alpha asymmetry and distinct patterns of neural inhibition, suggesting that frontal alpha asymmetry may influence the temporal dynamics of inhibitory control. Lower dispositional frontal alpha asymmetry (greater withdrawal motivation) was associated with reduced early inhibitory activity, as reflected by the Stop N2, and increased late inhibitory activity, as indicated by the Stop P3, especially within the reward condition. This finding could imply a dynamic relationship between the inferior and superior frontal gyri in regulating inhibitory control. If the early inhibitory signals from the inferior frontal gyrus are low, the brain may recruit the superior frontal gyrus to reinforce inhibitory control at a subsequent phase. Additionally, elevated activity in the

right frontal region, as indicated by frontal alpha asymmetry, could also help the compensatory strategy.

On the other hand, these results are the exact opposite of the results in Study 3. The discrepancy regarding the temporal dissociation in Study 3 and 4 may be because of group effects. Specifically, whereas the tDCS intervention was completely passive for both groups, muscle contractions (including BMC) were active. We employed the same experimental design across the neuromodulation studies to ensure comparability, enabling us to draw consistent conclusions about the effects of the interventions on frontal alpha asymmetry and self-regulation. This underscores the notion that engagement with various types of neuromodulation techniques and the inherent characteristics of these methods may also lead to contradicting results, highlighting the necessity for a new experimental design.

Overall, the studies imply that frontal alpha asymmetry is a multifaceted neural marker with the capacity to provide nuanced insights into the connection between motivational states, inhibitory control, and emotional regulation. They suggest that interventions aimed at modulating frontal alpha asymmetry, whether through behavioral means such as UMC or neuromodulation techniques like tDCS, must consider the underlying neural dynamics and individual differences to achieve meaningful outcomes. This body of work therefore advances the understanding of frontal alpha asymmetry as more than just a static indicator of hemispheric dominance but as a marker deeply intertwined with internal and external processes.

The dissociation observed between the Stop N2 and stop P3 components holds significant implications for understanding the temporal dynamics of inhibitory control and their relationship with frontal alpha asymmetry. Typically, these two event-related potential components are considered markers of inhibitory process (Schmajuk et al., 2006; Kenemans & Kähkönen, 2010). However, our findings from two different studies support that these components may not only index inhibition but could also reflect additional processes such as response evaluation (Jollans et al., 2017), conflict monitoring (Groom & Cragg, 2015), and compensatory mechanisms.

Despite the valuable insights provided by these studies, several limitations must be acknowledged. First, the similarity of participant samples (e.g., university students, young adults) across studies may have contributed to the insignificant results. These individuals might already possess a certain level of self-regulatory capacity. It is crucial to replicate the studies with clinically diagnosed individuals, as this would likely provide greater variability in frontal alpha asymmetry and self-regulatory abilities. While the Stop N2 and P3 components are traditionally viewed as markers of inhibitory control, the observed patterns in our studies suggest they may also be influenced by other affective, behavioral, and cognitive processes. This complicates the interpretation of N2 and P3 amplitudes as pure indicators of inhibitory control, indicating that these event-related potential components may reflect a broader set of processes that unfold sequentially during task performance. Consequently, they could be regarded as distinct components and representative of different phenomena. While frontal alpha asymmetry has been widely used as a marker of frontal lobe activity, it is not a direct measure of neural inhibition or motivation and may interact with other brain regions and networks not captured in these studies (Mirabella, 2014). Finally, while the studies offer initial support for frontal alpha asymmetry as a neural marker sensitive to some various factors, they underscore the need for more refined experimental designs that account for temporal dynamics, individual variability, and the contextual specificity of frontal alpha asymmetry responses. Future research should aim to disentangle these factors to clarify the precise role of frontal alpha asymmetry in emotion regulation and inhibitory control, potentially leading to more targeted and effective intervention strategies.

Conclusion

In summary, the four studies presented in this research contribute to a deeper understanding of the relationship between frontal alpha asymmetry and self-regulation and highlight potential limitations in research designs. While the findings reinforce the notion that frontal alpha asymmetry is a dynamic marker, responsive to situational factors and individual differences, they also underscore the complexity inherent in interpreting its implications. The evidence suggests that frontal alpha asymmetry is not merely a static measure of hemispheric dominance but rather reflects a multifaceted connection of motivational states and inhibitory control dynamics. This highlights the importance of context and methodology in shaping outcomes related to frontal alpha

asymmetry. Notably, the discrepancies between the studies regarding the temporal dissociation of Stop N2 and Stop P3 components reveal that frontal alpha asymmetry's relationship with inhibitory control may extend beyond traditional paradigms. Instead, it may also encompass processes like response evaluation and conflict detection. This necessitates a more nuanced approach in future research. Despite the valuable insights gained, the limitations identified, including sample homogeneity and the potential influence of intervention types, emphasize the need for future investigations to explore these relationships in more diverse populations, including clinically diagnosed individuals. Such studies may yield richer data and facilitate a deeper understanding of the role of frontal alpha asymmetry in self-regulatory processes. Ultimately, this body of work serves as a starting point for future research endeavors, encouraging the exploration of frontal alpha asymmetry as a dynamic and context-sensitive marker that can inform the development of targeted intervention strategies aimed at enhancing self-regulation in various clinical and non-clinical settings.

Supplementary Materials of Study 1

Please visit the following link:
https://osf.io/b5ep9

Supplementary Materials of Study 2

Please visit the following link:
https://osf.io/yn8mw/?view_only=12f2073be1ad4186a95af6cfcc8d9116

Supplementary Materials of Study 3

Supplementary Table 4.1.

Bayesian Repeated Measures ANOVA Results for Frontal Alpha Asymmetry in Study 3

Models	BF_{10}	Error (%)
FAA F4-F3 (EO) (*n* = 57)		
Group	0.20	±0.03
Time	0.19	±0.03
Time + Group	0.03	±1.76
Time + Group + Time x Group	0.01	±1.45
FAA F4-F3 (EC) (*n* = 58)		
Group	0.23	±0.03
Time	0.19	±0.03
Time + Group	0.04	±2.15
Time + Group + Time x Group	0.01	±2.05
FAA F4-F3 (int) (*n* = 61)		
Group	0.26	±0.01
FAA F8-F7 (EO) (*n* = 57)		
Group	4.94	±0.01
Time	0.19	±0.03
Time + Group	0.95	±2.07
Time + Group + Time x Group	0.25	±2
FAA F8-F7 (EC) (*n* = 58)		
Group	0.23	±0.03
Time	0.23	±0.03
Time + Group	0.05	±2.14
Time + Group + Time x Group	0.01	±2.03
FAA F8-F7 (int) (*n* = 61)		
Group	0.26	±0.01

Note. Participants with missing values were excluded from the analyses. “Int” refers to the frontal alpha asymmetry scores during the bilateral and unilateral hand muscle contraction interventions.

Supplementary Table 4.2.

Repeated Measures ANOVA Results for Frontal Alpha Asymmetry with in Study 3

Variables	*df*	*F*	*p*	η_p^2
FAA F4-F3 (EO) (*n* = 56)				
Time	1	0.41	.661	0.005
Group	1	0.02	.882	<0.001
Time x Group	1	0.12	.887	0.001
Residuals	162			
FAA F8-F7 (EO) (*n* = 56)				
Time	1	0.02	.972	<0.001
Group	1	5.34	.022*	0.031
Time x Group	1	1.17	.310	0.014
Residuals	162			

Note. Participants with missing values were excluded from the analyses. The analysis includes all three time points (pre-intervention, during the intervention, and post-intervention).

Supplementary Table 4.3.

Bayesian Repeated Measures ANOVA Results for Stop Signal Task Performance in Study 3

Models (*n* = 57)	BF_{10}	Error (%)
Group	0.18	±0.07
Time	0.22	±0.06
Group + Time	0.03	±1.55
Time + Group + Time x Group	<0.01	±2.5
Condition	0.18	±0.07
Group + Condition	0.03	±2.02
Time + Condition	0.04	±1.12
Group + Time + Condition	<0.01	±2.78
Group + Time + Group x Time + Condition	<0.01	±9.86
Group + Condition + Group x Condition	<0.01	±2.56
Group + Time + Condition + Group x Condition	<0.01	±2.04
Group + Time + Group x	<0.01	±2.46

Time + Condition + Group x Condition		
Time + Condition + Time x Condition	<0.01	±2.4
Group + Time + Condition + Time x Condition	<0.01	±3.15
Group + Time + Group x Time + Condition + Time x Condition	<0.01	±3.85
Group + Time + Condition + Group x Condition + Time x Condition	<0.01	±3.03
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition	7.37	±3.14
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition + Group x Time x Condition	1.91	±4.74

Note. Participants with missing data and <10% inhibition rates were excluded from the analysis for the calculation of stop-signal reaction times.

Supplementary Table 4.4.

Bayesian Repeated Measures ANOVA Results for Stop N2 at 172-192 ms in Study 3

Models (n = 53)	BF_{10}	Error (%)
Group	0.39	±0.04
Time	1.37	±0.01
Group + Time	0.53	±1.75
Time + Group + Time x Group	0.11	±1.38
Condition	0.25	±0.05
Group + Condition	0.09	±1.11
Time + Condition	0.34	±2.57
Group + Time + Condition	0.14	±1.39
Group + Time + Group x Time + Condition	0.03	±1.88
Group + Condition + Group x Condition	0.09	±10.09

Group + Time + Condition + Group x Condition	0.12	±4.01
Group + Time + Group x Time + Condition + Group x Condition	0.02	±3.95
Time + Condition + Time x Condition	0.10	±2.12
Group + Time + Condition + Time x Condition	0.04	±5.46
Group + Time + Group x Time + Condition + Time x Condition	<0.01	±3.59
Group + Time + Condition + Group x Condition + Time x Condition	0.03	±2.84
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition	<0.01	±6.1
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition + Group x Time x Condition	<0.01	±4.66

Note. Participants with missing data and low segments (≤ 2) were excluded from the analyses.

