DOCTORAL THESIS

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UNIVERSIDAD CATÓLICA
DE MURCIA

INTERNATIONAL DOCTORAL SCHOOL

Doctoral Programme in Sports Science

THE ACUTE EFFECTS OF TRANSCRANIAL DIRECT CURRENT STIMULATION ON THE PERFORMANCE OF VELOCITY-BASED SQUAT TRAINING

Author:
Tai-Chih Chen

Supervisors:
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THESIS SUPERVISORS' AUTHORISATION
FOR THESIS SUBMISSION

Prof. Salvador Romero-Arenas and Prof. José Manuel García de Frutos, as Supervisors[1] of the Doctoral Thesis "*The acute effects of transcranial direct stimulation on the performance of velocity-based squat training*" by Mr. Tai-Chih Chen in the Doctorate Programme Sports Science, **authorise(s) its submission**, given that it meets the required conditions for its defence.

Which I hereby sign in compliance with Spanish Royal Decree 99/2011, of 28 January, in Murcia, on 30th September 2024.

Firmado por ROMERO ARENAS SALVADOR - ***2702** el día 18/12/2024 con un certificado emitido por AC FNMT Usuarios

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ABSTRACT

Transcranial direct current stimulation (tDCS) has emerged as a promising neuromodulatory technique for enhancing sports performance, particularly by mitigating velocity loss (VL) during overloaded exercises. This effect is attributed to tDCS-induced modulation of cortical activity, potentially influencing motor output and fatigue resistance. However, uncertainty remains regarding the optimal cortical target for stimulation, as evidence comparing different brain regions is limited and inconclusive. Thus, the primary objective of this thesis was to investigate the acute effects of anodal tDCS applied to two distinct cortical areas—the dorsolateral prefrontal cortex (DLPFC) and the primary motor cortex (M1)—on performance metrics during velocity-based squat training. We specifically examined the number of repetitions completed, movement velocity, and perception of effort, and also performed a cluster analysis to identify potential "responders" and "non-responders." Fifteen healthy men (21.8 ± 2.6 years) participated in a randomized, double-blind, crossover design. Each completed three experimental sessions receiving 20 minutes of anodal tDCS (2.0 mA) over bilateral M1, over DLPFC, or SHAM (control) stimulation. Immediately after stimulation, participants performed five sets of back squats until a 15% VL threshold was reached, with standardized 90-second rests. Performance was quantified by total repetitions, mean propulsive velocity (assessed via a linear encoder), and ratings of perceived exertion (RPE) using the OMNI-RES scale. The cluster analysis identified that 13 of the 15 participants responded positively to tDCS. In the entire sample, only the DLPFC condition resulted in a modest but significant increase in total repetitions compared to SHAM ($p = 0.035$). Among responders, both DLPFC and M1 stimulation elicited substantially more repetitions than SHAM ($p < 0.01$), without significant differences between these two active conditions. Notably, these improvements in training volume were not accompanied by changes in mean velocity or RPE, suggesting that tDCS may enhance endurance capacity without altering perceived exertion or explosive performance. These findings indicate that anodal tDCS over DLPFC or M1 can improve total training volume during squat exercise, particularly in individuals who are responsive to neuromodulation. The results hold practical implications for

strength and conditioning professionals, suggesting that tDCS could be integrated into high-intensity training protocols to increase muscular endurance without increasing subjective effort. Future research should explore the underlying neural mechanisms, investigate longer-term adaptations, and identify markers that predict responsiveness to tDCS in diverse athletic populations.

Key words: velocity loss, number of repetitions, velocity-based training, rating of perceived effort.

RESUMEN

La estimulación transcraneal por corriente continua (tDCS) se ha revelado como una prometedora técnica neuromoduladora para mejorar el rendimiento deportivo, en particular mitigando la pérdida de velocidad (VL) durante ejercicios con sobrecarga. Este efecto se atribuye a la modulación de la actividad cortical inducida por la tDCS, que puede influir en el rendimiento motor y la resistencia a la fatiga. Sin embargo, sigue habiendo incertidumbre en cuanto al objetivo cortical óptimo para la estimulación, ya que las pruebas que comparan diferentes regiones cerebrales son limitadas y no concluyentes. Por lo tanto, el objetivo principal de esta tesis fue investigar los efectos agudos de la tDCS anodal aplicada a dos áreas corticales distintas - la corteza prefrontal dorsolateral (DLPFC) y la corteza motora primaria (M1) - sobre las métricas de rendimiento durante el entrenamiento en cuclillas basado en la velocidad. Examinamos específicamente el número de repeticiones completadas, la velocidad de movimiento y la percepción del esfuerzo, y también realizamos un análisis de conglomerados para identificar posibles «respondedores» y «no respondedores». Quince hombres sanos (21,8 ± 2,6 años) participaron en un diseño aleatorizado, doble ciego y cruzado. Cada uno completó tres sesiones experimentales recibiendo 20 minutos de tDCS anodal (2,0 mA) sobre M1 bilateral, sobre DLPFC, o estimulación SHAM (control). Inmediatamente después de la estimulación, los participantes realizaron cinco series de sentadillas de espalda hasta alcanzar un umbral de VL del 15%, con descansos estandarizados de 90 segundos. El rendimiento se cuantificó mediante el total de repeticiones, la velocidad de propulsión media (evaluada mediante un codificador lineal) y las calificaciones del esfuerzo percibido (RPE) utilizando la escala OMNI-RES. El análisis de conglomerados identificó que 13 de los 15 participantes respondieron positivamente a la tDCS. En toda la muestra, sólo la condición DLPFC dio lugar a un aumento modesto pero significativo de las repeticiones totales en comparación con SHAM ($p = 0,035$). Entre los respondedores, tanto la estimulación DLPFC como la M1 provocaron un número sustancialmente mayor de repeticiones que el SHAM ($p < 0,01$), sin diferencias significativas entre estas dos condiciones activas. En particular, estas mejoras en el volumen de entrenamiento no se acompañaron de cambios en la velocidad media o RPE, lo que sugiere que tDCS puede mejorar la capacidad de resistencia sin alterar el esfuerzo percibido o el rendimiento

explosivo. Estos resultados indican que la tDCS anodal sobre DLPFC o M1 puede mejorar el volumen total de entrenamiento durante el ejercicio de sentadilla, particularmente en individuos que responden a la neuromodulación. Los resultados tienen implicaciones prácticas para los profesionales de la fuerza y el acondicionamiento, lo que sugiere que la tDCS podría integrarse en protocolos de entrenamiento de alta intensidad para aumentar la resistencia muscular sin aumentar el esfuerzo subjetivo. La investigación futura debe explorar los mecanismos neuronales subyacentes, investigar las adaptaciones a largo plazo, e identificar marcadores que predicen la capacidad de respuesta a tDCS en diversas poblaciones atléticas.

Palabras clave: pérdida de velocidad, número de repeticiones, entrenamiento basado en la velocidad, calificación del esfuerzo percibido

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Nobody has been more important to me in the pursuit of this study than my parents. Thanks to their spiritual support, I was able to overcome each hardship that I encountered during the process of this study. This work would not have been completed without them.

"Originality consists in returning to the origin, so original is that it returns to the simplicity of the first solutions"

Antonio Gaudi (1852-1926).

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ACRONYMS AND ABBREVIATIONS

The abbreviations of the units from the International System Units and the abbreviations universally used in statistics are not included in the following list as there are internationally accepted standards for their use.

1RM	One-repetition maximum
ADP	Adenosine Diphosphate
ATP	Adenosine Triphosphatase
ATPase	Myosin Adenosine Triphosphatase
a-tDCS	Anodal tDCS
c-tDCS	Cathodal tDCS
CMJ	Countermovement Jump
DLPFC	Dorsolateral Prefrontal Cortex
FI	Fatigue Index
M1	Primary Motor Cortex
RPE	Perceived Exertion
Reps	Repetitions
tDCS	Transcranial direct current stimulation
T	Torque
VL	Velocity Loss
Velocity-based training	VBT
W	Work

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I – INTRODUCTION

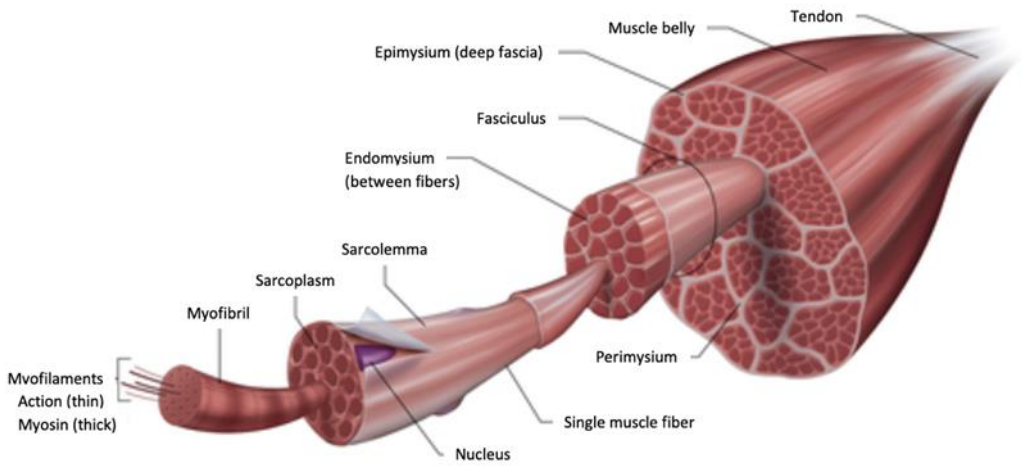
I - INTRODUCTION

1.1. STRUCTURE AND FUNCTION OF THE MUSCULOSKELETAL SYSTEM

1.1.1. Structure of skeletal muscle

Physical exercise and sport performance require effective body movement, which is a consequence of force generated in muscles that move our numerous body parts by acting through lever systems of the skeleton. Every skeletal muscle consists of muscle tissue, connective tissue, nerves, and blood vessels. More than 430 of our skeletal muscles are covered by fibrous tissues or epimysium (Figure 1), which is connected to the tendons at the ends of muscles. The tendon is attached to a specialized connective tissue, bone periosteum, which cover all bones. Therefore, when the muscle contraction pulls on the tendon, which in turn pulls on the bone (1).

Figure 1. A bundle of muscle fibers that constitute a muscle.



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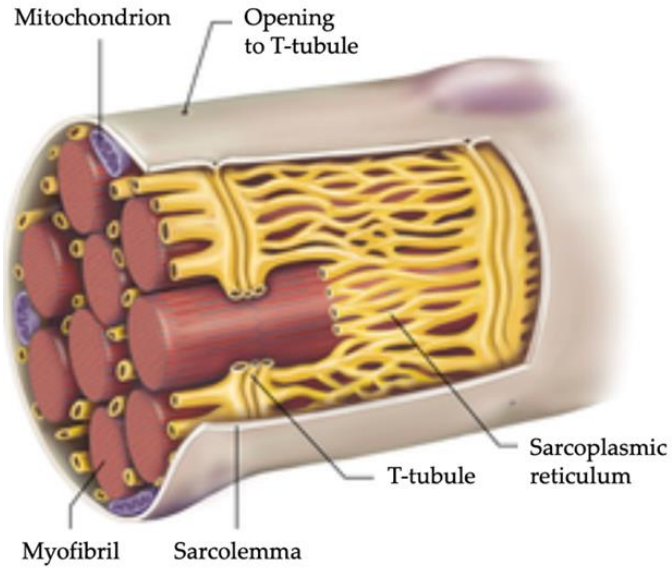
The skeletal muscles are made of a number of bundles of muscles fibers which are long and cylindrical cells (50-100 μm in diameter) (Figure 1). The cytoplasm of a muscle fiber, known as sarcoplasm, contains contractile components consisting of protein filaments, other proteins, stored glycogen and fat particles, enzymes, and specialized organelles such as mitochondria and the sarcoplasmic reticulum (Figure 2). However, the sarcoplasm consists of most of the myofibrils, which are responsible for the contraction of the muscle cell (1).

Figure 2. Internal structure of muscle fiber.



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Myofibrils are about 1 μm in diameter, 1/100 the diameter of a hair, and mainly consists of two types of myofilaments, myosin, and action (Figure 3). The myosin filaments are thicker (about 16nm in diameter, approximately 1/10,000 the diameter of a hair) and contain up to 200 myosin molecules. It has a globular head (a hinge point) and a fibrous tail. The globular head sticks out from the myosin filament at a regular interval and interact with actin, forming a cross bridge. On the other hand, the actin filaments are thin filaments, about 6 nm in diameter,

containing two strands arranged in a double helix. Myosin and actin filaments are arranged longitudinally in the smallest contractile unit of skeletal muscle, known as sarcomere, which is about 2.5 μm in length in a relaxed fiber (approximately 4,500 per centimeter of muscle length) and is repeated along the entire length of the muscle fiber (1,2).

Adjacent myosin filaments are fixed at the M-bridge in the center of sarcomere (the center of the H-zone) (Figure 3). Actin filaments are aligned at both ends of the sarcomere and are stabilized at the Z-line, which is repeated along the entire myofibril. Every myosin filament is surrounded by six actin filaments, and every actin filament is surrounded by three myosin filaments. This sarcomere arrangement appears as a striated, alternating dark and light pattern under amplification. The dark A-band is formed by the alignment of the myosin filaments, while the light I-band is formed by two adjacent sarcomeres that consists only of actin filaments (3). In the middle of the I-band, the Z-line appears as a thin, dark line running longitudinally through the I-band. In the center of the sarcomere, there appears the H-zone where only myosin filaments are seen. When muscle contracts, the H-zone shortens as the myosin pulls the actin toward the center of the sarcomere. Simultaneously, the I-band also shortens as the Z-lines are pulled toward the center of the sarcomere (1).

An intricate system of tubules, known as the sarcoplasmic reticulum (Figure 2), runs parallel to and surrounds each myofibril, terminating as vesicles in the vicinity of the Z-lines. In the vesicles, calcium ions are stored and regulated to control muscular contraction. Perpendicular to the sarcoplasmic reticulum are the T-tubules, or transverse tubules, which run along the Z-line between two vesicles. As T-tubules run between myofibrils and are connected to the sarcolemma at the surface of the myofibril, they can transmit the action potential (an electrical nerve impulse) from the surface to all depths of the muscle fiber; this process releases calcium throughout the muscle, producing tension development in muscle.