Supplementary Table 4.5.

Bayesian Repeated Measures ANOVA Results for Stop P3 at 191-141 ms Study 3

Models (n = 51)	BF_{10}	Error (%)
Group	0.15	±0.07
Time	1.10	±0.02
Group + Time	0.16	±2.14
Time + Group + Time x Group	0.03	±2.06
Condition	0.23	±0.05
Group + Condition	0.03	±1.77
Time + Condition	0.25	±3.29
Group + Time + Condition	0.04	±10.41
Group + Time + Group x Time + Condition	<0.01	±3.5

Group + Condition + Group x Condition	0.01	±1.93
Group + Time + Condition + Group x Condition	0.01	±8.45
Group + Time + Group x Time + Condition + Group x Condition	<0.01	±9.88
Time + Condition + Time x Condition	0.06	±1.41
Group + Time + Condition + Time x Condition	<0.01	±2.87
Group + Time + Group x Time + Condition + Time x Condition	<0.01	±2.32
Group + Time + Condition + Group x Condition + Time x Condition	<0.01	±3.66
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition	<0.01	±4
Group + Time + Group x Time + Condition + Group x Condition + Time x Condition + Group x Time x Condition	<0.01	±4.46

Note. Participants with missing data and low segments (≤ 2) were excluded from the analyses.

Supplementary Table 4.6.

Repeated Measures of ANOVA Results for Stop N2 at 160-180 ms in Study 3

Variables (n = 53)	*df*	*F*	*p*	η_p^2
Time	1	4.40	.037*	0.021
Condition	1	1.39	.239	0.006
Group	1	1.12	.289	0.005
Time x Condition	1	1.43	.233	0.006
Time x Group	1	0.04	.841	<0.001
Condition x Group	1	2.86	.092	0.013
Time x Condition x Group	1	0.12	.722	<0.001
Residuals	204			

Note: Participants with missing data and low segments (≤ 2) were excluded from the analyses.

Supplementary Table 4.7.

Repeated Measures ANOVA Results for Stop Signal Task Performance of Participants Initially Assigned to the Neutral Condition First in Study 3

Variables (n = 16)	*df*	*F*	*p*	η_p^2
SSRT				
Time	1	0.20	.653	0.007
Group	1	0.15	.697	0.005
Time x Group	1	2.07	.161	0.068
Residuals	28			

Note. Participants with missing data and <10% inhibition rates were excluded from the analysis for the calculation of stop-signal reaction times. Only participants receiving the neutral condition after the intervention were included in the analysis.

Supplementary Table 4.8.

Repeated Measures ANOVA Results for Stop Signal Task Performance of Participants Initially Assigned to the Reward Condition First in Study 3

Variables (n = 15)	*df*	*F*	*p*	η_p^2
SSRT				
Time	1	<0.01	.981	<0.001
Group	1	<0.01	.940	<0.001
Time x Group	1	0.06	.809	0.002
Residuals	26			

Note. Participants with missing data and <10% inhibition rates were excluded from the analysis for the calculation of stop-signal reaction times. Only participants receiving the reward condition after the intervention were included in the model.

Supplementary Materials of Study 4

Supplementary Table 5.1.

Bayesian Repeated Measures ANOVA Results for Frontal Alpha Asymmetry in Study 4

Model Comparison

Variables	P(M)	P(M|data)	BF_M	BF_{01}	error %
FAA F4-F3 (EO) (*n* = 51)					
Null model (incl. subject)	0.200	0.429	3.010	1.000	
Group	0.200	0.361	2.261	1.189	1.651
Time	0.200	0.102	0.456	4.198	1.555
Time + Group	0.200	0.082	0.357	5.240	1.211
Time + Group + Time x Group	0.200	0.025	0.104	17.017	2.310
FAA F4-F3 (EC) (*n* = 51)					
Null model (incl. subject)	0.200	0.533	4.561	1.000	
Time	0.200	0.199	0.994	2.676	2.779
Group	0.200	0.179	0.870	2.982	0.777
Time + Group	0.200	0.067	0.286	7.988	1.993
Time + Group + Time x Group	0.200	0.023	0.093	23.354	2.919
FAA F8-F7 (EO) (*n = 54*)					
Null model (incl. subject)	0.200	0.680	8.481	1.000	
Group	0.200	0.154	0.725	4.427	1.061
Time	0.200	0.128	0.589	5.297	1.043
Time + Group	0.200	0.030	0.123	22.772	2.960
Time + Group + Time x Group	0.200	0.009	0.036	76.645	3.470
FAA F8-F7 (EC) (*n* = 51)					
Null model (incl. subject)	0.200	0.601	6.029	1.000	
Group	0.200	0.184	0.903	3.263	0.576
Time	0.200	0.141	0.656	4.267	1.457
Time + Group	0.200	0.043	0.179	13.999	2.003
Time + Group + Time x Group	0.200	0.031	0.127	19.553	2.370

Note. All models include subject.

Supplementary Table 5.2.

Repeated Measures ANOVA Results for Frontal Alpha Asymmetry in Right-Handed Participants in Study 4

Variables	*df*	*F*	*p*	η_p^2
FAA F4-F3 (EO) (*n* = 51)				
Group	1	4.41	.038*	0.043
Time	1	2.15	.145	0.032
Group x Time	1	0.39	.529	0.004
Error	98			
FAA F4-F3 (EC) (*n* = 51)				
Group	1	0.44	.508	0.004
Time	1	4.16	.044*	0.040
Group x Time	1	0.03	.856	<0.001
Error	98			
FAA F8-F7 (EO) (*n* = 54)				
Group	1	0.01	.918	<0.001
Time	1	0.25	.613	0.002
Group x Time	1	0.07	.076	<0.001
Error	104			
FAA F8-F7 (EC) (*n* = 51)				
Group	1	0.03	.847	<0.001
Time	1	2.46	.119	0.024
Group x Time	1	0.04	.827	<0.001
Error	98			

Note. Left-handed participants, participants with missing values and outliers (based on 3 standard deviations from the mean) were excluded from the analyses.

Supplementary Table 5.3.

Correlation Analysis Results for Pre-Intervention Frontal Alpha Asymmetry, Stop Signal Task Performance, and Event-Related Potentials in Study 4

Variables		1	2	3	4	5	6	7	8
FAA F4-F3 (EO)									
FAA F4-F3 (EC)		0.33*							
	p value	.013							
	N	55							
SSRT neutral		0.10	0.28*						
	p value	.453	.037						
	N	55	55						
SSRT reward		0.04	0.46	0.58* <0.00					
	p value	.730	.741	1					
	N	55	55	55					
Stop N2 neutral		0.12	0.12	-0.16	-0.17				
	p value	.357	.247	.229	.213				
	N	55	55	55	55				
Stop N2 reward		-0.48*	0.05	0.17	0.19	0.07			
	p value	<.001	.697	.212	.153	.575			
	N	55	55	59	55	55			
Stop P3 neutral		0.01	0.01	-0.10	-0.21	0.30	0.01		
	p value	.892	.902	.440	.109	.025	.914		
	N	55	55	55	55	55	55		
Stop P3 reward		-0.34*	0.12	-0.03	0.06	0.11	0.69* <0.00	0.31*	
	p value	.010	.365	.844	.647	.411	1	.021	
	N	55	55	55	55	55	55	55	

Note. Participants with negative stop-signal reaction times, inhibition rates under 10%, erroneous data were excluded from the analysis. 2-tailed correlation was conducted.

Supplementary Table 5.4.

Bayesian Repeated Measures ANOVA Results for Stop Signal Task Performance in Study 4

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{01}	error %
Null model (incl. subject)	0.053	0.002	0.042	1.000	
Time	0.053	0.351	9.735	0.007	1.008
Time + Condition	0.053	0.189	4.186	0.012	2.510
Time + Group	0.053	0.155	3.298	0.015	5.702
Time + Condition + Group	0.053	0.078	1.525	0.030	3.715
Time + Condition + Group + Condition x Group	0.053	0.064	1.229	0.036	3.107
Time + Condition + Time x Condition	0.053	0.054	1.033	0.043	5.629
Time + Group + Time x Group	0.053	0.028	0.521	0.083	2.550
Time + Condition + Group + Time x Condition	0.053	0.021	0.383	0.112	2.535

Note. All models include subject.

Supplementary Table 5.5.

Repeated Measures ANOVA Results for Stop Signal Task Performance in Right-Handed Participants in Study 4

Variables (n = 50)	*df*	*F*	*p*	η_p^2
SSRT				
Time	1	5.93	.015*	0.029
Condition	1	0.88	.347	0.004
Group	1	0.25	.613	0.001
Time x Condition	1	0.41	.519	0.002
Time x Group	1	0.30	.581	0.001
Condition x Group	1	0.84	.358	0.004
Time x Condition x Group	1	0.11	.734	<0.001
Error	192			

Note. Left-handed participants, participants with missing values, outliers, negative values, and inhibition rates under 10% were excluded from the analysis.

Supplementary Table 5.6.

Bayesian Repeated Measures of ANOVA Results for Stop N2 in Study 4

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{01}	error %
Null model (incl. subject)	0.053	0.488	17.153	1.000	
Group	0.053	0.158	3.386	3.082	1.210
Time	0.053	0.136	2.832	3.590	0.910
Condition	0.053	0.082	1.604	5.964	2.722
Time + Group	0.053	0.046	0.873	10.554	3.248
Condition + Group	0.053	0.026	0.480	18.791	1.613
Time + Condition	0.053	0.022	0.399	22.522	1.106
Time + Condition + Time x Condition	0.053	0.010	0.184	48.106	3.651
Time + Group + Time x Group	0.053	0.010	0.177	50.067	3.678
Time + Condition + Group	0.053	0.008	0.147	60.375	7.363
Condition + Group + Condition x Group	0.053	0.006	0.101	87.624	3.487
Time + Condition + Group + Time x Condition	0.053	0.003	0.063	139.422	8.635
Time + Condition + Group + Time x Group	0.053	0.002	0.029	304.231	4.501
Time + Condition + Group + Condition x Group	0.053	0.002	0.028	317.775	3.400
Time + Condition + Group + Time x Condition + Time x Group	0.053	7.157e -4	0.013	681.742	3.677
Time + Condition + Group + Time x Condition + Condition x Group	0.053	6.863e -4	0.012	711.011	3.007
Time + Condition + Group + Time x Group + Condition x Group	0.053	3.143e -4	0.006	1552.344	3.705
Time + Condition + Group + Time x Condition + Time x Group + Condition x Group	0.053	1.601e -4	0.003	3047.575	7.730
Time + Condition + Group + Time x Condition + Time x Group + Condition x Group + Time x Condition x Group	0.053	5.555e -5	1.000e -3	8784.066	11.710

Note. All models include subject

Supplementary Table 5.7.

Bayesian Repeated Measures of ANOVA Results for Stop P3 in Study 4

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{01}	error %
Null model (incl. subject)	0.053	0.289	7.329	1.000	
Group	0.053	0.237	5.595	1.220	1.096
Condition	0.053	0.079	1.545	3.660	1.313
Time	0.053	0.070	1.354	4.136	1.038
Condition + Group	0.053	0.065	1.261	4.419	1.491
Time + Condition + Time x Condition	0.053	0.059	1.139	4.863	1.845
Time + Group	0.053	0.057	1.087	5.079	1.686
Time + Condition + Group + Time x Condition	0.053	0.052	0.983	5.589	3.294
Time + Condition	0.053	0.020	0.362	14.696	5.533
Time + Condition + Group	0.053	0.016	0.297	17.804	2.761
Condition + Group + Condition x Group	0.053	0.013	0.231	22.878	1.880
Time + Group + Time x Group	0.053	0.012	0.216	24.450	2.242
Time + Condition + Group + Time x Condition + Time x Group	0.053	0.011	0.197	26.787	4.990
Time + Condition + Group + Time x Condition + Condition x Group	0.053	0.010	0.179	29.342	6.399
Time + Condition + Group + Time x Group	0.053	0.003	0.060	86.768	3.169
Time + Condition + Group + Condition x Group	0.053	0.003	0.055	94.739	2.292
Time + Condition + Group + Time x Condition + Time x Group + Condition x Group	0.053	0.002	0.039	132.351	10.759
Time + Condition + Group + Time x Group + Condition x Group	0.053	6.307e -4	0.011	458.742	3.144
Time + Condition + Group + Time x Condition + Time x Group + Condition x Group + Time x Condition x Group	0.053	5.311e -4	0.010	544.781	3.379

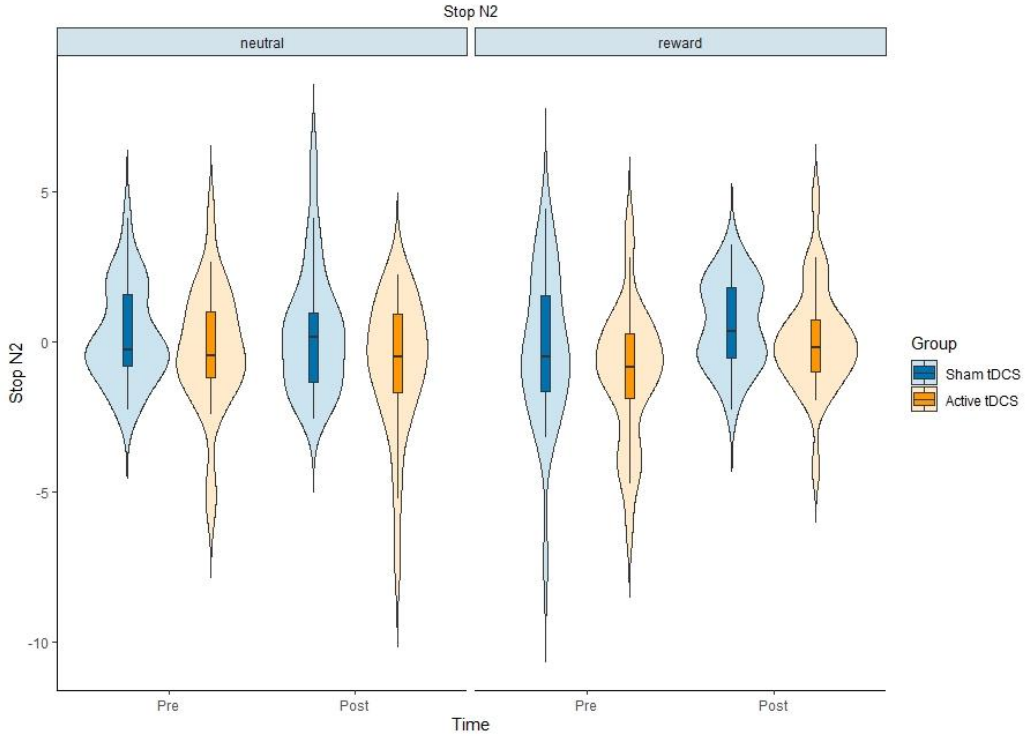
Note. All models include subject

Supplementary Table 5.8.

Repeated Measures of ANOVA Results for Event-Related Potentials in Right-Handed Participants in Study 4

Variables	*df*	*F*	*p*	η_p^2
Stop N2 at 166-188 ms. (F4) (*n* = 57)				
Time	1	1.96	.162	0.009
Condition	1	<0.01	.949	<0.001
Group	1	6.78	.009*	0.032
Time x Condition	1	4.46	.035*	0.021
Time x Group	1	0.04	.829	<0.001
Condition x Group	1	0.03	.857	<0.001
Time x Condition x Group	1	0.63	.425	0.003
Error	204			
Stop P3 at 211-271 ms. (Cz) (n = 57)				
Time	1	2.09	.149	0.010
Condition	1	0.87	.350	0.004
Group	1	8.59	.003*	0.040
Time x Condition	1	6.71	.010*	0.031
Time x Group	1	0.23	.627	0.001
Condition x Group	1	0.04	.836	<0.001
Time x Condition x Group	1	0.02	.888	<0.001
Error	204			

Note. Left-handed participants, participants with missing values, erroneous values, and outliers were excluded from the analyses.



Supplementary Figure 5.1. The figure shows the exact effect of time and condition interaction on the Stop N2. The x-axes represent the time factor; the y-axes represent the Stop N2 in microvolts.

References

Aghaei, S. (2012). Evolution of the World Wide Web : From Web 1.0 to Web 4.0. *International Journal of Web & Semantic Technology*, *3*, 1–10. https://doi.org/10.5121/ijwest.2012.3101

Akil, A. M., Cserjési, R., Németh, D., Nagy, T., Demetrovics, Z., & Logemann, H. N. A. (2023). The effect of transcranial direct current stimulation of the dorsolateral prefrontal cortex on behavioral and brain activity indices of visuospatial attention in intrinsic reward contexts. *BioRxiv*, 2023.08.01.551490. https://doi.org/10.1101/2023.08.01.551490

Akram, U., Drabble, J., Cau, G., Hershaw, F., Rajenthran, A., Lowe, M., Trommelen, C., & Ellis, J. (2020). Exploratory Study On The Role Of Emotion Regulation In Perceived Valence, Humour, And Beneficial Use Of Depressive Internet Memes In Depression. *Scientific Reports*, *10*. https://doi.org/10.1038/s41598-020-57953-4

Allen, J. J. B., Keune, P. M., Schönenberg, M., & Nusslock, R. (2018). Frontal EEG alpha asymmetry and emotion: From neural underpinnings and methodological considerations to psychopathology and social cognition. *Psychophysiology*, *55*(1), 1–6. https://doi.org/10.1111/psyp.13028

APA. (n.d.). DSM-5 (Diagnostic and Statistical Manual of Mental Disorders). In *Psychopathology and Psychotherapy: DSM-5 Diagnosis, Case Conceptualization, and Treatment*. https://doi.org/10.4324/9780203772287-23

Baayen, H., Davidson, D., & Bates, D. (2008). Mixed-effects modeling with crossed random effects for subjects and items. *Journal of Memory and Language*, *59*, 390–412. https://doi.org/10.1016/j.jml.2007.12.005

Barry, R. J., Clarke, A. R., Johnstone, S. J., Magee, C. A., & Rushby, J. A. (2007). EEG differences between eyes-closed and eyes-open resting conditions. *Clinical Neurophysiology*, *118*(12), 2765–2773. https://doi.org/https://doi.org/10.1016/j.clinph.2007.07.028

Bauhoff, S. (2011). Systematic self-report bias in health data: impact on estimating cross-sectional and treatment effects. *Health Services and Outcomes Research Methodology*, *11*(1), 44–53. https://doi.org/10.1007/s10742-011-0069-3

Baumeister, R. F., Heatherton, T. F., & Tice, D. M. (1994). Losing control: How and why people fail at self-regulation. In *Losing control: How and why people fail at self-regulation.* Academic Press.