1.1.2. Muscle contraction mechanism

As mentioned above, muscle contraction could result from the shortening of muscle fiber, where myosin pulls the actin filament at each end of the sarcomere inward, concurrently pulling the Z-line toward the center of the sarcomere and causing both the H-zone and I-band to shrink (Figure 4). However, as the displacement of actin filaments in each flexion of the myosin crossbridge is limited, the rapid and repeated flexions of the crossbridge throughout the entire muscle are needed to induce significant movement. Furthermore, as the force are generated by the crossbridge of the myofibril filaments, the level of force that could be generated are influenced by the length of sarcomeres. For example, at extreme lengthened length, the myosin binding site on the actin filaments move out of the range of the globular heads of the myosin filaments (Figure 4a); therefore, fewer myosin heads can bind with the actin filaments, and thereby lower force is generated from myofibril contraction (4). However, at their resting length, the peak tension is produced due to optimal alignment between actin and myosin filament (Figure 4b). Nevertheless, at extremely shortened lengths, the force potential is low as the overlap of actin filaments leads to no further potential for myofibrils to contract, resulting in little to no force generation (Figure 4c) (1,4).

Under resting conditions, little calcium is present in the myofibril (especially in the sarcoplasmic reticulum), resulting in very few of the myosin crossbridge bound to actin. Even though the resting condition has the actin binding site covered, myosin and actin still interact in a weak bond; however, the bond becomes stronger when the actin binding sites is exposed after the release of the stored calcium. When the sarcoplasmic reticulum is stimulated to release calcium ions, the calcium binds with troponin, a protein on the actin filament (Figure 3, 5a) that has a high affinity for calcium ions. This binding causes displacement of another protein molecule, tropomyosin, which runs along the actin filament in the groove of double helix and blocks the myosin binding site during resting condition (Figure 3, 5b). This binding-induced displacement exposes the myosin binding site on the actin filament (Figure 5b).

Figure 3. The myosin and actin protein filaments of myofibril.

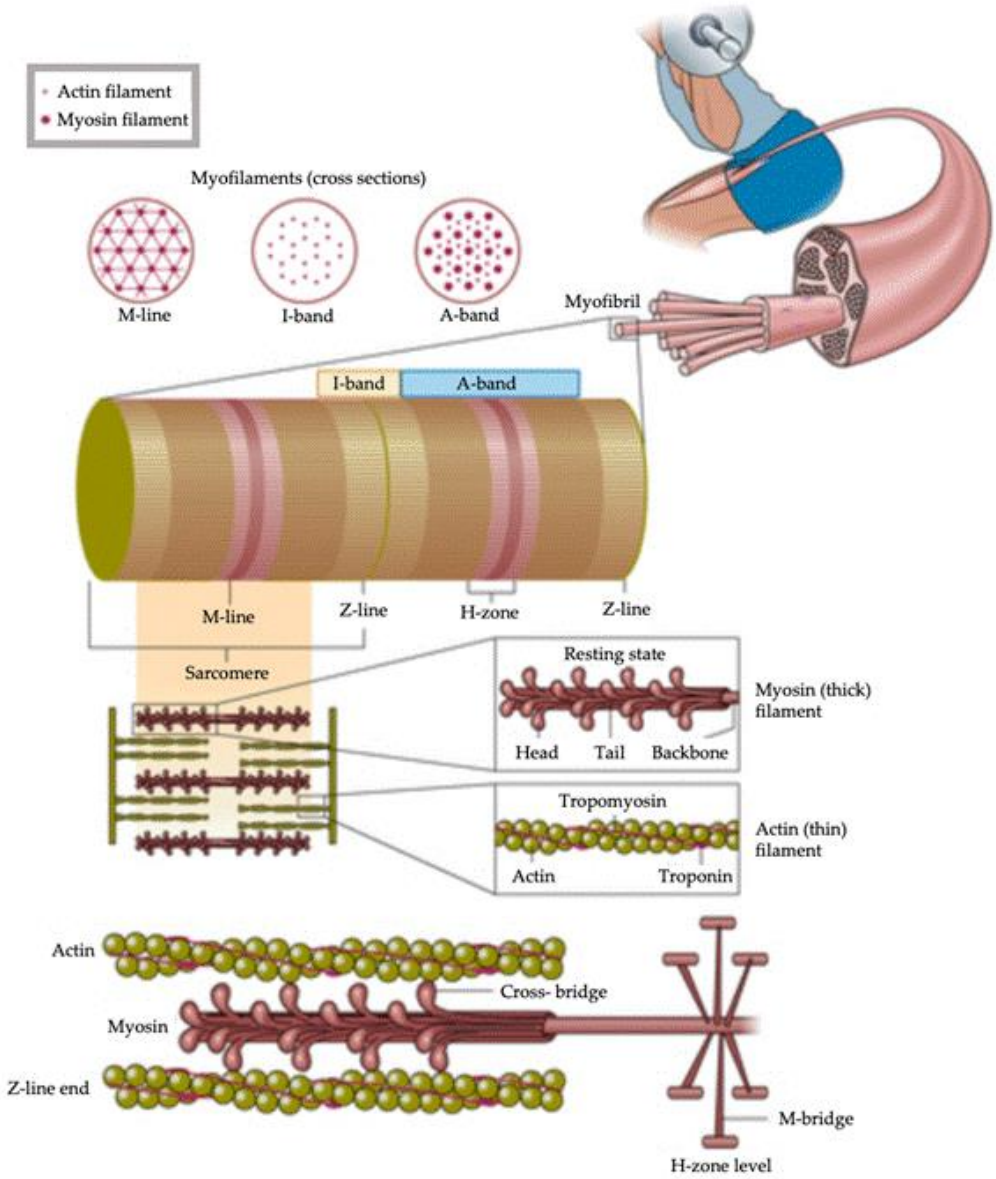

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Figure 4. Contraction of myofibril.

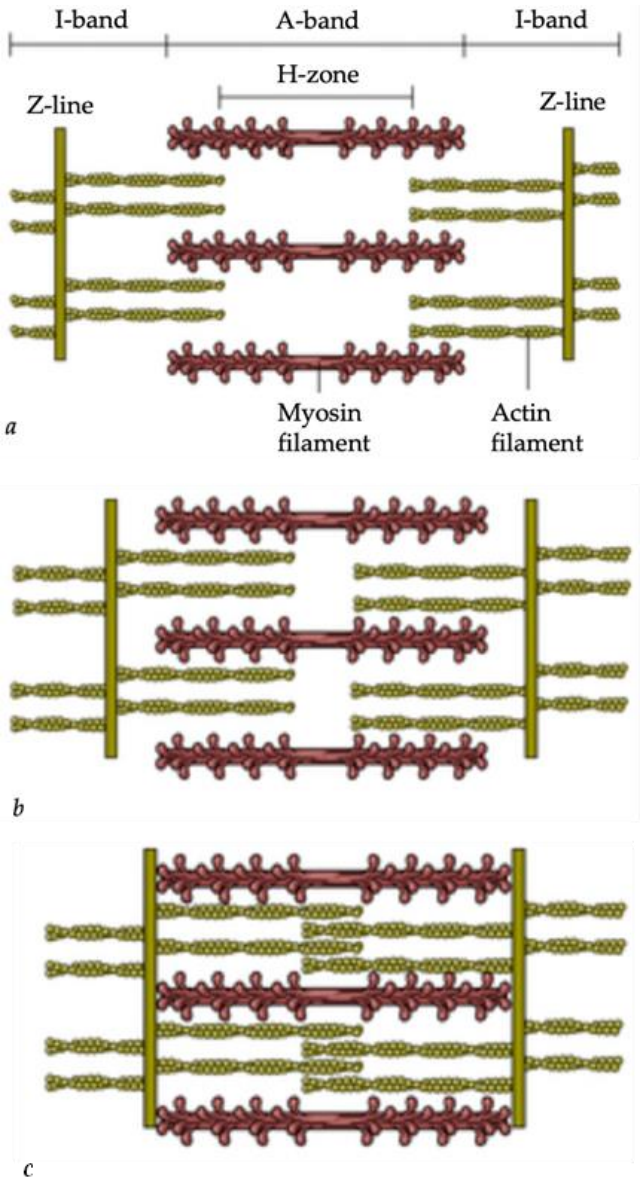

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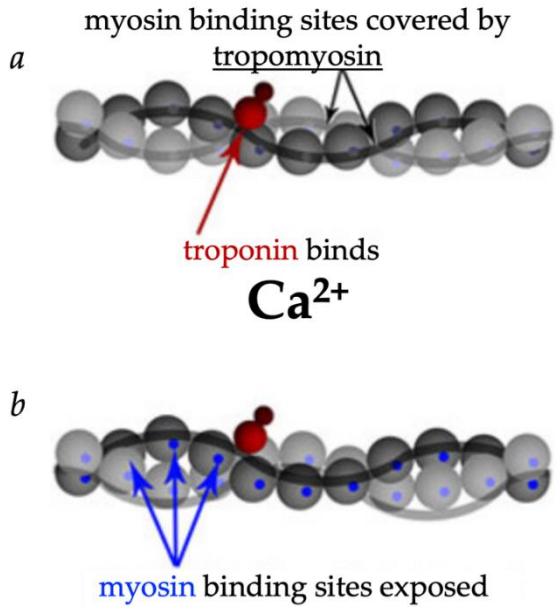
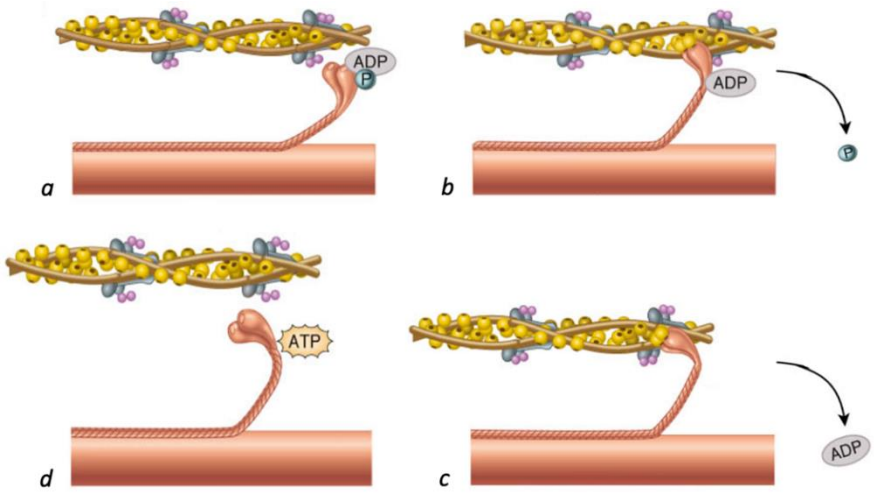
Figure 5. Myosin binding site exposure.



Note: Adapted from "Ca2+-induced tropomyosin movement in Limulus thin filaments revealed by three-dimensional reconstruction," by W. Lehman; R. Craig; P. Vibert, Nature 368, 65-67 (1994), All rights reserved.

At this stage, the head of each myosin unit, bound to an ADP (adenosine diphosphate) and a phosphate molecule remaining from the previous muscular contraction (Figure 6a), releases the phosphate molecule (Figure 6b). This release causes the myosin head to attach to the myosin binding site on the actin filament (Figure 6b) and pull the actin filament toward the center of the sarcomere while releasing the ADP molecule (Figure 6c). This pulling motion is halted when an adenosine triphosphatase (ATP) molecule binds to the myosin head, detaching the myosin crossbridge from the actin and returning the myosin head to its starting positions (Figure 6d). Simultaneously, the ATP molecule bound to myosin is decomposed by myosin adenosine triphosphatase (ATPase) into ADP and phosphate, storing the energy released by this reaction in the myosin head for the next cycle of movement (Figure 6a). This cycle continues as long as ATP, ATPase, and calcium are available (1,5).

Figure 6. Sliding filament theory of muscle contraction.

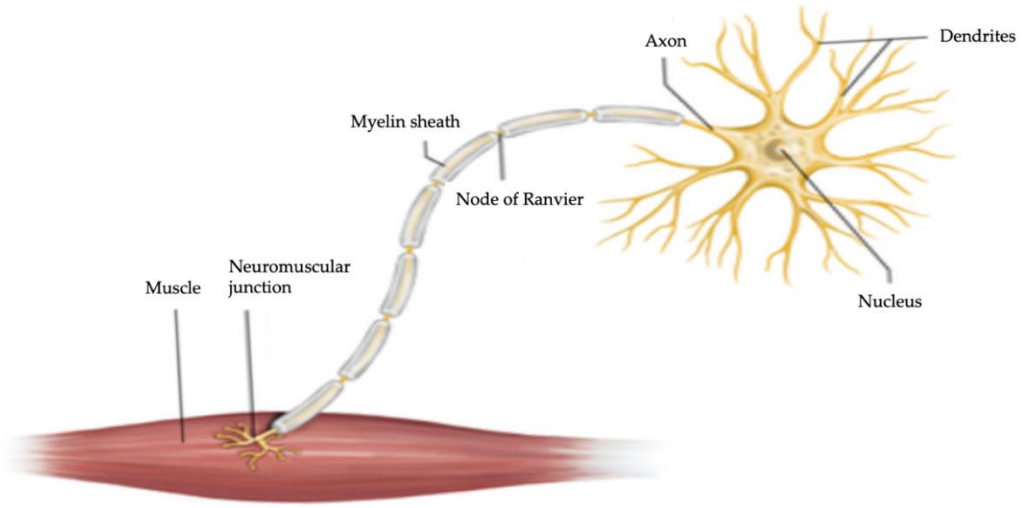


Note: Adapted from "Sliding Filament Theory of Muscle Contraction," (https://www.onlinebiologynotes.com/sliding-filament-model-of-muscle-contraction/). Copyright © 2024 | WordPress Theme by MH Themes

1.1.3. Neuromuscular system

To regulate the skeletal muscles and activate the muscle contraction mechanisms, the cerebral cortex activates the skeletal muscle cells or fibers through the motor neurons of the peripheral nervous system (1). Numerous muscle fibers are innervated by a motor neuron that has a bunch of terminal branches at the end of its axon, where the muscle fibers are innervated through neuromuscular junction (Figure 7). A motor neuron and its innervating muscle fibers are considered as a motor unit. All the muscle fibers of a motor unit contract at the same time when they are stimulated by the motor unit that transmits impulse in the form of electrochemical signals from the spinal to them. The number of fibers per each motor unit determines how that muscle function. For example, the muscle group that need greater precision, such as eye muscles, may have motor units with fewer muscle fibers. On the other hand, the muscle group that needs to generate bigger force with lower precision may have more fibers innervated by every motor unit (1).

Figure 7. A motor unit with its innervating muscle fibers.



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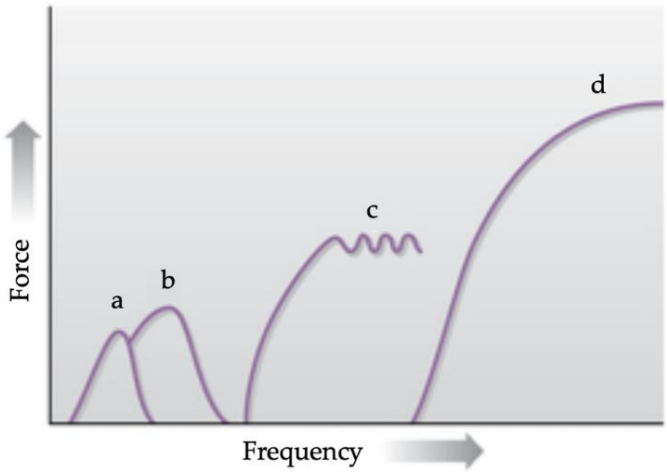
Regardless of muscle group type, the motor neuron must fire an action potential and send it toward the innervated muscle. Upon the arrival of an action potential at the nerve terminal, the neurotransmitter, acetylcholine, is released and diffuses across the neuromuscular junction, inducing excitation of the sarcolemma (Figure 8). Subsequently, an action potential is generated within the sarcolemma when the released amount of acetylcholine reaches the threshold, causing the contraction of the entire muscle fiber in the motor unit. This event is known as the all-or-none principle, as it has not been proven that only some muscle fibers contract when a motor neuron stimulates; furthermore, a stronger action potential does not lead to a greater contraction (1).

When an action potential travels through a motor neuron, it leads to a short period of activation of the muscle fibers within the motor unit. This activates the sarcolemma and thereby induces the release of calcium within the muscle fibers, causing a brief contraction referred to as a twitch. However, regardless of the amount of the released calcium that might reach optimal activation of actin and myosin to obtain maximal force of the fibers, calcium is removed before force

reaches its maximum and muscle relaxes (Figure 8a). However, when a second twitch is induced before the fibers of first twitch completely relax, the force of these two twitches is added up, resulting in greater force than that produced by a single twitch (Figure 8b). Furthermore, when more twitches are produced with shorter intervals, greater summation of crossbridge binding and force is triggered, resulting in a merging of twitches, known as tetanus, which reaches the maximal amount of force that the motor unit can develop (Figure 8c and d) (1).

Figure 8. Twitch and its summation in a motor unit at different frequency.



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The twitch of a muscle fiber varies in velocity among the different types of muscle fibers due to distinct morphological and physiological characteristics. Consequently, twitch time is a well-known criterion for classifying muscle fiber types, with terms such as slow-twitch and fast twitch fiber. Moreover, since a motor unit is composed of the same type of muscle fibers, it also serves as a classification criterion. Specifically, a fast-twitch motor unit develops and relaxes force rapidly, whereas a slow-twitch motor unit develops and relaxes force slowly and has a longer twitch time. Furthermore, as the content of myosin ATPase and myosin heavy chain varies among different muscle fiber types, detecting these amounts is

also a technique for identifying them. Commonly identified fibers through histochemical staining for myosin ATPase content are Type I (slow-twitch), Type IIa (fast-twitch), and Type IIx (fast-twitch) (1).

The difference in mechanical characteristics of Type I and II fibers lies in their ability to demand and supply energy for contraction and their resistance to fatigue. Type I fibers are generally efficient and fatigue-resistant, with a high capacity for aerobic energy supply. However, they have less potential for explosive force development, as characterized by low myosin ATPase and anaerobic power (6,7). On the other hand, Type II motor units are inefficient and fatigable with low aerobic power, rapid force development, high myosin ATPase activity, and high anaerobic power (6,7). Nevertheless, there is a difference between Type IIa and Type IIx fibers, particularly in their capacity for aerobic-oxidative energy supply. Type IIa have greater capacity for aerobic metabolism with more capillaries surrounding them, thus showing greater resistance to fatigue (1,8–10). Accordingly, these muscle types could be distributed to the certain muscle group based on their role. For example, postural muscles, such as the soleus, have a high composition of Type I fibers, whereas large locomotor muscle, such as the quadriceps group, have a mixture of Type I and Type II fibers to allow both low and high-power output exercise (e.g., jogging and sprinting, respectively) (1).

For some sports events, such as sprint cycling, sustaining maximal effort for a certain period of time (e.g., ~15-60s) is highly required (11). This type of exercise necessitates the recruitment of fatigable fast-twitch fibers, and places substantial reliance on anaerobic energy production (12–14), leading to a faster accumulation of metabolic by-products, such as inorganic phosphate (Pi), hydrogen ion (H^+), and ADP (15–18). The accumulation of these molecules can interfere (e.g., directly or via a reduction of cytosolic pH) with glycolytic enzyme (e.g., phosphofructokinase) activity, myofilament sensitivity to $Ca2^+$, cross bridge kinetics (e.g., actomyosin binding number, force, and cycling rate), sarcoplasmic reticulum $Ca2^+$ release, and reuptake kinetics, resulting in impaired force production and shortening velocity (11). Furthermore, metabolite accumulation stimulates group III and IV chemo- and nociceptive muscle afferents (19), inducing a downregulation of central motor drive with sensations of discomfort or pain, inhibiting motor neuron recruitment and firing, thereby resulting in a decline in motor output (20–22).

To extend the duration of high intensity exercise, it might be necessary to increase maximal power to mitigate the potential increase in substrate depletion and metabolite accumulation resulting from enhanced metabolic and mechanical power output (23,24). This could be achieved through high-intensity exercise, such as sprint interval training, which may enhance the buffering of fatiguing metabolites by increasing glycolytic flux (e.g., higher lactate production) for a given exercise bout, without causing changes to local pH values (11). This attenuation of metabolite-induced downregulation of contractile performance could help sustain high-intensity efforts (25,26). However, an improvement in high intensity sustaining capacity could also be achieved by increasing cortical activity through neuromodulators, such as transcranial direct current stimulation (tDCS), even when no voluntary contraction is involved in the intervention (27,28). Therefore, enhanced cortical activity after non-invasive brain stimulation may counteract the negative impact of efferent signals from metabolite accumulation on motor output, thereby prolonging the duration of high-intensity exercise.

1.2. NEUROSCIENCE APPLIED TO SPORT AND NON-INVASIVE BRAIN STIMULATION

For the treatment of psychopathologies, various forms of electrical stimulation have been developed to modify electrical brain processes and enhance human brain function by delivering electrical current over the scalp. At the same time, relevant investigation has also been widely conducted to improve the understanding of brain physiology. At the beginning, the electric currents generated from torpedo fish and electric catfish were proposed as a treatment for headache and epilepsy (29,30). However, to conduct controlled transcranial direct stimulation, an electrical battery was subsequently implemented on clinical population (30), and concurrently investigated to evaluate its effect in treatment (e.g., symptoms of melancholia) (31). Accordingly, more controlled, safe, and effective stimulation device have been developed, with the promising evidence showing their interaction with the central nervous system (32).

Electrical brain stimulation can be classified into an invasive and non-invasive approach. The most representative technique among invasive brain stimulation methods is deep brain stimulation, which involves implanting electrodes directly in the target area, thereby enabling subsequent control of the on-

off switching of electrical current delivery. This approach has demonstrated several benefits in the treatment of Parkinson's disease (33), Tourette syndrome (34), depression (35), and pain relief (36). However, even if this method could reach subcortical and subthalamic brain areas, its application may cause the spread of stimulation to adjacent structures, inducing side-effects, such as visual hallucinations (37), compulsive behaviors, postoperative depression (38), or infection (39,40). Hence, brain stimulation has been subsequently developed in a non-invasive form, which can change the cortical excitability without the need of surgery (41).

Multiple types of non-invasive brain stimulation have been developed, such as repetitive transcranial magnetic stimulation, galvanic vestibular stimulation, and transcranial alternating current stimulation. These types of stimulation have showed positive effect on cognitive ability. For example, galvanic vestibular stimulation was found to increase tactile sensitivity in healthy participants, measured in the somatosensory signal detection task. Furthermore, transcranial alternating current stimulation was shown to facilitate attention, perception, memory processes, and working memory storage capacity (42). Nevertheless, among these types of stimulation, transcranial direct current stimulation is considered as relatively low cost, user-friendly, and almost free of side effects. This stimulation type represents one of the most powerful approaches for priming the cortical excitability and modify performance of a subsequent task or demand (43).

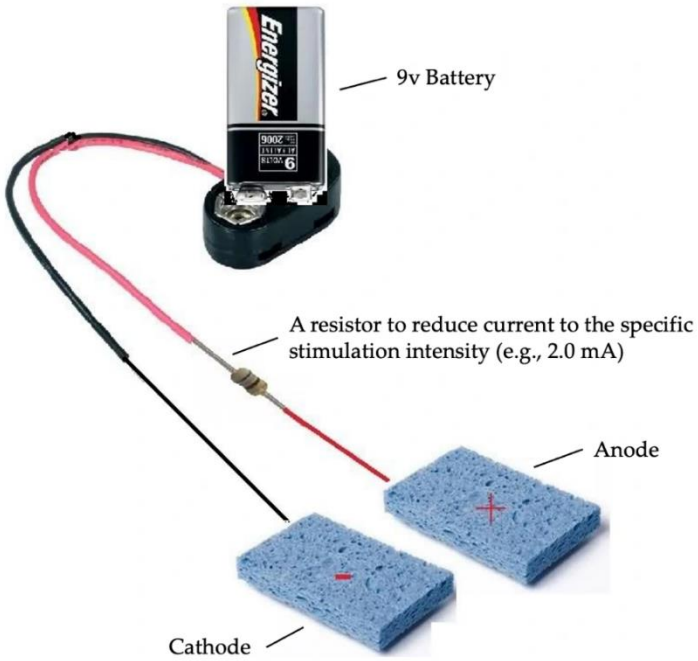
1.2.1. Transcranial direct current stimulation

1.2.1.1. Fundamentals and mechanisms of action

The tDCS is known as a non-invasive brain stimulation which delivers a constant and weak direct current typically by using a 9V battery (Figure 9) to the brain through two or more non-invasive scalp electrodes (anodal and cathodal electrodes) (Figure 9) placed over the scalp (Figure 10) (44,45). Either one of the electrodes (anode or cathode) can be placed on the targeted region as active electrode to module the neural excitability, while the other one is placed on an area less involved in the task as reference electrode, forming a current circuit to induce intracerebral current flow (46). The current flow then either increases or decreases

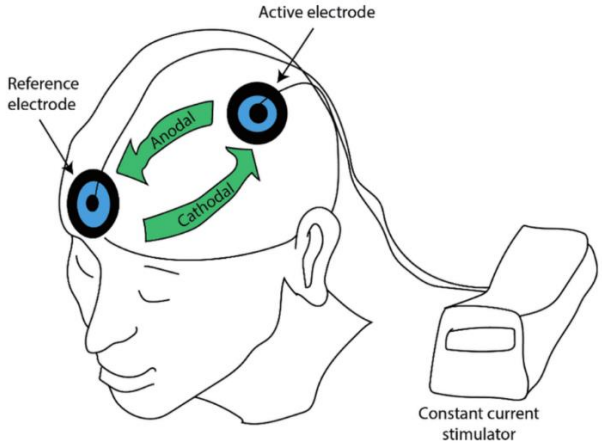
the neuronal excitability in the area being stimulated through the active electrode, depending on the type of active electrode used (46). When an anodal electrode is employed and placed on the targeted area as the active electrode, known as anodal tDCS (a-tDCS), the current causes a depolarization of the resting membrane potential, increasing neuronal excitability and allowing for more spontaneous cell firing (Figure 11a). On the other hand, when the targeted region is stimulated by the cathodal electrode as the active electrode, known as cathodal tDCS (c-tDCS), the current causes a hyperpolarization of the resting membrane potential, decreasing neuron excitability due to the decreased spontaneous cell firing (45,46) (Figure 11b).

Figure 9. Transcranial direct current stimulator (driven by 9VDC)



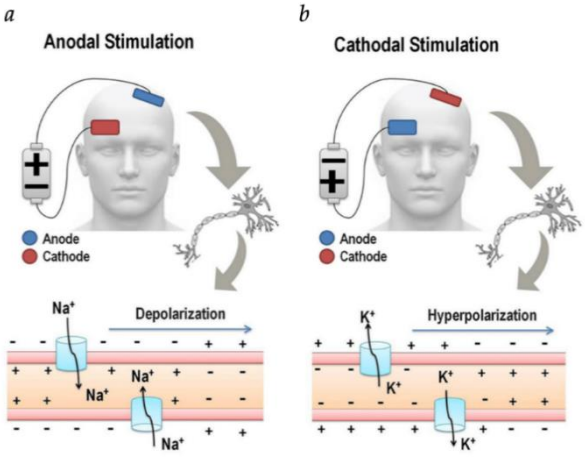
Note: Adapted from "tDCS – Thoughts on Safety for the Amateur," (*https://speakwisdom.wordpress.com/2012/08/27/tdcs-thoughts-on-safety-for-the-amateur/*).

Figure 10. Transcranial direct current stimulation set up & its induced current flow in motor cortex.



Note: Adapted from "Noninvasive Modalities Used in Spinal Cord Injury Rehabilitation," (https://www.researchgate.net/figure/Transcranial-direct-current-stimulation-delivers-continuous-low-current-stimulation-by_fig2_330753963). © 2008-2024 ResearchGate GmbH. All rights reserved.

Figure 11. Impact of anodal & cathodal transcranial direct current stimulation on cortical activity.



Note: Adapted from "Transcranial direct current stimulation and neuroplasticity," (https://www.researchgate.net/figure/Effects-of-anodal-and-cathodal-on-membrane-polarization-Figure-in-the-left-represents_fig1_320233137). © 2008-2024 ResearchGate GmbH. All rights reserved.