Beaton, E. A., Schmidt, L. A., Ashbaugh, A. R., Santesso, D. L., Antony, M. M., &

McCabe, R. E. (2008). Resting and reactive frontal brain electrical activity (EEG) among a non-clinical sample of socially anxious adults: Does concurrent depressive mood matter? *Neuropsychiatric Disease and Treatment*, *4*(1), 187–192. https://doi.org/10.2147/ndt.s1388

Bergmann, T. O., & Hartwigsen, G. (2021). Inferring Causality from Noninvasive Brain Stimulation in Cognitive Neuroscience. *Journal of Cognitive Neuroscience*, *33*(2), 195–225. https://doi.org/10.1162/jocn_a_01591

Berkowitz, L. (1999). Anger. In *Handbook of cognition and emotion.* (pp. 411–428). John Wiley & Sons Ltd.

Bjureberg, J., Ljótsson, B., Tull, M., Hedman, E., Sahlin, H., Lundh, L.-G., Bjärehed, J., DiLillo, D., Messman, T., Hellner, C., & Gratz, K. (2015). Development and Validation of a Brief Version of the Difficulties in Emotion Regulation Scale: The DERS-16. *Journal of Psychopathology and Behavioral Assessment*, *38*. https://doi.org/10.1007/s10862-015-9514-x

Blechert, J., Sheppes, G., Tella, C., Williams, H., & Gross, J. (2012). See What You Think: Reappraisal Modulates Behavioral and Neural Responses to Social Stimuli. *Psychological Science*, *23*, 346–353. https://doi.org/10.1177/0956797612438559

Bradley, S. J. (2000). Affect regulation and the development of psychopathology. In *Affect regulation and the development of psychopathology.* Guilford Press.

Bradley, M. M. (2000). Emotion and motivation. In *Handbook of psychophysiology, 2nd ed.* (pp. 602–642). Cambridge University Press.

Braver, T. S. (2012). The variable nature of cognitive control: a dual mechanisms framework. *Trends in Cognitive Sciences*, *16*(2), 106–113. https://doi.org/https://doi.org/10.1016/j.tics.2011.12.010

Brown, K. W., Ryan, R. M., & Creswell, J. D. (2007). Mindfulness: Theoretical foundations and evidence for its salutary effects. *Psychological Inquiry*, *18*(4), 211–237. https://doi.org/10.1080/10478400701598298

Burgess, E. E., Sylvester, M. D., Morse, K. E., Amthor, F. R., Mrug, S., Lokken, K. L., Osborn, M. K., Soleymani, T., & Boggiano, M. M. (2016). Effects of transcranial direct current stimulation (tDCS) on binge eating disorder. In *International Journal of Eating Disorders* (Vol. 49, pp. 930–936). John Wiley & Sons. https://doi.org/10.1002/eat.22554

Carey, K., Neal, D., & Collins, S. (2004). A psychometric analysis of the Self-Regulation Questionnaire. *Addictive Behaviors*, *29*(2), 253–260.

https://doi.org/10.1016/j.addbeh.2003.08.001

Cavanagh, A., Wilson, C. J., Kavanagh, D. J., & Caputi, P. (2017). Differences in the expression of symptoms in men versus women with depression: A systematic review and meta-analysis. *Harvard Review of Psychiatry*, *25*(1), 29–38. https://doi.org/10.1097/HRP.0000000000000128

Chami, R., Cardi, V., Lautarescu, A., Mallorquí Bagué, N., & McLoughlin, G. (2019). Neural responses to food stimuli among individuals with eating and weight disorders: a systematic review of event-related potentials. *International Review of Psychiatry*, *31*, 1–14. https://doi.org/10.1080/09540261.2019.1622515

Choi, J.-S., Park, S. M., Roh, M.-S., Lee, J.-Y., Park, C.-B., Hwang, J. Y., Gwak, A. R., & Jung, H. Y. (2014). Dysfunctional inhibitory control and impulsivity in Internet addiction. *Psychiatry Research*, *215*(2), 424–428. https://doi.org/https://doi.org/10.1016/j.psychres.2013.12.001

Christensen, H., Jorm, A. F., Mackinnon, A. J., Korten, A. E., Jacomb, P. A., Henderson, A. S., & Rodgers, B. (1999). Age differences in depression and anxiety symptoms: a structural equation modelling analysis of data from a general population sample. *Psychological Medicine*, *29*(2), 325–339. https://doi.org/10.1017/S0033291798008150

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review*, *30*(2), 203–216. https://doi.org/https://doi.org/10.1016/j.cpr.2009.11.003

Coan, J. A., & Allen, J. J. B. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, *67*(1–2), 7–50. https://doi.org/10.1016/j.biopsycho.2004.03.002

Coan, J., Allen, J., & Mcknight, P. (2006). A capability model of individual differences in frontal EEG activity. *Biological Psychology*, *72*, 198–207. https://doi.org/10.1016/j.biopsycho.2005.10.003

Cohen, J. D. (2017). Cognitive control: Core constructs and current considerations. In *The Wiley handbook of cognitive control.* (pp. 3–28). Wiley Blackwell. https://doi.org/10.1002/9781118920497.ch1

Cole, J., Chengappa, D., Gershon, K., & Levine, J. (2001). Anxiety disorders and major depression, together or apart. *Depression and Anxiety*, *14*(2), 94–104. https://doi.org/10.1002/da.1051

Common European Framework of Reference for Languages: Learning, Teaching,

Assessment. (2001). Cambridge University Press.

Cooper, S. J. (2008). From Claude Bernard to Walter Cannon. Emergence of the concept of homeostasis. *Appetite*, *51*(3), 419–427. https://doi.org/https://doi.org/10.1016/j.appet.2008.06.005

Crawford, J. R., & Henry, J. D. (2003). The Depression Anxiety Stress Scales (DASS): Normative data and latent structure in a large non-clinical sample. *The British Journal of Clinical Psychology*, *42*(Pt 2), 111–131. https://doi.org/10.1348/014466503321903544

Crino, N., Touyz, S., & Rieger, E. (2019). How eating disordered and non-eating disordered women differ in their use (and effectiveness) of cognitive self-regulation strategies for managing negative experiences. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, *24*(5), 897–904. https://doi.org/10.1007/s40519-017-0448-z

Dan-Glauser, E., & Scherer, K. (2011). The Geneva affective picture database (GAPED): A new 730-picture database focusing on valence and normative significance. *Behavior Research Methods*, *43*, 468–477. https://doi.org/10.3758/s13428-011-0064-1

Davidson, R. J. (1998). Affective Style and Affective Disorders: Perspectives from Affective Neuroscience. *Cognition and Emotion*, *12*(3), 307–330. https://doi.org/10.1080/026999398379628

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-Withdrawal and Cerebral Asymmetry: Emotional Expression and Brain Physiology I. *Journal of Personality and Social Psychology*, *58*(2), 330–341. https://doi.org/10.1037/0022-3514.58.2.330

De Jong, R., Coles, M. G. H., & Logan, G. D. (1995). Strategies and mechanisms in nonselective and selective inhibitory motor control. In *Journal of Experimental Psychology: Human Perception and Performance* (Vol. 21, Issue 3, pp. 498–511). American Psychological Association. https://doi.org/10.1037/0096-1523.21.3.498

de Jong, R., Coles, M. G. H., Logan, G. D., & Gratton, G. (1990). In search of the point of no return: The control of response processes. In *Journal of Experimental Psychology: Human Perception and Performance* (Vol. 16, Issue 1, pp. 164–182). American Psychological Association. https://doi.org/10.1037/0096-1523.16.1.164

de Klerk, M. T., Smeets, P. A. M., & la Fleur, S. E. (2023). Inhibitory control as a potential treatment target for obesity. *Nutritional Neuroscience*, *26*(5), 429–444.

https://doi.org/10.1080/1028415X.2022.2053406

Disner, S. G., Beevers, C. G., Haigh, E. A. P., & Beck, A. T. (2011). Neural mechanisms of the cognitive model of depression. *Nature Reviews Neuroscience*, *12*(8), 467–477. https://doi.org/10.1038/nrn3027

Dru, V., & Cretenet, J. (2008). Influence of unilateral motor behaviors on the judgment of valenced stimuli. *Cortex*, *44*(6), 717–727. https://doi.org/10.1016/j.cortex.2006.11.004

Dubuc-Charbonneau, N., & Durand-Bush, N. (2015). Moving to Action: The Effects of a Self-Regulation Intervention on the Stress, Burnout, Well-Being, and Self-Regulation Capacity Levels of University Student-Athletes. *Journal of Clinical Sport Psychology*, *9*(2), 173–192. https://doi.org/10.1123/jcsp.2014-0036

Eisenberg, N., Hofer, C., & Vaughan, J. (2007). Effortful Control and Its Socioemotional Consequences. In *Handbook of emotion regulation.* (pp. 287–306). The Guilford Press.

Ekman, P., Friesen, W., O'Sullivan, M., Chan, A., Diacoyanni-Tarlatzis, I., Heider, K., Krause, R., LeCompte, W., Pitcairn, T., Ricci Bitti, P., Scherer, K., Tomita, M., & Tzavaras, A. (1987). Universals and Cultural Differences in the Judgments of Facial Expressions of Emotion. *Journal of Personality and Social Psychology*, *53*, 712–717. https://doi.org/10.1037/0022-3514.53.4.712

El Habbal Jadayel, R., Medlej, K., & Jadayel, J. (2018). MENTAL DISORDERS: A GLAMOROUS ATTRACTION ON SOCIAL MEDIA? *International Journal of Teaching & Education*, *7*, 465–476.