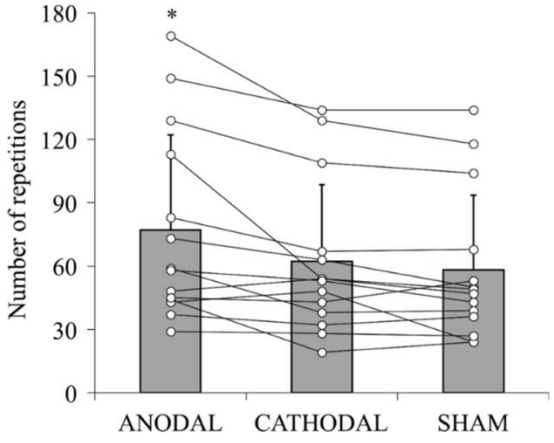
1.2.1.2. Clinical and sport applications

Initially, the tDCS was developed as a treatment for various neurological (47,48) and psychiatric disorders/conditions (49,50), as well as for learning (51) and memory (52). In addition to the clinical usage, tDCS was found to show various behavioral effects on gross motor performance (53), particularly concerning muscle strength and the ability to sustain maximal or submaximal efforts over a certain period (54–58). For example, the individual receiving a-tDCS was found to perform more repetitions during high-intensity resistance training (e.g., explosive bench press) before reaching failure (Figure 12) (27). Furthermore, they could perform the training with a lower rate of velocity loss (VL) (Figure 13) and perceived effort (27). A similar finding was also observed in other exercises, such as sprinting (28), showing that the application of a-tDCS could enable participants to complete multiple sets of a certain distance with a lower rate of speed decline (Figure 14), as measured by the fatigue index (FI) formula below (28).

Equation 1.

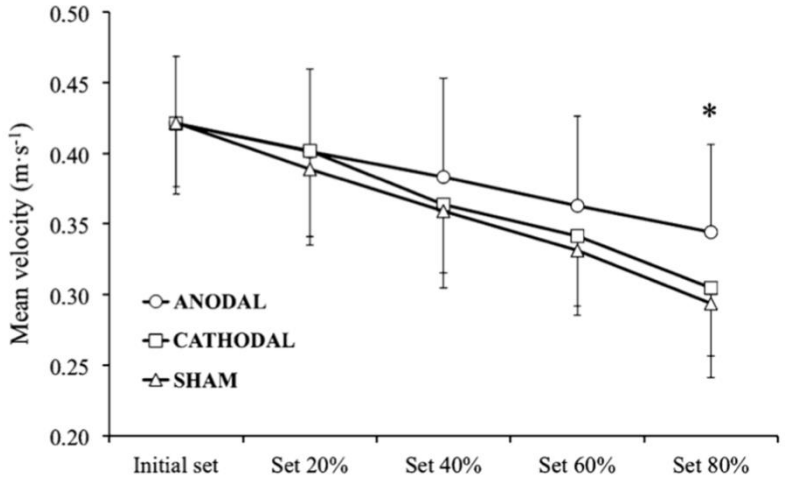
$$FI = [100 \times (total\ sprint\ time / ideal\ sprint\ time)] - 100$$

Figure 12. Number of repetitions in the explosive bench press after anodal, cathodal, and sham transcranial direct current stimulation.



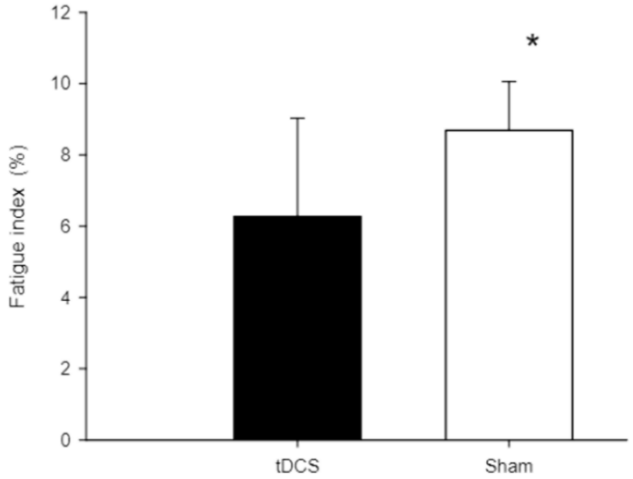
Note: Adapted from "Anodal transcranial direct current stimulation enhances strength training volume but not the force–velocity profile," (https://pubmed.ncbi.nlm.nih.gov/32533243/). © Springer-Verlag GmbH Germany, part of Springer Nature 2020.

Figure 13. Mean the velocity of the explosive bench press after anodal, cathodal, and sham transcranial direct current stimulation.



Note: Adapted from "Anodal transcranial direct current stimulation enhances strength training volume but not the force–velocity profile," (https://pubmed.ncbi.nlm.nih.gov/32533243/). *© Springer-Verlag GmbH Germany, part of Springer Nature 2020.*

Figure 14. Rate of sprinting fatigue index after anodal and sham transcranial direct current stimulation.

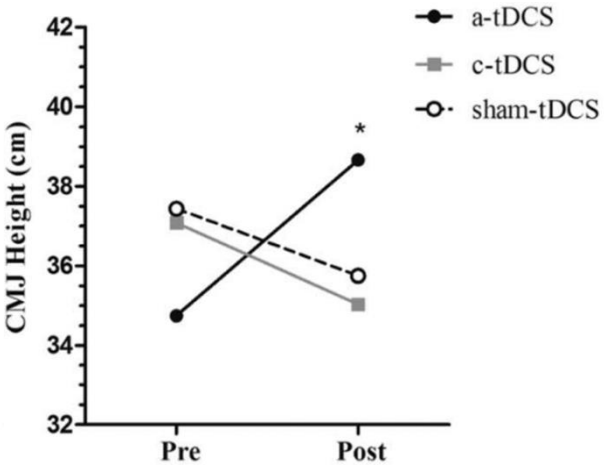


Note: Adapted from "Transcranial Direct Current Stimulation Decrease the Decline of Speed during Repeated Sprinting in Basketball Athletes," (https://pubmed.ncbi.nlm.nih.gov/34209833/). *© 2021 by the authors. Licensee MDPI, Basel, Switzerland.*

Accordingly, an enhancement in motor cortex activity after a-tDCS could counteract the negative afferent impact of metabolite accumulation, thereby allowing individuals to maintain high-power output for more repetitions.

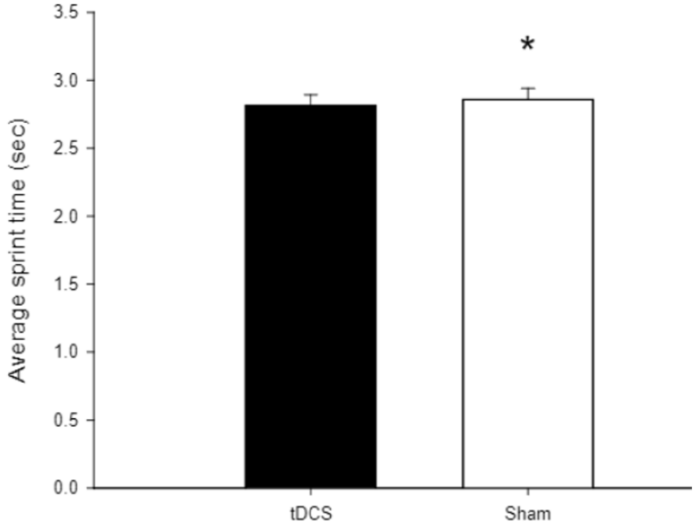
In addition to the increasing effect of a-tDCS on fatigue resistance, other aspects of fitness could also benefit from tDCS application, such as explosive performance. Particularly, the participants undergoing a-tDCS intervention were observed to perform the countermovement jump (CMJ) at a higher height (Figure 15) (59,60) or completed the multiple sets of sprinting in a shorter time (Figure 16) (28). Taken together, the increased cortical activity after a-tDCS may facilitate motor cortex neurons in forming neural connections and altering firing frequency (28). This, in turn, increases motor drive to muscles, promoting the recruitment of motor units and thus improving anaerobic fitness, such as fatigue resistance and explosive performance during high-intensity exercise (28). However, even if the improvements in both fatigue tolerance and power following a-tDCS were mainly facilitated through a similar mechanism (increased cortical activity), these alterations were not always correlated (27). For instance, the reduced rate in VL induced by a-tDCS, as mentioned above, was not found to be accompanied by an improvement in maximal power but rather by a reduction in the rating of perceived exertion (RPE) (27). Nevertheless, these alterations were detected when the tDCS was placed over dorsolateral prefrontal cortex (DLPFC) (27). However, when the tDCS targeted the primary motor cortex (M1), the improvement in fatigue resistance (less rate of sprinting speed loss) coincided with the enhancement in explosive performance (the height of CMJ) (28). Nonetheless, these changes did not occur with perceptual alteration (28). Therefore, depending on the stimulated areas, alterations in fatigue resistance after a-tDCS might occur with perceptual or explosive changes during high-intensity exercise.

Figure 15. CMJ height after anodal, cathodal, sham transcranial direct current stimulation.



Note: Adapted from "Can transcranial Direct Current Stimulation improve muscle power in individual with advanced weight-training experience?" (https://pubmed.ncbi.nlm.nih.gov/28426515/). © 2017 National Strength and Conditioning Association.

Figure 16. Average sprinting time after anodal and sham transcranial direct current stimulation.



Note: Adapted from "Transcranial Direct Current Stimulation Decrease the Decline of Speed during Repeated Sprinting in Basketball Athletes," (https://pubmed.ncbi.nlm.nih.gov/34209833/). © 2021 by the authors. Licensee MDPI, Basel, Switzerland.

1.2.1.3. Effects of tDCS on motor control and muscle fatigue

Fatigue was considered as a decrease in physical and/or mental performance that resulted from changes in central, psychological, and/or peripheral factors (61). Performance fatigability, defined as "the magnitude or rate of change in a performance criterion relative to a reference value over a given time to task performance (61), has been examined across various types of exercise. In addition to the sprinting and bench press mentioned above, knee extension, abduction of the first dorsal interosseous muscle, cycling, and squatting were also employed to evaluate the effect of tDCS on muscle fatigue. Nevertheless, except for sprinting and bench press, the alteration in the amplitude of performance reduction after tDCS was not obvious in those exercises. For example, the work (W) and torque (T) fatigue index (equations 2 and 3, respectively) (62–66), slope (67), mean T across a certain repetition (68), and peak T during maximal repetitions of knee extension (69) did not change after tDCS application. For the abduction of the first dorsal interosseous muscle, no evident alteration in peak T was observed during 10 repetitions, 30s-isometric contraction task after a-tDCS (70). Regarding cycling, there was no difference in fatigue index (equation 4 and 5) during 30s all-out cycling after tDCS and SHAM (71,72). With respect to the squat, rather than no change, the fatigue magnitude, measured by VL levels, increased during 3 sets of 12 repetitions after tDCS (73). Accordingly, the effect of tDCS on fatigue resistance may vary across various types of exercise. However, contrasting results were still observed even when the same exercise (e.g., sprinting) was examined (74), contrasting the assumption of exercise type-based tDCS effect. Nevertheless, the difference in exercise protocols (e.g., distance, sets, rest) and stimulated regions (DLPFC and M1) among the studies (28,74) might have concealed the effect. Therefore, continuous investigation with distinct exercise type, protocols, and stimulated regions may be needed to explore the optimal tDCS application.

Equation 2.

$$FI_W = \frac{Average\ W_{first\ repetitions} - Average\ W_{last\ repetitions}}{Average\ W_{first\ repetitions}} \times 100$$

Equation 3.

$$FI_T = \frac{Average\ T_{first\ f\ repetitions} - Average\ T_{last\ repetitions}}{Average\ T_{first\ repetitions}} \times 100$$

Equation 4.

$$FI_{power} = \frac{[(Peak\ power - Less\ power\ in\ the\ last\ 20\ seconds) \times 100]}{Peak\ power}$$

Equation 5.

$$FI_{peak\ power} = \frac{(Peak\ power - Minimum\ power)}{Peak\ power \times 100}$$

1.3. ELECTRODE PLACEMENT IN TDCS

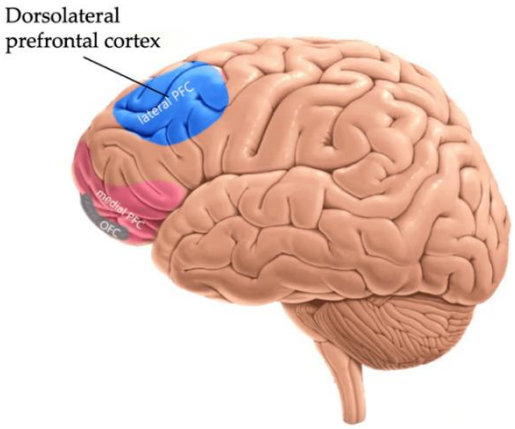
1.3.1. Dorsolateral Prefrontal Cortex

The dorsolateral prefrontal cortex (DLPFC) is a region in the prefrontal cortex, especially located on the lateral portion of the prefrontal cortex, which is the anterior part of the frontal lobes. Furthermore, it is roughly found in the middle of the forehead, extending back toward the top of the head (Figure 17) (75).

The DLPFC is composed of spatially selective neurons and a neural circuitry that encompasses the entire range of sub-functions necessary for an integrated response, such as sensory input, retention in short-term memory, and motor signaling (76). Its connection with multiple cortices, including the superior temporal cortex, posterior parietal cortex, anterior and posterior cingulate, premotor cortex, retrosplenial cortex, and neocerebellum, allow the DLPFC to regulate and be regulated by these regions (77). This region plays a key role in inhibitory control, essential for both behavioral self-regulation and exercise regulation, by integrating cognitive and peripheral information (78,79). This function is particularly important in inhibiting subjective fatigue (27,74), as high fatigue may require the prefrontal cortex to inhibit the anterior cingulate and

insula, which are activated in proportion to the degree of subjective fatigue (80), to maintain the exercise performance (74).

Figure 17. Location of dorsolateral prefrontal cortex.



Note: Adapted from "Hot and cold executive functions in the brain: A prefrontal- cingular network," (https://www.researchgate.net/figure/Lateral-view-of-the-prefrontal-cortex-PFC-regions-and-association-with-hot-and-cold_fig4_350102535) © 2008-2024 ResearchGate GmbH. All rights reserved.

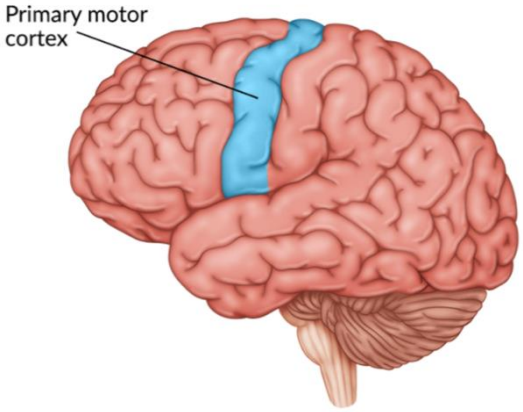
1.3.2. Primary motor cortex

The primary motor cortes (M1) is a brain region located in the dorsal portion of the frontal lobe, particularly on the anterior wall of the central sulcus. It extends anteriorly out of the sulcus and partly onto the precentral gyrus (Figure 18) (81). Anteriorly, it is bordered by a set of areas on the precentral gyrus that are considered part of the lateral premotor cortex, and posteriorly, it is bordered by the primary somatosensory cortex on the posterior wall of the central sulcus. Ventrally, it is bordered by the insular cortex in the lateral sulcus and extends dorsally to the top of the hemisphere, reaching the medial wall of the hemisphere (82).

The M1 is the primary region of motor system, working with other motor areas, including the premotor cortex, supplementary motor area, posterior parietal cortex, and several subcortical brain regions, to plan and execute voluntary movement (81). It integrates information from the sensory and premotor cortices, and subsequently transmits appropriate motor commands to the spinal cord and

basal ganglia. (83). This area contains the largest neuron in the central system, known as Betz cells, which, along with other cortical neurons, send long axons down the spinal cord to synapse onto the interneuron circuitry of the spinal cord and the alpha motor neurons connected to the muscle (81).

Figure 18. Location of primary motor cortex.



Note: Adapted from "Primary Motor Cortex Damage: What to Expect & How to Treat." (https://www.flintrehab.com/primary-motor-cortex-damage/). Copyright 2024 © All rights Reserved. Design by Elementor.

1.3.3. Comparison of Effect Based on Placements

Although both the DLPFC and M1 are regarded as important regions in physical performance, there are slight differences in the role of their contribution, as mentioned above. Collectively, the DLPFC modulates the motor drive based on cognitive and peripheral information (79). Hence, the importance of DLPFC in the ability to maintain physical activities has been highlighted through its influence on aspects such as motivation and decision-making (79,84). In contrast, the M1 integrates information from the sensory and premotor cortices to activate the motor units through the corticospinal tract (83,85). As such M1 may directly influence the muscle's ability to execute specific tasks (86,87). Accordingly, the increased activity in the DLPFC and M1 after a-tDCS might selectively benefit distinct performance variables, such as perceived efforts or explosive performance, respectively.

Due to the slight difference in their roles as discussed above, a-tDCS to the DLPFC and M1 might facilitate fatigue resistance through different mechanisms, such as suppressing perceptual fatigue or directly increasing motor units' recruitment, respectively. This assumption may align with the earlier-mentioned findings where a reduction in RPE and an improvement in fatigue resistance were observed when the DLPFC was stimulated (27), and both fatigue resistance and explosive performance (e.g., higher CMJ and shorter sprint times) improved when M1 was stimulated (28). Nevertheless, even though the M1 has a slightly different role compared to the DLPFC, the tDCS-induced additional activity in M1 was postulated to lessen the need for higher areas to provide neural drive to M1, and therefore, lower effort should also have been perceived during a relative period of exercise (58). Furthermore, as mentioned above, the effect of muscle fatigue varies across different types of exercise, suggesting the possibility that the area-specific effects of tDCS might manifest differently depending on the exercise.

As discussed above, when the M1 was stimulated, specific sprinting distances were completed with a lower rate of speed decline (28). However, this application could not enable individual to perform the abduction of the first dorsal interosseous muscle (70), knee extension (62–67,69), and cycling with lower performance reduction (72). On the other hand, when the DLPFC was targeted, no lower performance reduction was indicated in sprint (74), squat (73), and cycling (72), except for bench press (27). Accordingly, sprinting speed could be sustained for a longer period when the M1 was stimulated but not the DLPFC, whereas bench press velocity could be maintained when the DLPFC was stimulated. This finding may suggest the possibility of region-specific effect based on the area stimulated. Nevertheless, the fatigue resistance of more than half of the types of exercises (e.g., bench press, knee extension, the abduction of the first dorsal interosseous muscle, squat) was examined with either DLPFC, or M1 (27,62–67,69,70,73). Furthermore, except for cycling, the inconsistent results on sprint between different targeted areas were obtained from different sprinting protocol (e.g., distance, sets, rest) (28,74). Taken together, the electrode placement-based difference in the effects of a-tDCS may need additional investigation. However, the documented ergogenic effect of a-tDCS on VL reduction (27) might imply its potential benefit for the training program where the training volume is based on the VL tolerance, such as velocity-based training (VBT).

1.4. VELOCITY-BASED TRAINING

1.4.1. Fundamentals of velocity-based training and its relation to concentric motion velocity

The VBT is a training approach which dynamically prescribes the training volume by VL amplitude (88). For example, if the 10% VL is set as the termination threshold, a training set that started with a maximal speed of 0.7 m/s would continue until the velocity drops below 0.63 m/s (Figure 19b) (89). Therefore, this training method needs to be performed with the velocity tracking technology (e.g., linear position transducer (Figure 19a)) that provides real-time feedback on velocity and VL percentage (based on the fastest) (90). This volume prescribing method is different from the typical approach where a specified number of sets and repetitions to complete (e.g., 5 sets of 10 repetitions) at a given load is assigned (91).

Loading, volume, exercise type and order, rest duration, and movement velocity are variables that mediate the adaptive response to the training (92,93). However, within these variables, the level of effort experienced in every training set was considered as a key role in the magnitude and specificity of neuromuscular adaptations (94,95). The level of effort is based on the interaction between training intensity and volume and has been previously determined by the number of repetitions performed in each set with respect to the maximum number that can be completed against a given load (94,96,97). It was previously assumed that the training should be conducted to the point of muscle failure in order to maximize gains in strength and muscle mass (98). Nevertheless, such approach was shown to induce a fast-to-slow phenotypic remodeling in muscle fiber type, which is not desirable for competitive sports where high-speed, "explosive" actions are decisive for performance (99–105). Hence, the administration of level of effort (repetitions) could be a significant component of training program for sports performance development.

However, the number of repetitions that can be performed with a given % of one-repetition maximum (1RM) differs between athletes due to several factors such as training history, gender, absolute strength levels, and recent training exposure (106,107). Thus, to accomplish a specific training volume (e.g., 5 sets of 8 repetitions) at a certain percentage of 1RM, some work to concentric failure, and

some complete it with relative ease (107). Therefore, assigning the same number of sets and repetitions for all athletes may induce different levels of effort and fatigue (108,109). Hence, the adjustment of the level of effort through the prescription of repetitions might be imprecise and could therefore interfere with the development of targeted type of physical performance (e.g., explosive movement). In this context, it may be necessary to explore alternative methods that can accurately adjust the level of effort.

As a progressively increasing external mass is loaded, a reduction in lifting velocity occurs and continues until a 1RM load is reached (110). Aligning with this finding, a nearly perfect linear relationship between velocity and intensity (e.g., % of 1RM) has been consistently demonstrated across a range of exercises and submaximal loads (110). Furthermore, as fatigue increases, there is a transient decline in muscle fibers shortening speeds, relaxation times, and force-generating capacity that cause subsequent reductions in voluntary exercise velocity (110). These fundamental concepts have led to the recommendation that velocity output be used to accurately and objectively prescribe external loads and training volumes, regardless of fluctuations in fatigue and readiness (110). In line with this, VBT has been found to precisely stimulate the targeted neuromuscular adaptations (101,111,112).

The VBT approach was indicated to effectively avoid excessive fatigue (e.g., close to failure) that might result in undesired adaptations (e.g., fast-to-slow phenotypic remodeling in muscle fiber type) (101,102), and instead target specific adaptations (113). For instance, the VBT with low to moderate VL threshold (< 30%) were found to induce additional improvement in strength gain, especially in high velocity actions (e.g., jump performance, sprint ability) compared to the VBT with higher threshold (>30%VL) (101,111,112). Nevertheless, the VBTs with moderate to high VL thresholds resulted in greater muscle hypertrophy (114,115). Accordingly, the training load management through the adjustment of VL magnitude could be relatively effective in controlling the level of effort and optimizing the targeted neuromuscular adaptation (101,114,116). In addition, its strong relationship with a wide range of performance and physiological variables further supports its accuracy in adjusting the level of effort (88,89,109,117).

1.4.2. Performance measurement in VBT: repetition, speed and perception of effort.

As the training set progresses with maximal intended velocity for each repetition, a gradual and unintentional decrease in velocity is observed as fatigue develops and the number of repetitions approaches failure (88,118). Such VL was reported to reflex the impaired neuromuscular function (88), and its magnitude (percentage of VL relative to the fastest repetition) was found to have a strong positive relationship with the percentage repetitions that can be completed before muscle failure ($R^2 = 0.93–0.97$) (109). Besides, it was found that the changes in mean and peak velocity, power output, and force resulting from different percentage of VL (e.g., 10%, 20%, or 30%VL) were only slightly different between athletes across multiple sets of the back squat (89). Furthermore, the greater increase in the VL percentage was shown to produce more reductions in neuromuscular performance (countermovement jump), and higher increase in perceptual and metabolic response (lactate, ammonia) to a similar extent within and between athletes (88,117). Hence, the extent of the VL has been a precise indicator of the level of effort (93,109,119) that accounts for differences in individual work capacity (110). Furthermore, the VL amplitude could be a precise alterative measurement variable for maximal repetitions, velocity, and perceived effort.

1.4.3. Influence of fatigue and brain stimulation on VBT

As the training set progresses, fatigue is induced, leading to a transient decline in muscle fiber shortening speeds, relaxation times, and force-generating capacity, consequently causing a reduction in voluntary exercise velocity (110). Hence, the ability to withstand this reduction would mediate the volume in VBT, given that the VBT protocol terminates the training set when the targeted VL level is reached during exercise (89). As noted earlier, when the DLPFC was stimulated by a-tDCS, individual could perform bench press for more repetitions before reaching failure (figure 12) (27). Furthermore, their velocity during the bench press decreased at a slower rate (figure 13) (27), allowing them to perform more repetitions at higher velocity. Therefore, tDCS may increase VBT volume by enabling the stimulation receiver to sustain repetitions at a higher velocity for more

repetitions before the determined VL threshold is reached. Nevertheless, contrasting results were also observed, indicating that the tDCS was not able to reduce the VL during bench press and squat performed for a certain volume even if the same area (DLPFC) was stimulated (73,120). The lack of studies on the effect of tDCS on VL led to the difficulty to detect the potential underlying mechanism of confounding factors. However, the difference in exercise design among the studies (27,73,120) (training performed until failure or for a certain amounts) implies that the effect of tDCS on VL could manifest differently across distinct exercise modalities, and further exploration is warranted.

1.5. RELATIONSHIP BETWEEN TRANSCRANIAL DIRECT CURRENT STIMULATION AND VELOCITY-BASED TRAINING PERFORMANCE

1.5.1. Impact of a-tDCS on movement speed and neuromuscular fatigue

In addition to VL, movement W, T, power (62–66,71,72), or completing time (e.g., sprinting) were also employed to evaluate the effect of tDCS on fatiguability (28,74). The amplitude of movement W, T, and power reduction during high intensity exercise, such as knee extension, cycling was not altered by the tDCS (62–66,71,72). However, the level of completing time-based fatigue index during sprinting (equation 1) was reduced when tDCS was applied to M1, but not DLPFC (28,74). These findings might suggest that the effect of tDCS on fatigue is partly based on exercise or stimulated areas. Nevertheless, most of the absent effect of tDCS on fatiguability during knee extension was only obtained from tDCS targeting M1 (62,64–66,71,72). Furthermore, the contrasting results on fatigue during sprinting resulted from different exercise protocol (e.g., distance, number of sets, rest). Accordingly, the effect of tDCS on fatigue endurance may require further investigation to determine the optimal tDCS set up for each exercise.

Although the effects of tDCS on fatigue endurance are not fully understood due to the mixed results mentioned above, the positive report on its reducing effect on VL, as noted earlier (27), still deserves attention, and it implies the potential to enhance VBT volume by allowing more repetitions before reaching the assigned VL threshold.

1.5.2. Scientific evidence on tDCS and performance in strength sports

Along with the improvement in fatigue endurance of bench press after tDCS, such as the VL reduction mentioned above, a lower perceived effort was also found (27). This finding implies that the previously assumed enhancement in VBT volume after tDCS could coincide with a reduction in perceived effort. However, the improvement in fatigue endurance during other exercises, such as sprinting, did not occur with lower perceived effort after tDCS (28). Therefore, the tDCS-induced alteration in fatigue resistance may be perceived differently across various exercises. Nevertheless, the performance alterations in these exercises were triggered by different stimulated areas. When the DLPFC was stimulated, the VL reduction during bench press was accompanied by lower perceived effort (27). In contrast, when the M1 was targeted, the lower time-based FI during sprinting was observed with no change in perception (28). These findings suggest that the reduction in perceived effort may occur when the VBT volume was improved by the tDCS applied to DLPFC.

The effect of tDCS on perceptual alteration has also been examined in other exercises, such as leg press (57), squat (121), and cycling (58). However, the perceptual changes during these exercises did not seem to be mediated by the stimulated area, as assumed above. For example, even when the DLPFC was targeted and physical performance was consequently improved, no perceptual change was also observed (57). Furthermore, the tDCS to M1 was also found to lead to an improvement in fitness along with reduction in perceived effort (58). Nevertheless, these perceptual alterations were found with the change in endurance (e.g., maximal repetition, and time to failure), but not fatigue resistance (e.g., performance reduction rate). Furthermore, the studies on the change in perceptual and fatigue resistance after tDCS were lacking. Hence, additional studies might be needed to facilitate the understanding of tDCS's effect on fatigability from psychological aspect.