Elis, J. D., Schroder, H. S., Patrick, C. J., & Moser, J. S. (2017). Emotional reactivity and regulation in individuals with psychopathic traits: Evidence for a disconnect between neurophysiology and self-report. *Psychophysiology*, *54*(10), 1574–1585. https://doi.org/https://doi.org/10.1111/psyp.12903

Eysenck, M. W., & Fajkowska, M. (2018). Anxiety and depression: toward overlapping and distinctive features. *Cognition & Emotion*, *32*(7), 1391–1400. https://doi.org/10.1080/02699931.2017.1330255

Farhane-Medina, N. Z., Luque, B., Tabernero, C., & Castillo-Mayén, R. (2022). Factors associated with gender and sex differences in anxiety prevalence and comorbidity: A systematic review. *Science Progress*, *105*(4), 368504221135469. https://doi.org/10.1177/00368504221135469

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible

statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175–191. https://doi.org/10.3758/BF03193146

Feil, J., Sheppard, D., Fitzgerald, P. B., Yücel, M., Lubman, D. I., & Bradshaw, J. L. (2010). Addiction, compulsive drug seeking, and the role of frontostriatal mechanisms in regulating inhibitory control. *Neuroscience & Biobehavioral Reviews*, *35*(2), 248–275. https://doi.org/https://doi.org/10.1016/j.neubiorev.2010.03.001

Fingelkurts, A. A., & Fingelkurts, A. A. (2015). Altered Structure of Dynamic Electroencephalogram Oscillatory Pattern in Major Depression. *Biological Psychiatry*, *77*(12), 1050–1060. https://doi.org/https://doi.org/10.1016/j.biopsych.2014.12.011

Fingelkurts, A. A., & Fingelkurts, A. A. (2015). Altered structure of dynamic electroencephalogram oscillatory pattern in major depression. *Biological Psychiatry*, *77*(12), 1050–1060. https://doi.org/10.1016/j.biopsych.2014.12.011

Friehs, M. A., Frings, C., & Hartwigsen, G. (2021). Effects of single-session transcranial direct current stimulation on reactive response inhibition. *Neuroscience & Biobehavioral Reviews*, *128*, 749–765. https://doi.org/https://doi.org/10.1016/j.neubiorev.2021.07.013

Garcia-Martin, M., Ruiz, F., Bedoya-Valderrama, L., Segura Vargas, M., Peña-Vargas, A., Avila-Campos, J., Gomez, J., & Calle-Arciniegas, V. (2021). Inhibitory Control in Individuals with Clinical Levels of Depression and Anxiety Symptoms. *The Spanish Journal of Psychology*, *24*, 1–9. https://doi.org/10.1017/SJP.2021.18

Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality and Individual Differences*, *30*, 1311–1327. https://doi.org/10.1016/S0191-8869(00)00113-6

Gauggel, S., Rieger, M., & Feghoff, T.-A. (2004). Inhibition of ongoing responses in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery &Amp; Psychiatry*, *75*(4), 539 LP – 544. https://doi.org/10.1136/jnnp.2003.016469

Goldstein, R., & Volkow, N. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews. Neuroscience*, *12*, 652–669. https://doi.org/10.1038/nrn3119

Gorber, S. C., & Tremblay, M. S. (2016). *Self-Report and Direct Measures of Health: Bias and Implications BT - The Objective Monitoring of Physical Activity:*

Contributions of Accelerometry to Epidemiology, Exercise Science and Rehabilitation (R. J. Shephard & C. Tudor-Locke (eds.); pp. 369–376). Springer International Publishing. https://doi.org/10.1007/978-3-319-29577-0_14

Gotlib, I., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annual Review of Clinical Psychology*, *6*, 285–312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41–54. https://doi.org/10.1023/B:JOBA.0000007455.08539.94

Grimshaw, G., & Carmel, D. (2014). An asymmetric inhibition model of hemispheric differences in emotional processing. *Frontiers in Psychology*, *5*, 489. https://doi.org/10.3389/fpsyg.2014.00489

Groom, M. J., & Cragg, L. (2015). Differential modulation of the N2 and P3 event-related potentials by response conflict and inhibition. *Brain and Cognition*, *97*, 1–9. https://doi.org/https://doi.org/10.1016/j.bandc.2015.04.004

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. In *Journal of Personality and Social Psychology* (Vol. 74, Issue 1, pp. 224–237). American Psychological Association. https://doi.org/10.1037/0022-3514.74.1.224

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, *2*(3), 271–299. https://doi.org/10.1037/1089-2680.2.3.271

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, *10*(6), 214–219. https://doi.org/10.1111/1467-8721.00152

Gross, J. J. (2014). Emotion regulation: Conceptual and empirical foundations. In *Handbook of emotion regulation, 2nd ed.* (pp. 3–20). The Guilford Press.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011a). Taking one's lumps while doing the splits: A big tent perspective on emotion generation and emotion regulation. *Cognition and Emotion*, *25*(5), 789–793. https://doi.org/10.1080/02699931.2011.586590

Gross, J. J., Sheppes, G., & Urry, H. L. (2011b). Cognition and Emotion Lecture at the

2010 SPSP Emotion Preconference. *Cognition and Emotion*, *25*(5), 765–781. https://doi.org/10.1080/02699931.2011.555753

Hampshire, A., & Sharp, D. J. (2015). Contrasting network and modular perspectives on inhibitory control. *Trends in Cognitive Sciences*, *19*(8), 445–452. https://doi.org/https://doi.org/10.1016/j.tics.2015.06.006

Han, Y., Dai, Z., Chattun, M. R., Lin, P., Zhou, H., Wang, H., Yao, Z., & Lu, Q. (2020). Connectivity of the Frontal Cortical Oscillatory Dynamics Underlying Inhibitory Control During a Go/No-Go Task as a Predictive Biomarker in Major Depression. *Frontiers in Psychiatry*, *11*. https://doi.org/10.3389/fpsyt.2020.00707

Harmon-Jones, E. (2006). Unilateral right-hand contractions cause contralateral alpha power suppression and approach motivational affective experience. *Psychophysiology*, *43*(6), 598–603. https://doi.org/https://doi.org/10.1111/j.1469-8986.2006.00465.x

Harmon-Jones, E., Gable, P. A., & Peterson, C. K. (2010). The role of asymmetric frontal cortical activity in emotion-related phenomena: A review and update. *Biological Psychology*, *84*(3), 451–462. https://doi.org/10.1016/j.biopsycho.2009.08.010

Harmon-Jones, E., Harmon-Jones, C., & Summerell, E. (2017). On the Importance of Both Dimensional and Discrete Models of Emotion. *Behavioral Sciences*, *7*, 66. https://doi.org/10.3390/bs7040066

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology*, *7*(4), 476–489. https://doi.org/10.1037/0894-4105.7.4.476

Hellige, J. B. (1993). Hemispheric asymmetry: What's right and what's left. In *Hemispheric asymmetry: What's right and what's left.* Harvard University Press.

Hofmann, S., Moscovitch, D., Litz, B., Kim, H.-J., Davis, L., & Pizzagalli, D. (2005). The Worried Mind: Autonomic and Prefrontal Activation During Worrying. *Emotion*, *5*, 464–475. https://doi.org/10.1037/1528-3542.5.4.464

Houben, K., Nederkoorn, C., & Jansen, A. (2014). Eating on impulse: The relation between overweight and food-specific inhibitory control. *Obesity*, *22*(5), 2013–2015. https://doi.org/10.1002/oby.20670

IBM Corporation. (n.d.). *IBM SPSS Statistics for Windows* (No. 26). https://www.ibm.com/products/spss-statistics

Inzlicht, M., Werner, K., Briskin, J., & Roberts, B. (2020). Integrating Models of Self-

Regulation. *Annual Review of Psychology*, 72. https://doi.org/10.1146/annurev-psych-061020-105721

Iyer, M. B., Mattu, U., Grafman, J., Lomarev, M., Sato, S., & Wassermann, E. M. (2005). Safety and cognitive effect of frontal DC brain polarization in healthy individuals. *Neurology*, *64*(5), 872 LP – 875. https://doi.org/10.1212/01.WNL.0000152986.07469.E9

Jacobson, L., Koslowsky, M., & Lavidor, M. (2012). tDCS polarity effects in motor and cognitive domains: a meta-analytical review. *Experimental Brain Research*, *216*(1), 1–10. https://doi.org/10.1007/s00221-011-2891-9

Janacsek, K., Ambrus, G. G., Paulus, W., Antal, A., & Nemeth, D. (2015). Right Hemisphere Advantage in Statistical Learning: Evidence From a Probabilistic Sequence Learning Task. *Brain Stimulation*, *8*(2), 277–282. https://doi.org/https://doi.org/10.1016/j.brs.2014.11.008

JASP Team. (2024). No Title (Version 0.18.3).

Jesulola, E., Sharpley, C. F., Bitsika, V., Agnew, L. L., & Wilson, P. (2015). Frontal alpha asymmetry as a pathway to behavioural withdrawal in depression: Research findings and issues. *Behavioural Brain Research*, *292*, 56–67. https://doi.org/https://doi.org/10.1016/j.bbr.2015.05.058

Johnstone, T., Van Reekum, C., Urry, H., Kalin, N., & Davidson, R. (2007). Johnstone T, van Reekum CM, Urry HL, Kalin NH, Davidson RJ. Failure to regulate: counterproductive recruitment of top-down prefrontal-subcortical circuitry in major depression. J Neurosci 27: 8877-8884. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience*, *27*, 8877–8884. https://doi.org/10.1523/JNEUROSCI.2063-07.2007

Jollans, L., Whelan, R., Venables, L., Turnbull, O. H., Cella, M., & Dymond, S. (2017). Computational EEG modelling of decision making under ambiguity reveals spatio-temporal dynamics of outcome evaluation. *Behavioural Brain Research*, *321*, 28–35. https://doi.org/https://doi.org/10.1016/j.bbr.2016.12.033

Kaiser, A. K., Gnjezda, M.-T., Knasmüller, S., & Aichhorn, W. (2018). Electroencephalogram alpha asymmetry in patients with depressive disorders: Current perspectives. In Neuropsychiatric Disease and Treatment (Vol. 14, pp. 1493–1504). Dove Medical Press Ltd. https://doi.org/10.2147/NDT.S137776

Kalanthroff, E., Henik, A., Simpson, H. B., Todder, D., & Anholt, G. E. (2017). To Do or Not to Do? Task Control Deficit in Obsessive-Compulsive Disorder. *Behavior*

Therapy, *48*(5), 603–613. https://doi.org/https://doi.org/10.1016/j.beth.2017.01.004

Kappenman, E., & Luck, S. (2012). *The Oxford Handbook of Event-Related Potential Components*. https://doi.org/10.1093/oxfordhb/9780195374148.001.0001

Kekic, M., McClelland, J., Bartholdy, S., Boysen, E., Musiat, P., Dalton, B., Tiza, M., David, A. S., Campbell, I. C., & Schmidt, U. (2017). Single-Session Transcranial Direct Current Stimulation Temporarily Improves Symptoms, Mood, and Self-Regulatory Control in Bulimia Nervosa: A Randomised Controlled Trial. *PLOS ONE*, *12*(1), e0167606. https://doi.org/10.1371/journal.pone.0167606

Kelley, N. J., Hortensius, R., Schutter, D. J. L. G., & Harmon-Jones, E. (2017). The relationship of approach/avoidance motivation and asymmetric frontal cortical activity: A review of studies manipulating frontal asymmetry. *International Journal of Psychophysiology*, *119*, 19–30. https://doi.org/10.1016/j.ijpsycho.2017.03.001

Kemp, A., Griffiths, K., Felmingham, K., Shankman, S., Drinkenburg, P., Arns, M., Clark, C. R., & Bryant, R. (2010). Disorder specificity despite comorbidity: Resting EEG alpha asymmetry in major depressive disorder and post-traumatic stress disorder. *Biological Psychology*, *85*(2), 350–354. https://doi.org/10.1016/j.biopsycho.2010.08.001

Kenemans, J., & Kähkönen, S. (2010). How Human Electrophysiology Informs Psychopharmacology: From Bottom-up Driven Processing to Top-Down Control. *Neuropsychopharmacology : Official Publication of the American College of Neuropsychopharmacology*, *36*, 26–51. https://doi.org/10.1038/npp.2010.157

Kenemans, J., Schutte, I., van Bijnen, S., & Logemann, H. N. A. (2023). *How salience enhances inhibitory control: An analysis of electro-cortical mechanisms*. https://doi.org/10.1101/2022.01.12.475991

Kinney, K. L., Burkhouse, K. L., & Klumpp, H. (2019). Self-report and neurophysiological indicators of emotion processing and regulation in social anxiety disorder. *Biological psychology*, *142*, 126–131. https://doi.org/10.1016/j.biopsycho.2019.01.019

Kocovski, N. L., & Endler, N. S. (2000). Social anxiety, self-regulation, and fear of negative evaluation. *European Journal of Personality*, *14*(4), 347–358. https://doi.org/https://doi.org/10.1002/1099-0984(200007/08)14:4<347::AID-PER381>3.0.CO;2-7

Kok, A., Ramautar, J. R., De Ruiter, M. B., Band, G. P. H., & Ridderinkhof, K. R.

(2004). ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology*, *41*(1), 9–20. https://doi.org/https://doi.org/10.1046/j.1469-8986.2003.00127.x

Koole, S. (2009). The psychology of emotion regulation: An integrative review. *Cognition & Emotion - COGNITION EMOTION*, *23*, 4–41. https://doi.org/10.1080/02699930802619031

Kring, A. M., & Werner, K. H. (2004). Emotion Regulation and Psychopathology. In *The regulation of emotion.* (pp. 359–385). Lawrence Erlbaum Associates Publishers.

Laakso, I., Tanaka, S., Koyama, S., De Santis, V., & Hirata, A. (2015). Inter-subject Variability in Electric Fields of Motor Cortical tDCS. *Brain Stimulation*, *8*(5), 906–913. https://doi.org/https://doi.org/10.1016/j.brs.2015.05.002

Laakso, I., Tanaka, S., Mikkonen, M., Koyama, S., Sadato, N., & Hirata, A. (2016). Electric fields of motor and frontal tDCS in a standard brain space: A computer simulation study. *NeuroImage*, *137*. https://doi.org/10.1016/j.neuroimage.2016.05.032

Langens, T. A., & Mörth, S. (2003). Repressive coping and the use of passive and active coping strategies. *Personality and Individual Differences*, *35*(2), 461–473. https://doi.org/https://doi.org/10.1016/S0191-8869(02)00207-6

Lansbergen, M., Böcker, K., Bekker, E., & Kenemans, J. (2007). Neural correlates of stopping and self-reported impulsivity. *Clinical Neurophysiology : Official Journal of the International Federation of Clinical Neurophysiology*, *118*, 2089–2103. https://doi.org/10.1016/j.clinph.2007.06.011

Lapenta, O. M., Sierve, K. Di, de Macedo, E. C., Fregni, F., & Boggio, P. S. (2014). Transcranial direct current stimulation modulates ERP-indexed inhibitory control and reduces food consumption. *Appetite*, *83*, 42–48. https://doi.org/https://doi.org/10.1016/j.appet.2014.08.005

Larose, R., Eastin, M., & Gregg, J. (2001). Reformulating the Internet Paradox: Social Cognitive Explanations of Internet Use and Depression. *Journal of Online Behavior*, *1*.

Leuchter, A., Cook, I., Hunter, A., Cai, C., & Horvath, S. (2012). Resting-state quantitative electroencephalography reveals increased neurophysiologic connectivity in depression. *PLoS One*, *7*(2), e32508. https://doi.org/10.1371/journal.pone.0032508

Liu, L., & Zhou, R. (2020). The Functional Role of Individual Alpha-Based Frontal Asymmetry in the Processing of Fearful Faces. *Frontiers in Psychology*, *11*, 1412. https://doi.org/10.3389/fpsyg.2020.01412

Logan, G. D., Cowan, W. B., & Davis, K. A. (1984). On the ability to inhibit simple and choice reaction time responses: A model and a method. *Journal of Experimental Psychology: Human Perception and Performance*, *10*(2), 276–291. https://doi.org/10.1037/0096-1523.10.2.276

Logemann, H.N.A, Böcker, K., Deschamps, P., Kemner, C., & Kenemans, J. (2013). The effect of noradrenergic attenuation by clonidine on inhibition in the stop signal task. *Pharmacology, Biochemistry, and Behavior*, *110*. https://doi.org/10.1016/j.pbb.2013.06.007

Logemann, H. N., Böcker, K. B., Deschamps, P. K., Kemner, C., & Kenemans, J. L. (2014a). The effect of attenuating noradrenergic neurotransmission by clonidine on brain activity measures of visuospatial attention. *Human psychopharmacology*, *29*(1), 46–54. https://doi.org/10.1002/hup.2367

Logemann, H N A, Böcker, K. B. E., Deschamps, P. K. H., Kemner, C., & Kenemans, J. L. (2014b). The effect of the augmentation of cholinergic neurotransmission by nicotine on EEG indices of visuospatial attention. *Behavioural Brain Research*, *260*, 67–73. https://doi.org/https://doi.org/10.1016/j.bbr.2013.11.044

Love, J., Selker, R., Marsman, M., Jamil, T., Dropmann, D., Verhagen, J., Ly, A., Gronau, Q. F., Šmíra, M., Epskamp, S., Matzke, D., Wild, A., Knight, P., Rouder, J. N., Morey, R. D., & Wagenmakers, E.-J. (2019). JASP: Graphical Statistical Software for Common Statistical Designs. *Journal of Statistical Software*, *88*(2 SE-Articles), 1–17. https://doi.org/10.18637/jss.v088.i02

Lovibond, S. H., & Lovibond, P. F. (1995). Manual for the Depression Anxiety Stress Scales (2nd ed.). Sydney: Psychology Foundation of Australia.