In addition to perceptual measure, improvement in fatigue resistance after t-DCS were also found to occur alongside changes in explosive strength, such as in countermovement jump and shorter sprinting time (28). However, such a link between explosive and fatigability performance was observed only when the M1 was stimulated (28). When the tDCS was applied to DLPFC, the induced reduction

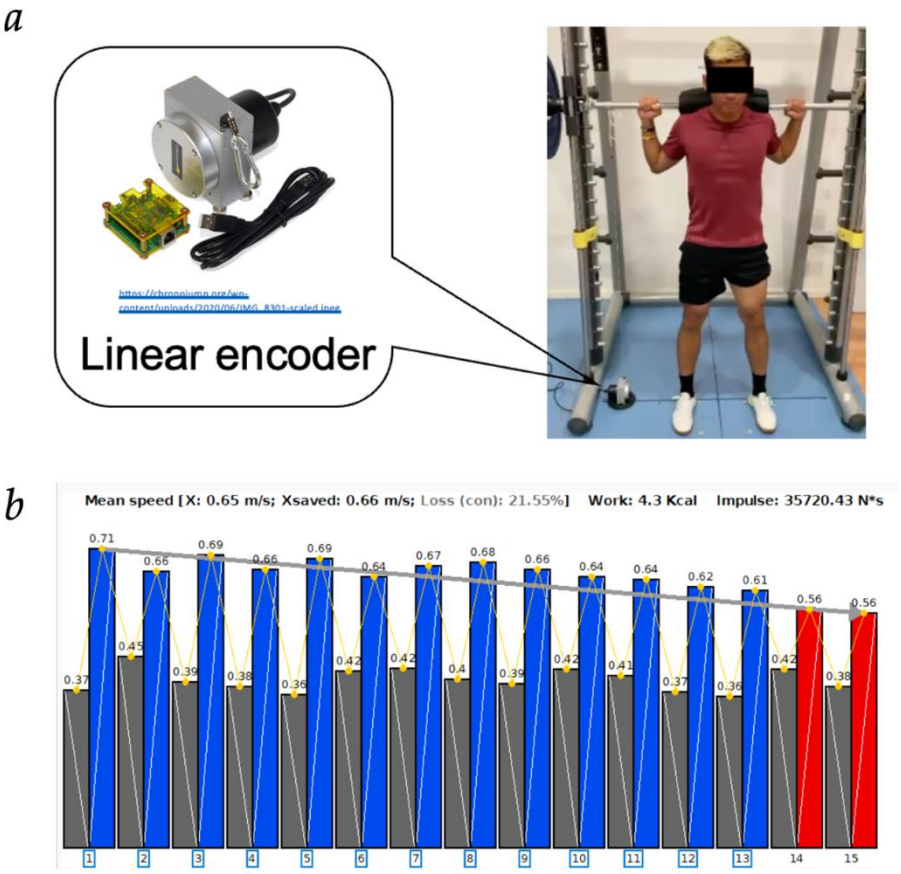
in VL was observed with no change in explosive performance, such as power output of the bench press (27). Therefore, the improvement in explosive performance of repetitive high-intensity activity may be detected when the reduction in fatigability is induced by the tDCS targeting M1. This assumption may be supported by the previous findings showing that most of the improvements in explosive strength (e.g., CMJ, power output, rate of force development) were obtained from the tDCS applied to the M1 (28,59,60,122–126), but not the DLPFC (27,72–74,123,127). However, contrasting results were still observed among the study employing tDCS over M1, even though this approach has been substantiated to improve explosive strength in various exercises, as mentioned above. Nevertheless, these mixed results were obtained among participants with distinct characteristics (48) or sex (49), as well as across different tDCS configurations (e.g., tDCS type, density, duration) (50)(51)(44) and exercise protocol (e.g., duration of sprinting) (50)(44). Therefore, confirmatory analysis might be needed to explore the optimal configuration of tDCS applied to M1 for certain types of population and exercise protocol.

Despite the contradictory finding on the effect of tDCS to M1 on explosive performance, as mentioned above, the nearly entire improvements of explosive strength resulting from the tDCS to M1 may reinforce assumption of the area-based effect of tDCS on explosive performance. However, these findings were obtained with the intention to only explore the acute effect of tDCS on explosive performance, but not to evaluate the relationship between the tDCS-induced change in explosive and fatigability performance (28,59,60,122–126). Furthermore, the reference for literature on this link is lacking. Therefore, more studies are warranted, and the explosive component of VBT, such as contraction velocity or power output, may be worth investigating to see its alteration and VBT volume after tDCS application.

Explosive performance seems to be reflected in the contraction speed of exercise (128). For example, changes in the mean propulsive velocity of the back squat and CMJ in rugby players at 30 minutes and 48 hours after the match, as well as in strength-trained men after resistance exercise training, were found to be correlated (128,129). This was thought to be due to the fact that both need maximal effort of major lower-body musculature, and that velocity of movement and CMJ

height are influenced by muscle-shortening velocity (129). Thus, the velocity of exercise during VBT may also be altered by the tDCS. In addition, considering the earlier discussed potential link between the changes in fatigue resistance and explosive strength following the tDCS to the M1, the assumed enhancement in VBT volume after tDCS may coincide with improvement in VBT velocity when M1 was stimulated.

Figure 19. Linear position transducer setup.



Note: The image of Linear encoder is from "https://chronojump.org/product/linear-encoder-kit/." Figure a represent the tDCS set up. Figure b represent the real-time feedback data on speed, with blue, gray, and red bars standing for the concentric speed, eccentric speed, and concentric speed lower than the determined velocity loss threshold, respectively.

II – JUSTIFICATION

II - JUSTIFICATION

The ability to attenuate fatigue (e.g., power loss) is crucial for exercises requiring sustained maximal efforts for a specific duration (e.g., ~15-60s) (11). Over time, repeated exposure to specific physical training will be the main intervention to achieve a chronic reduction in exercise-induced fatigue. However, other techniques, such as a-tDCS, can induce faster, acute reductions of exercise-induced fatigue through cortical modulation (27,28). This non-invasive brain stimulation technique has shown various behavioral effects on gross motor performance (53), particularly concerning muscle strength and the ability to sustain maximal or submaximal efforts over a certain period (54–58). For example, a-tDCS allowed individuals to perform more repetitions with a reduced rate of VL and perceived exertion during high-intensity resistance training (e.g., explosive bench press) before reaching failure (27). In addition to its potentially positive impact on fatigue tolerance, a-tDCS may also enhance maximum explosive performance, such as the height of countermovement jump (CMJ) (59,60) and sprint times (28).

Despite the improvements in fatigue tolerance and power production associated with a-tDCS, these benefits may not occur simultaneously. For instance, when the DLPFC is stimulated, the reduction in VL induced by a-tDCS (i.e., increased fatigue tolerance) is not accompanied by improved maximal power but rather by a reduction in the RPE (27). In contrast, stimulation of the M1 has shown benefits in explosive actions such as jumping (59,60) or sprinting (28), coinciding with improvement in fatigue resistance (less rate of sprinting speed loss), but not with perceptual alteration (28). Therefore, the physical performance aspect that the ergogenic effect of a-tDCS acts on might depend on the stimulated area.

The DLPFC modulates the motor cortex by integrating cognitive and peripheral information, playing an important role in inhibition of subjective fatigue (27,74). On the other hand, the M1 is the main area controlling voluntary human movement (72). Thus, the increased activity in these two areas after a-tDCS might develop fatigue resistance (e.g., VL) through different mechanism. The stimulation of DLPFC might modulate the perceived effort during exercise, reducing the performance decline. The stimulation over M1 might enhance the magnitude and efficacy of the supraspinal drive, leading to the increased motoneuron recruitment

and/or firing rate (124,125), reducing the input needed to recruit the necessary motoneurons for muscle contraction (58), thereby prolonging the maximal motor output. Accordingly, alterations in fatigue resistance after a-tDCS might coincide with perceptual or explosive changes during exercise, depending on the stimulated areas.

The ergogenic effect of a-tDCS on fatigue resistance might be observed in the exercise where training volume is determined by the level of fatigue, such as VBT. The VBT uses lifting velocity of each repetition during a resistance training as a proxy for instantaneous neuromuscular performance (88). By monitoring lifting velocity, the fatigue induced by the resistance training stimulus can be managed by setting a VL threshold. When this threshold is exceeded, the set is stopped, indirectly influencing the number of repetitions performed in each set and the total training volume (88). This approach allows monitoring the effect of a-tDCS over instantaneous maximum neuromuscular performance against a submaximal load (i.e. power production) in an unfatigued state (first repetitions of the first set) but also determining its effects on the individual's ability to withstand fatigue (VL).

III – HYPOTHESES AND OBJECTIVES

III - HYPOTHESES AND OBJECTIVES

OBJECTIVE

To investigate the acute effects of anodal transcranial direct current stimulation (a-tDCS) on velocity-based squat training (VBT), focusing on changes in performance and perception under a 15% velocity loss threshold.

Specific Objectives:

1. Examine how a-tDCS influences the number of repetitions performed, mean concentric movement velocity, and ratings of perceived exertion (RPE) during VBT in the back squat exercise, applying a 15% velocity loss criterion.
2. Determine whether stimulation over different cortical areas (M1 vs. DLPFC) results in distinct effects on explosive performance and subjective effort during VBT.
3. Evaluate how a-tDCS-mediated changes in fatigue resistance, explosive strength, and perceived exertion interact, thereby providing insight into the neuromuscular mechanisms underlying any observed improvements in performance.

HYPOTHESIS

We hypothesized that a-tDCS targeting the DLPFC will lead to the enhancement in number of repetitions and induce reduction in RPE during VBT. However, these alterations would not occur with the change in movement velocity. On the other hand, we assumed that a-tDCS targeting M1 would also enable individuals to perform more repetitions during VBT. Furthermore, this alteration would coincide with the increase in movement velocity. However, the RPE does not seem to be altered.

IV – MATERIAL AND METHODS

IV -MATERIAL AND METHODS

4.1. PARTICIPANTS

The sample size was calculated using G*POWER software (version 3.1.9.6, University of Kiel, Germany) with a power (1-β err prob) of 0.8, α = 0.05 and an effect size of 0.35 (86). This calculation indicated that 15 participants were needed for the pre-specified study. Due to the variability in tDCS-induced alteration of motor cortical excitability between different sexes (130), only male participants were recruited in this study. Fifteen young adult males [age = 21.8 ± 2.6 years, body mass = 77.1 ± 8.5 kg, height = 177.9 ± 6.6 cm, one repetition maximum 1RM/body mass for squats = 1.2 ± 0.22 /kg] volunteered to participate in this study. All participants were recreationally resistance trained males with 3 ± 1.7 years of resistance training experience, as determined by a questionnaire and interview. Prior to the study, they signed an informed consent (see appendix 1) and completed the tDCS assessment questionnaire (see appendix 2) to assess their suitability to participate in the tDCS intervention. However, two participants dropped out of the study for reasons unrelated to the intervention, resulting in a final sample of 13 subjects. The study protocol adhered to the tenets of the Declaration of Helsinki and was approved by the Institutional Review Board (No. CE031908).

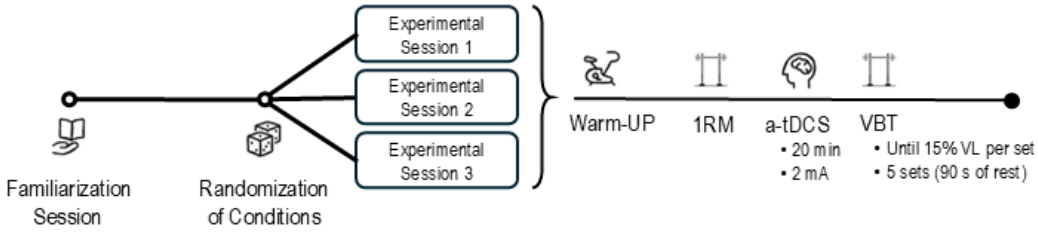
4.2. STUDY DESIGN

A crossover randomized controlled trial design was employed in this study. Participants attended the laboratory four times separated by a week. To minimize any variability in performance, the first visit was conducted as a familiarization session where participants were introduced to the a-tDCS, VBT protocol, and the Smith machine used for subsequent sessions. In this session, the foot position for the squat was recorded and replicated in the experimental sessions to avoid potential technical variability. During the experimental sessions, all participants completed the three distinct a-tDCS conditions one week apart in a randomized order (using https://www.randomizer.org) to ensure an equal balance between treatments for each participant. In addition, the study was designed to be double-

blinded, meaning neither the participants nor the researchers knew which a-tDCS condition was applied during each session, except for the researcher administering the intervention. Participants were instructed to avoid any strenuous exercise 48 hours before each session, and refrain from caffeine and alcohol intake 24 hours before each session.

As shown in Figure 22 with the procedures detailed in the next subsection, each session began with a warm-up, then the estimated 1RM test for the squat exercise in Smith machine (Technogym, Cesena, Italy) was measured, followed by the a-tDCS intervention. Following the a-tDCS intervention, the VBT was performed. The repetitions performed (reps), movement velocity, and RPE were recorded during the VBT. To ensure consistency of measurements, all experimental sessions were conducted in the same room with controlled environmental conditions (i.e., temperature and humidity). In addition, all sessions were held in the morning at the same time for each participant to minimize the effects of circadian rhythm on participants' performance.

Figure 20. Flow chart of experimental procedure.



4.3. PROCEDURE

4.3.1. Load-velocity test procedure and estimated 1RM

Each session began with the warm-up consisting of a five min of cycling (ergometer) at 100-120 watts and several joint mobilization exercises (i.e., hip extension, hip abduction, hip adduction, hip rotation, and sumo squat lung rotation on each side). Thereafter, all participants were tested for their estimated squat 1RM using the Smith machine. During squats, the barbell rested across the back at the level of the acromion and upper trapezius. The squat began from an upright position with the knees and hips fully extended, feet approximately shoulder-width apart, flat on the floor either parallel or externally rotated to a comfortable degree. To avoid the rebound effect within and between each repetition, the participants were instructed to perform the squat as follows: After the verbal command "go", participants began descending (eccentric phase) at a controlled velocity (~0.30 - 0.48 m/s) following a verbal pacing of "1, 2," until reaching a 90° knee angle, and then immediately reversed the motion and ascended back (concentric phase) at maximal intended velocity to the upright position. Upon returning to the standing position, they were instructed to maintain their position until another verbal command of "go" was given. The timing between the "go" commands was arbitrarily determined. The safety catches of the smith machine were placed at the height where the angle of participants' knee was ~90° when they descended to the bottom of the movement. To replicate stance width, feet position, and eccentric ROM, these were recorded on the first visit (familiarization session) and individually adjusted on the subsequent visits.