Mancini, C., Cardona, F., Baglioni, V., Panunzi, S., Pantano, P., Suppa, A., & Mirabella, G. (2018). Inhibition is impaired in children with obsessive-compulsive symptoms but not in those with tics. *Movement Disorders*, *33*(6), 950–959. https://doi.org/https://doi.org/10.1002/mds.27406

Mancini, C., & Mirabella, G. (2021). Handedness Does Not Impact Inhibitory Control, but Movement Execution and Reactive Inhibition Are More under a Left-Hemisphere Control. *Symmetry*, *13*. https://doi.org/10.3390/sym13091602

Mancini, C., Modugno, N., Santilli, M., Pavone, L., Grillea, G., Morace, R., &

Mirabella, G. (2019). Unilateral Stimulation of Subthalamic Nucleus Does Not Affect Inhibitory Control. *Frontiers in Neurology*, *9*. https://www.frontiersin.org/journals/neurology/articles/10.3389/fneur.2018.01149

Mathersul, D., Williams, L. M., Hopkinson, P. J., & Kemp, A. H. (2008). Investigating models of affect: Relationships among EEG alpha asymmetry, depression, and anxiety. In. *Emotion (Washington, D.C.)*, *8*(4), 560–572. https://doi.org/10.1037/a0012811

Mathôt, S., Schreij, D., & Theeuwes, J. (2012). OpenSesame: An open-source, graphical experiment builder for the social sciences. *Behavior Research Methods*, *44*(2), 314–324. https://doi.org/10.3758/s13428-011-0168-7

Mattia, M., Spadacenta, S., Pavone, L., Quarato, P., Esposito, V., Sparano, A., Sebastiano, F., Di Gennaro, G., Morace, R., Cantore, G., & Mirabella, G. (2012). Stop-event-related potentials from intracranial electrodes reveal a key role of premotor and motor cortices in stopping ongoing movements . In *Frontiers in Neuroengineering* (Vol. 5). https://www.frontiersin.org/articles/10.3389/fneng.2012.00012

McNair, D., Lorr, M., & Droppleman, C. (1971). The Profile of Mood States Manual. *San Diego: EdITS Educational and Industrial Testing Service*.

Milner, R. (2016). *The World Made Meme: Public Conversations and Participatory MediaPublic Conversations and Participatory Media*. https://doi.org/10.7551/mitpress/9780262034999.001.0001

Miltner, K. (2014). "There's no place for lulz on LOLCats": The role of genre, gender, and group identity in the interpretation and enjoyment of an Internet meme. *First Monday*, *19*. https://doi.org/10.5210/fm.v19i8.5391

Mirabella, G. (2021). Inhibitory control and impulsive responses in neurodevelopmental disorders. *Developmental Medicine & Child Neurology*, *63*(5), 520–526. https://doi.org/https://doi.org/10.1111/dmcn.14778

Mirabella, G. (2014). Should I stay or should I go? Conceptual underpinnings of goal-directed actions. *Frontiers in Systems Neuroscience*, *8*, 206. https://doi.org/10.3389/fnsys.2014.00206

Mirabella, G. (2023). *Beyond Reactive Inhibition: Unpacking the Multifaceted Nature of Motor Inhibition*. *13*, 804. https://doi.org/10.3390/brainsci13050804

Monai, H., & Hirase, H. (2017). Astrocytes as a target of transcranial direct current stimulation (tDCS) to treat depression. *Neuroscience Research*, *126*.

https://doi.org/10.1016/j.neures.2017.08.012

Monai, H., Ohkura, M., Tanaka, M., Oe, Y., Konno, A., Hirai, H., Mikoshiba, K., Itohara, S., Nakai, J., Iwai, Y., & Hirase, H. (2016). Calcium imaging reveals glial involvement in transcranial direct current stimulation-induced plasticity in mouse brain. *Nature Communications*, *7*(1), 11100. https://doi.org/10.1038/ncomms11100

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? In *Psychological Bulletin* (Vol. 126, Issue 2, pp. 247–259). American Psychological Association. https://doi.org/10.1037/0033-2909.126.2.247

Neuhaus, A., Bajbouj, M., Kienast, T., Kalus, P., von Haebler, D., Winterer, G., & Gallinat, J. (2006). Persistent dysfunctional frontal lobe activation in former smokers. In *Psychopharmacology* (Vol. 186, Issue 2, pp. 191–200). Springer. https://doi.org/10.1007/s00213-006-0366-7

Neumann, R., Förster, J., & Strack, F. (2003). Motor compatibility: The bidirectional link between behavior and evaluation. In *The psychology of evaluation: Affective processes in cognition and emotion.* (pp. 371–391). Lawrence Erlbaum Associates Publishers.

Nexus-*32*. (n.d.). Mind Media. www.mindmedia.com

Niedenthal, P., Barsalou, L., Winkielman, P., Krauth-Gruber, S., & Ric, F. (2005). Embodiment in Attitudes, Social Perception, and Emotion. *Personality and Social Psychology Review : An Official Journal of the Society for Personality and Social Psychology, Inc*, *9*, 184–211. https://doi.org/10.1207/s15327957pspr0903_1

Noël, X., Sferrazza, R., Van der Linden, M., Paternot, J., Verhas, M., Hanak, C., Pelc, I., & Verbanck, P. (2002). Contribution of frontal cerebral blood flow measured by 99mTc-BICISATE SPECT and executive function deficits to predicting treatment outcome in alcohol-dependent patients. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *37*, 347–354. https://doi.org/10.1093/alcalc/37.4.347

Opitz, A., Paulus, W., Will, S., Antunes, A., & Thielscher, A. (2015). Determinants of the electric field during transcranial direct current stimulation. *NeuroImage*, *109*. https://doi.org/10.1016/j.neuroimage.2015.01.033

Palmiero, M., & Piccardi, L. (2017). Frontal EEG asymmetry of mood: A mini-review. *Frontiers in Behavioral Neuroscience*, *11*(November), 1–8. https://doi.org/10.3389/fnbeh.2017.00224

R software: a tool analysing experimental data. (2016).

Rosenman, R., Tennekoon, V., & Hill, L. G. (2011). Measuring bias in self-reported data. *International Journal of Behavioural and Healthcare Research*, *2*(4), 320–332. https://doi.org/10.1504/IJBHR.2011.043414

Russell, J. (1980). A Circumplex Model of Affect. *Journal of Personality and Social Psychology*, *39*, 1161–1178. https://doi.org/10.1037/h0077714

Salk, R. H., Hyde, J. S., & Abramson, L. Y. (2017). Gender differences in depression in representative national samples: Meta-analyses of diagnoses and symptoms. *Psychological Bulletin*, *143*(8), 783–822. https://doi.org/10.1037/bul0000102

Saturnino, G., Antunes, A., & Thielscher, A. (2015). On the importance of electrode parameters for shaping electric field patterns generated by tDCS. *NeuroImage*, *120*. https://doi.org/10.1016/j.neuroimage.2015.06.067

Schaakxs, R., Comijs, H. C., van der Mast, R. C., Schoevers, R. A., Beekman, A. T. F., & Penninx, B. W. J. H. (2017). Risk factors for depression: Differential across age? *The American Journal of Geriatric Psychiatry: Official Journal of the American Association for Geriatric Psychiatry*, *25*(9), 966–977. https://doi.org/10.1016/j.jagp.2017.04.004

Scheepens, D. S., van Waarde, J. A., Lok, A., de Vries, G., Denys, D. A. J. P., & van Wingen, G. A. (2020). The link between structural and functional brain abnormalities in depression: A systematic review of multimodal neuroimaging studies. *Frontiers in Psychiatry*, *11*, 485. https://doi.org/10.3389/fpsyt.2020.00485

Schestatsky, P., Morales-Quezada, L., & Fregni, F. (2013). Simultaneous EEG monitoring during transcranial direct current stimulation. *Journal of Visualized Experiments : JoVE*, *76*, 1–11. https://doi.org/10.3791/50426

Schiff, B. B., & Lamon, M. (1994). Inducing emotion by unilateral contraction of hand muscles. In *Cortex: A Journal Devoted to the Study of the Nervous System and Behavior* (Vol. 30, Issue 2, pp. 247–254). Masson Italia. https://doi.org/10.1016/S0010-9452(13)80196-7

Schmajuk, M., Liotti, M., Busse, L., & Woldorff, M. G. (2006). Electrophysiological activity underlying inhibitory control processes in normal adults. *Neuropsychologia*, *44*(3), 384–395. https://doi.org/10.1016/j.neuropsychologia.2005.06.005

Schmeichel, B. J., Demaree, H. A., Robinson, J. L., & Pu, J. (2006). Ego depletion by response exaggeration. *Journal of Experimental Social Psychology*, *42*(1), 95–102.

https://doi.org/https://doi.org/10.1016/j.jesp.2005.02.005

Schroeder, P. A., Schwippel, T., Wolz, I., & Svaldi, J. (2020). Meta-analysis of the effects of transcranial direct current stimulation on inhibitory control. *Brain Stimulation*, *13*(5), 1159–1167. https://doi.org/https://doi.org/10.1016/j.brs.2020.05.006

Senqing, Q., Basanovic, J., Wang, L., Xiang, S., Hu, W., & Yi, X. (2020). Regulation of Negative Emotions through Positive Reappraisal and Distancing in High-Trait-Anxious Women. *Journal of Affective Disorders*, *267*. https://doi.org/10.1016/j.jad.2020.02.027

Shifman, L. (2014). *Memes in Digital Culture*. https://doi.org/10.7551/mitpress/9429.001.0001

Smith, E. E., Reznik, S. J., Stewart, J. L., & Allen, J. J. B. (2017a). Assessing and conceptualizing frontal EEG asymmetry: An updated primer on recording, processing, analyzing, and interpreting frontal alpha asymmetry. *International Journal of Psychophysiology*, *111*, 98–114. https://doi.org/10.1016/j.ijpsycho.2016.11.005

Smith, E. E., Reznik, S. J., Stewart, J. L., & Allen, J. J. B. (2017b). Assessing and conceptualizing frontal EEG asymmetry: An updated primer on recording, processing, analyzing, and interpreting frontal alpha asymmetry. *International Journal of Psychophysiology : Official Journal of the International Organization of Psychophysiology*, *111*, 98–114. https://doi.org/10.1016/j.ijpsycho.2016.11.005

Spielberg, J. M., Stewart, J. L., Levin, R. L., Miller, G. A., & Heller, W. (2008). Prefrontal Cortex, Emotion, and Approach/Withdrawal Motivation. *Social and Personality Psychology Compass*, *2*(1), 135–153. https://doi.org/https://doi.org/10.1111/j.1751-9004.2007.00064.x