A linear encoder (Chronojump, Barcelona, Spain) attached perpendicularly to the barbell was used to register bar velocity and estimate the 1RM. Initial load was set at ~30-50 kg for all participants and was gradually increased in ~10 kg increments until the mean velocity was lower than ~0.60 m/s, which corresponds to ~85% 1RM (131). During the test, three repetitions were executed for light (≤ 50% 1RM), two for medium (50% - 80% 1RM), and only one for the heaviest loads (≥ 80% 1RM). Inter set rests ranged from three (for light load) to five min (for heavy loads). Only the best repetition at each load, according to the criteria of fastest MEAN VELOCITY, was considered for subsequent analysis. The 1RM was

calculated from the lowest MEAN VELOCITY (Vmin in m/s) attained against the heaviest load (Lmax in kg) lifted in the progressive loading test using the following equation 5 (131):

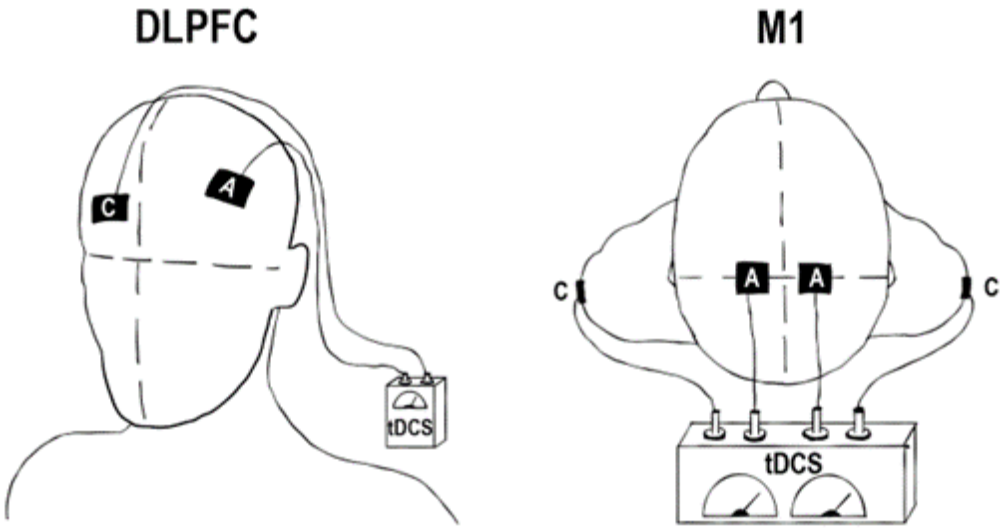
Equation 5.

$$1RM = (100 \times Lmax) / (-12.87 \times Vmin^2 - 50.71 \times Vmin + 117)$$

4.3.2. Transcranial direct current stimulation procedure

Participants remained seated during the implementation of a-tDCS and were stimulated for 20 min at 2.0 mA. The a-tDCS was applied using a constant current electrical stimulator (ApeX Type A 18V, ApeX Electronics, NY, USA) connected to a pair of rectangular rubber electrodes covered with a sponge moisturized by 0.9% NaCL saline. (Figure 23). In the M1 condition, two tDCS devices were utilized to place an anodal electrode on each side of the vertex (estimated leg muscles M1 representation), the cathodal electrodes were placed on the ipsilateral shoulders (i.e. bihemispheric M1 stimulation). In the DLPFC condition, a single tDCS device was used, with the anode on the left DLPFC with the cathode on the right orbitofrontal cortex. The electrode placements were determined according with the international 10–20 system EEG. The stimulation ramped up over 30 s until the target intensity of 2.0 mA was reached and then remained for 20 min before ramping back down to 0 mA over 30 s. For the placebo stimulation condition (SHAM), the electrodes were placed in the same position as for the M1 or DLPFC, and the stimulation ramped up over 30 s and immediately ramped down to 0 mA to imitate the initial sensations of active stimulation to maintain participants' blinding to the received type of tDCS. To hold the electrodes on the scalp and shoulders, the DigichargeTM adjustable elastic head straps with Velcro (8.3 cm × 57.3 cm) were used, respectively.

Figure 21. Placement of tDCS setup.



4.3.3. Velocity-based training

The same Smith machine used for estimating the squat 1RM was used for VBT. Before VBT, the same warm-up was performed before the 1RM test was completed. Thereafter, participants performed a specific warm-up consisting of two sets of eight and four repetitions (90 s rest) with a load of 40% and 60% of 1RM, respectively. After 90 s of rest following the specific warm-up, participants performed the VBT consisting of five sets at 70% of 1RM. During each set, participants performed as many repetitions as possible until a 15% VL was exceeded and rested 90 s until the next set. During training, participants were encouraged to perform each repetition at maximum intended lifting velocity in a consistent and standardized manner by the same researcher to ensure that all participants received comparable motivational support to maximize performance across all sets. The 15% VL threshold was measured using a linear encoder to monitor the lifting velocity of each repetition, defined as a 15% reduction compared to the velocity of the first repetition. The mean velocity of each repetition and the total number of repetitions of each set were measured. In addition, the RPE value

was assessed before the start of the session and just after each training series using the OMNI-RES scale (with scores ranging from 0 to 10 points) (132) to measure the participants' subjective perception of exertion and its evolution throughout the training.

4.3.4. Statistical analysis

All statistical analyses were performed using JAMOVI software (version 2.3.28). The normality of the distributions of the dependent variables was assessed using the Shapiro-Wilk test. Subsequently, to identify possible responders and non-responders to the a-tDCS interventions, a k-means cluster analysis was employed, classifying participants into two groups based on normalized total repetitions and normalized mean velocity. The normalized total repetitions and normalized mean velocity were calculated for each active condition (DLPFC and M1) by subtracting the values obtained in the SHAM condition from those in the respective condition. This approach allowed for the evaluation of changes in individual performance attributable to each type of stimulation, considering both the capacity to perform more repetitions and improvements in execution speed. The inter-session reliability of the 1RM estimation under the SHAM, DLPFC and M1 conditions was assessed using intraclass correlation coefficients (ICC) employing a mixed-effects model. ICC values were interpreted according to the following categories: < 0.5 (poor reliability), 0.5–0.75 (moderate reliability), 0.75–0.90 (good reliability), and > 0.90 (excellent reliability) (133).

Additionally, analyses of variance (ANOVA) were conducted to evaluate the effect of the different stimulation conditions (SHAM, DLPFC, and M1) on the variables of interest. For the variables of repetitions per set, mean velocity, and RPE, a two-factor repeated measures ANOVA (CONDITION × SET) was performed. This analysis allowed us to examine the main effects of CONDITION (SHAM, DLPFC, M1) and SET (Sets 1 to 5), as well as the interaction between both factors. To compare the total number of repetitions among the different stimulation conditions, a one-factor repeated measures ANOVA (CONDITION) was employed. The assumption of sphericity was verified using Mauchly's test. In cases where the sphericity assumption was violated, Greenhouse-Geisser corrections were applied. When the ANOVAs revealed significant effects, post hoc tests with

Bonferroni correction were performed to identify specific differences between conditions and sets. Effect sizes were calculated using partial eta squared (η_p^2). The statistical significance level was set at $p < 0.05$ for all tests. All data are presented as means ± standard deviations.

V – RESULTS

V - RESULTS

Table 1 shows the comparison of the 1RM obtained in each experimental condition. The ICC indicated excellent reliability between the measurements performed in the different sessions. In addition, repeated measures ANOVA revealed no significant differences in 1RM estimates between stimulation conditions.

Table 1. Comparison of the estimated 1RM (kg) between each experimental condition (n = 15).

Condition			Reliability Analysis	
SHAM	M1	DLPFC	ICC (95%CI)	ANOVA
95.8 ± 19.3	93.1 ± 18.8	93.5 ± 17	0.93 (0.84, 0.97)	$p = 0.908$

ICC: Intraclass correlation coefficient; CI: Confidence interval; SHAM: placebo stimulus; M1: primary motor cortex; DLPFC: dorsolateral prefrontal cortex.

After k-means cluster analysis was applied to the normalized differences in velocity and total number of repetitions between the active conditions (DLPFC and M1) with respect to SHAM, two clusters with clearly differentiated profiles were identified (see Table 2). Non-responders showed reductions in both velocity and total number of repetitions, whereas Responders showed improvements in total number of repetitions without substantial changes in velocity.

Table 2. Classification of response pattern based on the number of repetition during VBT.

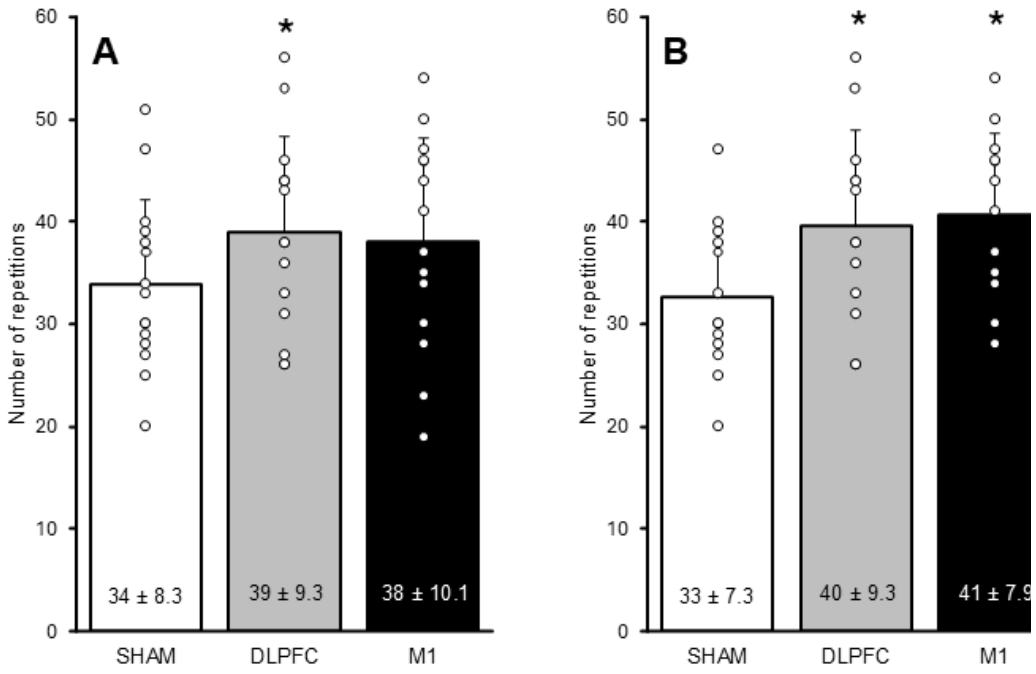
Cluster	N	Centroid of clusters			
		DLPFC (mean velocity)	M1 (mean velocity)	DLPFC (total reps)	M1 (total reps)
Responders	13	-0.000	-0.026	7.000	8.154
Non-responders	2	-0.032	-0.066	-7.000	-21.500

Note: N = number of subjects. All the statistics in the centroid of clusters columns indicate the normalized values of DLPFC and M1 condition subtracted from the SHAM condition.

Number of repetitions

A repeated-measures ANOVA for total repetitions across all participants revealed no significant main effect of Condition ($F_{(2,28)} = 2.32$, $p = 0.117$, $\eta_p^2 = 0.142$), although post-hoc comparisons indicated that participants performed approximately five more repetitions under DLPFC compared to SHAM (mean difference = –5.133, $p = 0.035$), with no significant differences observed between SHAM and M1 or between DLPFC and M1 (Figure 22A). In contrast, among responders (n = 13), the condition effect was significant ($F_{(2,24)} = 11.3$, $p < 0.001$, $\eta_p^2 = 0.485$), and post-hoc tests showed that both DLPFC (mean difference vs. SHAM = –7.00, $p = 0.001$) and M1 (mean difference vs. SHAM = –8.15, $p = 0.001$) produced a substantially higher total number of repetitions than SHAM, with no difference between DLPFC and M1 (Figure 22B).

Figure 22. Total repetitions performed under each stimulation condition. (A) Results for the entire sample (n = 15), and (B) for the responder subgroup (n = 13).



Note: *: statistically significant differences compared to the SHAM condition ($p < 0.05$). DLPFC: Dorsolateral prefrontal cortex; M1: Primary motor cortex.

When examining repetitions per set in the total sample (Table 3), the repeated-measures ANOVA did not reveal significant main effects or interactions. Specifically, there were no significant differences across Set ($F_{(4,168)} = 1.975$, $p = 0.101$, $\eta_p^2 = 0.045$) or in the Set × Condition interaction ($F_{(8,168)} = 0.701$, $p = 0.690$, $\eta_p^2 = 0.032$), and the between-subjects effect of Condition was also not significant ($F_{(2,42)} = 1.30$, $p = 0.282$, $\eta_p^2 = 0.058$). However, for responders, the repeated measures ANOVA showed a significant main effect of Set ($F_{(4,144)} = 2.887$, $p = 0.031$, $\eta_p^2 = 0.074$) and a significant main effect of Condition ($F_{(2,36)} = 3.73$, $p = 0.034$, $\eta_p^2 = 0.172$), although the Set × Condition interaction remained non-significant ($F_{(8,144)} = 0.698$, $p = 0.693$, $\eta_p^2 = 0.037$). Post-hoc comparisons for the responders indicated no statistically

significant differences between specific sets after Bonferroni adjustment, but did reveal that M1 led to a greater number of repetitions per set than SHAM (mean difference = –1.631, $p = 0.049$). No significant differences were observed between DLPFC and SHAM (mean difference = –1.400, $p = 0.111$) or between DLPFC and M1 (mean difference = –0.231, $p = 1.000$).

Table 3. Data on the number of repetitions and speed per set.