Stoet, G. (2010). PsyToolkit: A software package for programming psychological experiments using Linux. *Behavior Research Methods*, *42*(4), 1096–1104. https://doi.org/10.3758/BRM.42.4.1096

Stoet, G. (2016). PsyToolkit: A Novel Web-Based Method for Running Online Questionnaires and Reaction-Time Experiments. *Teaching of Psychology*, *44*. https://doi.org/10.1177/0098628316677643

Stoet, G. (2017). PsyToolkit: A Novel Web-Based Method for Running Online Questionnaires and Reaction-Time Experiments. *Teaching of Psychology*, *44*(1), 24–31. https://doi.org/10.1177/0098628316677643

Strack, F., Martin, L., & Stepper, S. (1988). Inhibiting and Facilitating Conditions of the Human Smile: A Nonobtrusive Test of the Facial Feedback Hypothesis. *Journal of Personality and Social Psychology*, *54*, 768–777. https://doi.org/10.1037/0022-3514.54.5.768

Strauman, T. J. (2002). Self-Regulation and Depression. *Self and Identity*, *1*(2), 151–157. https://doi.org/10.1080/152988602317319339

Swick, D., Ashley, V., & Turken, A. (2008). Left inferior fronal gyrus is critical for response inhibition. *BMC Neuroscience*, *9*, 102. https://doi.org/10.1186/1471-2202-9-102

Thiruchselvam, R., Blechert, J., Sheppes, G., Rydstrom, A., & Gross, J. J. (2011). The temporal dynamics of emotion regulation: An EEG study of distraction and reappraisal. *Biological Psychology*, *87*(1), 84–92. https://doi.org/https://doi.org/10.1016/j.biopsycho.2011.02.009

Thompson, R. (1991). Emotional Regulation and Emotional Development. *Educational Psychology Review*, *3*, 269–307. https://doi.org/10.1007/BF01319934

Tice, D. M., & Bratslavsky, E. (2000). Giving in to feel good: The place of emotion regulation in the context of general self-control. *Psychological Inquiry*, *11*(3), 149–159. https://doi.org/10.1207/S15327965PLI1103_03

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: If you feel bad, do it! In *Journal of Personality and Social Psychology* (Vol. 80, Issue 1, pp. 53–67). American Psychological Association. https://doi.org/10.1037/0022-3514.80.1.53

Tremblay, S., Lepage, J., Latulipe-Loiselle, A., Fregni, F., Pascual-Leone, A., & Théoret, H. (2014). The Uncertain Outcome of Prefrontal tDCS. *Brain Stimulation*, *7*. https://doi.org/10.1016/j.brs.2014.10.003

Truong, D., Magerowski, G., Blackburn, G., Bikson, M., & Alonso-Alonso, M. (2013). Computational modeling of transcranial direct current stimulation (tDCS) in obesity: Impact of head fat and dose guidelines. *NeuroImage : Clinical*, *2*, 759–766. https://doi.org/10.1016/j.nicl.2013.05.011

Tsegaye, A., Guo, C., Stoet, G., Cserjési, R., Kökönyei, G., & Logemann, H. N. A. (2022). The relationship between reward context and inhibitory control, does it depend on BMI, maladaptive eating, and negative affect? *BMC Psychology*, *10*(1), 4. https://doi.org/10.1186/s40359-021-00712-5

Vallortigara, G., & Rogers, L. (2005). Survival with an asymmetrical brain: Advantages

and disadvantages of cerebral lateralization. *The Behavioral and Brain Sciences*, *28*, 575–589; discussion 589. https://doi.org/10.1017/S0140525X05000105

van der Vinne, N., Vollebregt, M. A., van Putten, M. J. A. M., & Arns, M. (2017). Frontal alpha asymmetry as a diagnostic marker in depression: Fact or fiction? A meta-analysis. *NeuroImage. Clinical*, *16*, 79–87. https://doi.org/10.1016/j.nicl.2017.07.006

Van Dillen, L. F., & Koole, S. L. (2007). Clearing the mind: A working memory model of distraction from negative mood. In *Emotion* (Vol. 7, Issue 4, pp. 715–723). American Psychological Association. https://doi.org/10.1037/1528-3542.7.4.715

Vanderhasselt, M.-A., Koster, E., Onraedt, T., Bruyneel, L., Goubert, L., & De Raedt, R. (2014). Adaptive cognitive emotion regulation moderates the relationship between dysfunctional attitudes and depressive symptoms during a stressful life period: A prospective study. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 291–296. https://doi.org/10.1016/j.jbtep.2014.01.003

Verbruggen, F., Chambers, C. D., & Logan, G. D. (2013). Fictitious Inhibitory Differences: How Skewness and Slowing Distort the Estimation of Stopping Latencies. *Psychological Science*, *24*(3), 352–362. https://doi.org/10.1177/0956797612457390

Vohs, K. D., & Baumeister, R. F. (2004). Understanding Self Regulation. In *Handbook of Self-Regulation : Research, Theory, and Applications*.

Wagenmakers, E.-J., Marsman, M., Jamil, T., Ly, A., Verhagen, J., Love, J., Selker, R., Gronau, Q. F., Šmíra, M., Epskamp, S., Matzke, D., Rouder, J. N., & Morey, R. D. (2018). Bayesian inference for psychology. Part I: Theoretical advantages and practical ramifications. In *Psychonomic Bulletin & Review* (Vol. 25, Issue 1, pp. 35–57). Springer. https://doi.org/10.3758/s13423-017-1343-3

Wagenmakers, E.-J., Wetzels, R., Borsboom, D., & van der Maas, H. L. J. (2011). Why psychologists must change the way they analyze their data: The case of psi: Comment on Bem (2011). In *Journal of Personality and Social Psychology* (Vol. 100, Issue 3, pp. 426–432). American Psychological Association. https://doi.org/10.1037/a0022790

Wang, J., Wang, H., Yu, H., Wang, J., Guo, X., Tong, S., Bao, Y., & Hong, X. (2022). Neural mechanisms of inhibitory control deficits in obesity revealed by P3 but not N2 event-related potential component. *Appetite*, *171*, 105908. https://doi.org/10.1016/j.appet.2021.105908

Yadollahpour, A., & Jalilifar, M. (2019). Single session anodal transcranial direct current stimulation alters the prefrontal and temporal alpha asymmetrical indexes in healthy individual performing a visual attention task. *Brain Stimulation*, *12*, 405–406. https://doi.org/10.1016/j.brs.2018.12.310

Zandbelt, B., Bloemendaal, M., Neggers, S., Kahn, R., & Vink, M. (2013). Expectations and violations: Delineating the neural network of proactive inhibitory control. *Human Brain Mapping*, *34*. https://doi.org/10.1002/hbm.22047

Zhang, J., Hua, Y., Xiu, L., Oei, T. P., & Hu, P. (2020). Resting state frontal alpha asymmetry predicts emotion regulation difficulties in impulse control. *Personality and Individual Differences*, *159*(January), 109870. https://doi.org/10.1016/j.paid.2020.109870

Zheng, D., Oka, T., Bokura, H., & Yamaguchi, S. (2008). The Key Locus of Common Response Inhibition Network for No-go and Stop Signals. *Journal of Cognitive Neuroscience*, *20*, 1434–1442. https://doi.org/10.1162/jocn.2008.20100

Appendices

Appendix A - Profile of Mood States Short Form (POMS-SF)

Below is a list of words that describe feelings people have. Please indicate your feelings.

- Unhappy
- Sad
- Blue
- Hopeless
- Discouraged
- Miserable
- Helpless
- Worthless
- {reverse} Lively
- {reverse} Active
- {reverse} Energetic
- {reverse} Cheerful
- {reverse} Full of pep
- {reverse} Vigorous
- Angry
- Peeved
- Annoyed
- Grouchy
- Resentful
- Bitter
- Furious
- Tense
- On edge
- Uneasy
- Restless
- Nervous
- Anxious
- Confused

- Unable to concentrate
- Bewildered
- Forgetful
- Uncertain
- Worn out
- Fatigued
- Exhausted
- Weary
- Bushed

Appendix B - Difficulties in Emotion Regulation Scale-16 (DERS-16)

Please indicate how often the following statements apply to you by writing the appropriate number from the scale above (1 – 5) in the box alongside each item.

1. I have difficulty making sense out of my feelings. [CLARITY*]

2. I am confused about how I feel. [CLARITY]

3. When I'm upset, I have difficulty getting work done. [GOALS]

4. When I'm upset, I become out of control. [IMPULSE]

5. When I'm upset, I believe that I will remain that way for a long time. [STRATEGIES]

6. When I'm upset, I believe that I'll end up feeling very depressed. [STRATEGIES]

7. When I'm upset, I have difficulty focusing on other things. [GOALS]

8. When I'm upset, I feel out of control. [IMPULSE]

9. When I'm upset, I feel ashamed with myself for feeling that way. [NONACCEPTANCE]

10. When I'm upset, I feel like I am weak. [NONACCEPTANCE]

11. When I'm upset, I have difficulty controlling my behaviors. [IMPULSE]

12. When I'm upset, I believe that there is nothing I can do to make myself feel better.

[STRATEGIES]

13. When I'm upset, I become irritated with myself for feeling that way.
[NONACCEPTANCE]

14. When I'm upset, I start to feel very bad about myself. [STRATEGIES]

15. When I'm upset, I have difficulty thinking about anything else. [GOALS]

16. When I'm upset, my emotions feel overwhelming. [STRATEGIES]