Nº of reps per set	Set-1	Set-2	Set-3	Set-4	Set-5
Total ($n = 15$)					
SHAM	7 ± 3.1	6 ± 2.1	7 ± 2.8	7 ± 2.6	7 ± 2.2
DLPFC	9 ± 3.2	8 ± 2.7	8 ± 3.4	7 ± 2.1	7 ± 2.6
M1	8 ± 3.2	8 ± 2.5	7 ± 2.4	7 ± 2.8	8 ± 2.9
Responders ($n = 13$)					
SHAM	7 ± 2.9	6 ± 2.0	6 ± 2.6	7 ± 2.7	6 ± 2.2
DLPFC	10 ± 2.6	7 ± 2.7	8 ± 3.6	7 ± 2.2	7 ± 2.7
M1	9 ± 2.6	9 ± 2.4	7 ± 2.0	8 ± 2.7	8 ± 2.9

Mean velocity	Set-1	Set-2	Set-3	Set-4	Set-5
Total ($n = 15$)					
SHAM	0.59 ± 0.04	0.59 ± 0.05	0.58 ± 0.05	0.57 ± 0.06	0.56 ± 0.06
DLPFC	0.58 ± 0.05	0.57 ± 0.05	0.57 ± 0.05	0.56 ± 0.05	0.56 ± 0.06
M1	0.60 ± 0.06	0.59 ± 0.07	0.59 ± 0.06	0.58 ± 0.07	0.58 ± 0.06
Responders ($n = 13$)					
SHAM	0.58 ± 0.04	0.58 ± 0.05	0.57 ± 0.05	0.56 ± 0.06	0.56 ± 0.06
DLPFC	0.58 ± 0.05	0.57 ± 0.05	0.57 ± 0.06	0.56 ± 0.05	0.57 ± 0.06
M1	0.61 ± 0.06	0.59 ± 0.07	0.59 ± 0.07	0.59 ± 0.07	0.59 ± 0.05

SHAM: placebo stimulus; M1: primary motor cortex; DLPFC: dorsolateral prefrontal cortex.

Lifting velocity

In the total sample (Table 3), the analysis of execution velocity across sets revealed a significant main effect of Set ($F_{(4,168)}$ = 5.807, p < 0.001, η_p^2 = 0.121), indicating that the average velocity changed throughout the five evaluated sets. Bonferroni-adjusted post-hoc comparisons showed significant differences between Set-1 and Set-4 (mean difference = 0.021, p = 0.016), as well as between Set-1 and Set-5 (mean difference = 0.024, p = 0.021), suggesting that initial execution velocity was higher than in the later sets. In contrast, there was no significant main effect of condition ($F_{(2,42)}$ = 0.519, p = 0.599, η_p^2 = 0.024), nor a significant Set × Condition interaction ($F_{(8,168)}$ = 0.424, p = 0.905, η_p^2 = 0.020). Overall, these results indicate that in the total sample, execution velocity declined as the sets progressed, while brain stimulation did not significantly influence this pattern.

In the responder subgroup (Table 3), a repeated-measures ANOVA again revealed a significant main effect of Set ($F_{(4,144)}$ = 3.245, p = 0.014, η_p^2 = 0.083), indicating changes in execution velocity over the course of the sets; however, after Bonferroni adjustments, none of the individual set-to-set comparisons reached statistical significance. Similar to the total sample, there was no significant main effect of Condition ($F_{(2,36)}$ = 1.09, p = 0.348, η_p^2 = 0.057) and no significant interaction between Set and Condition ($F_{(8,144)}$ = 0.446, p = 0.892, η_p^2 = 0.024). In sum, while responders exhibited a statistically significant overall effect of set progression on execution velocity, this did not translate into significant pairwise differences between sets or conditions after adjustment, and the pattern of no discernible influence of brain stimulation on velocity remained consistent.

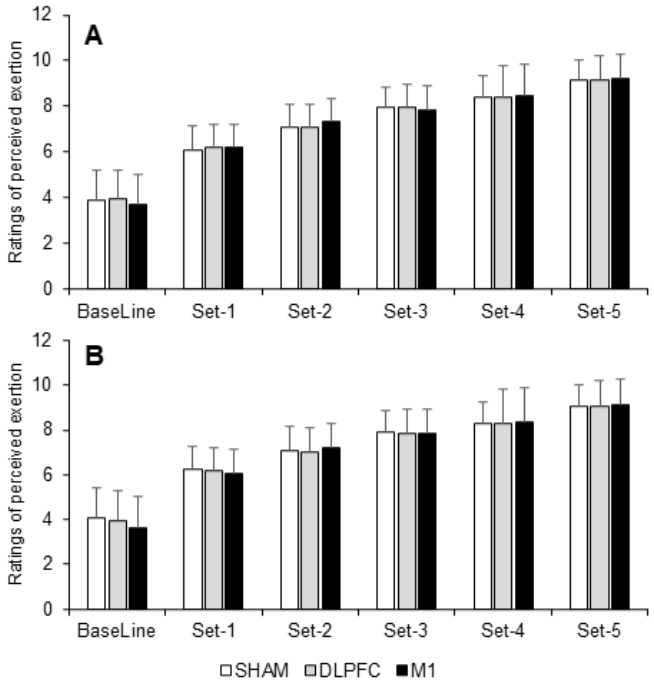
Rating of perceived exertion

In the total sample (Figure 23A), the repeated-measures ANOVA for RPE revealed a significant main effect of Set ($F_{(5,210)}$ = 429.871, p < 0.001, η_p^2 = 0.911), indicating a progressive increase in perceived exertion as the sets progressed. Bonferroni-adjusted post hoc comparisons showed that RPE increased significantly from the baseline to every subsequent set, as well as between different sets, suggesting a marked rise in perceived effort as fatigue accumulated. In contrast, there were no significant differences in RPE between stimulation conditions ($F_{(2,42)}$ = 0.0169, p = 0.983, η_p^2 = 0.001), nor were there any significant Set × Condition

interactions ($F_{(10,210)}$ = 0.314, p = 0.977, η_p^2 = 0.015). Overall, these findings indicate that in the total sample, RPE increased substantially over the course of the sets, regardless of the type of stimulation applied.

Among the responders (Figure 23B), a similar pattern emerged. The repeated-measures ANOVA also indicated a significant main effect of Set ($F_{(5,180)}$ = 359.253, p < 0.001, η_p^2 = 0.909), reflecting a progressive increase in RPE across the exercise sets. As in the total sample, the responders showed no significant main effect of stimulation Condition ($F_{(2,36)}$ = 0.0233, p = 0.977, η_p^2 = 0.001) and no Set × Condition interaction ($F_{(10,180)}$ = 0.499, p = 0.889, η_p^2 = 0.027). The Bonferroni-adjusted post hoc comparisons confirmed that perceived exertion increased consistently as the sets progressed, mirroring the results observed in the overall group.

Figure 23. Ratings of perceived exertion values reported per set for (A) the entire sample (n = 15) and (B) the responder subgroup (n = 13).



Note: DLPFC: Dorsolateral prefrontal cortex; M1: Primary motor cortex.

VI – DISCUSSION

VI -DISCUSSION

This study aimed to examine the acute effects of anodal transcranial direct current stimulation (a-tDCS) over the M1 and DLPFC on total repetitions, movement velocity, and RPE during velocity-based back-squat training to a 15% velocity loss threshold. Our findings indicate that, when considering the entire sample, a-tDCS over DLPFC led to a modest but significant increase in total training volume compared to SHAM, while M1 stimulation did not produce consistent improvements across the group. However, in a subset of participants (i.e., responders), both M1 and DLPFC stimulation conditions elicited a greater number of total repetitions than SHAM, underscoring notable inter-individual variability in the response to cortical neuromodulation (134). Interestingly, these enhancements in training volume were not accompanied by concurrent improvements in execution velocity or reductions in RPE, suggesting that the mechanism through which a-tDCS augments performance is not directly related to perceived exertion or explosive neuromuscular output per se. Instead, these results point to more subtle neuromodulatory effects, possibly linked to enhanced cortical excitability or altered central fatigue perception thresholds. Such modulation may influence an individual's capacity to sustain output over multiple sets rather than altering the characteristics of each individual repetition. Although previous studies have reported that M1 stimulation can enhance power-related measures and that DLPFC stimulation can reduce subjective effort perception (27), our data highlight the complexity of these effects, emphasizing that acute a-tDCS does not uniformly improve all aspects of neuromuscular performance and that its ergogenic potential may be highly dependent on individual responsiveness and cortical target site.

The variability in individual responses to a-tDCS observed in our study underscores the complexity of its neuromodulatory effects and the challenges associated with predicting who may benefit most. Numerous factors could influence the outcome of tDCS interventions, including cranial and brain anatomy, local inhibitory-excitatory balance, baseline neuromuscular function, psychological status, neurotransmitter systems, and genetic predispositions (74). Such inter-individual variability in cortical responsiveness has been well-documented (134),

and our data further suggests that these differences may translate into distinct performance outcomes. However, the precise neurophysiological mechanisms by which increased cortical excitability—if present—enhances or fails to enhance neuromuscular performance remain unclear. We did not assess cortical excitability directly, and previous research has yet to establish a definitive link between changes in cortical activity and acute improvements in resistance training performance (74). Moving forward, it will be essential to integrate neurophysiological assessments alongside performance measures to identify reliable biomarkers of a-tDCS efficacy. Such an approach may help refine individualized neuromodulation strategies, improving the likelihood of achieving meaningful performance gains in applied settings.

The observed increase in total training volume following a-tDCS in our study appears to stem from subtle, cumulative improvements across multiple sets rather than pronounced effects within each individual set. Although no significant main effect of stimulation condition emerged for total repetitions in the entire sample, post-hoc comparisons indicated that DLPFC stimulation led to approximately five additional repetitions compared to SHAM, and responders exhibited substantial increases in total repetitions under both DLPFC and M1 conditions. Interestingly, these enhancements were not accompanied by changes in mean velocity or RPE, and per-set analyses did not reveal statistically significant differences except in the responder subgroup, where M1 slightly outperformed SHAM. Such findings suggest that the ergogenic benefits of a-tDCS may not manifest uniformly at the set level, possibly because the modest central facilitation induced by a-tDCS (e.g., altered transmembrane potentials, modulated cortical excitability) (135,136) influences overall fatigue tolerance rather than immediate performance metrics within each bout. These results align partially with previous studies reporting that M1 stimulation can mitigate the decline in sprint performance (28) and that DLPFC stimulation can reduce velocity loss during high-intensity bench press exercises (27). However, the present protocol—characterized by a 15% velocity loss threshold—may have induced primarily peripheral fatigue, potentially masking the central effects of a-tDCS on fatigue resistance. Since a-tDCS primarily targets central mechanisms, its impact may become more evident as exercise approaches volitional failure or under conditions that elicit greater central fatigue (72-74). Additionally, the chosen tDCS configuration and exercise modality may have

limited the transfer of central neuromodulatory benefits into observable changes in velocity or perceived exertion.

In previous studies, improvements in explosive performance following a-tDCS were generally evaluated under predefined workloads (137), such as completing a fixed number of vertical jumps or executing a short, time-bound sprint (60,124). In contrast, our assessment of velocity changes took place within a VBT protocol, where exercise volume was not predetermined but rather dictated by a velocity loss threshold. Under these conditions, participants faced two simultaneous objectives: maximizing repetition velocity and performing as many repetitions as possible before reaching the predefined velocity decrement. This dual intention may have led them to strategically modulate their effort, potentially withholding maximum velocity on individual repetitions to prolong their set and thus obscuring any direct a-tDCS-induced enhancements in explosive output. Moreover, the tDCS configuration employed in our study differed in certain parameters from the setups used in other investigations reporting positive effects on explosive performance (59,124). Such discrepancies in stimulation parameters could also limit the comparability of results and diminish the observable impact on power-related measures. Taking together, these factors highlight the importance of considering both the nature of the exercise task and the specific tDCS setup when interpreting the influence of neuromodulation on explosive performance metrics.

Considering these discrepancies in tDCS protocols and outcomes, it is worth noting that the electrode size and stimulation parameters employed in the present study were comparable to those used in previous research that has reported positive effects on explosive performance (27,59,124,125). Furthermore, our participants shared similar characteristics (e.g., healthy, young adults) and underwent stimulation intensities and durations aligned with studies where enhanced explosive strength has been observed (28,59,122,124-126). Despite these similarities, the lack of a measurable effect on squat velocity in our investigation suggests that other variables—such as exercise modality, intensity, or the specific demands imposed by a velocity-based training protocol—may critically modulate the impact of a-tDCS.

Differences in baseline performance levels, psychological factors, or individual susceptibility to cortical stimulation may further contribute to inter-

study variability (138,139). Likewise, subtle distinctions in tDCS waveform characteristics, electrode placements, or current density can influence cortical excitability and, in turn, performance outcomes (72,140-142). Consequently, the absence of improved explosive squat performance in the present study underscores the multifaceted nature of neuromuscular function, highlighting the need for carefully tailored tDCS protocols that consider exercise-specific demands, participant attributes, and the interplay of central and peripheral factors governing explosive output.

Our findings regarding RPE further underscore the context-dependent nature of a-tDCS effects. While previous work has shown that a-tDCS can reduce perceived effort under conditions involving significant velocity loss and exercises performed until or near failure (27), our results did not replicate this outcome. Given that perceived exertion is closely linked to central rather than peripheral fatigue (143,144), the absence of an RPE reduction may suggest that the training volume and intensity employed in our study were insufficient to elicit the level of central fatigue at which tDCS-induced modulations of effort perception become evident. Indeed, central fatigue typically emerges as exercise approaches volitional failure (145), and studies reporting reduced RPE following a-tDCS have generally involved protocols reaching that threshold (27,58), whereas intermittent, sub-failure tasks often show no such effect (28,72,74). Thus, the mismatch between our protocol and those that have demonstrated diminished perceived exertion may explain the discrepancy in our findings. In this sense, the potential central mechanism underlying a-tDCS's influence on perceived exertion may remain masked unless the exercise stimulus places participants closer to their neuromuscular or volitional limits. Future research incorporating direct measures of neuromuscular fatigue, alongside carefully calibrated training loads, will be critical for clarifying the conditions under which a-tDCS can reliably modulate RPE in velocity-based resistance training contexts.

Despite the careful considerations made in this study, several limitations may have masked the full extent of a-tDCS's potential effects and should be addressed in future research. First, anatomical variability in brain regions such as the M1 and DLPFC (146-148) may have influenced the precision of electrode placement. Employing neuroimaging or neurophysiological techniques (e.g., TMS-based

mapping) could enhance localization accuracy, ensuring more consistent stimulation of the intended cortical targets. Regarding our assessment of explosive performance, the velocity-based training task, with its dual intentions of maximizing both speed and repetition count, may have diluted the clarity of neuromodulatory effects. Future studies could isolate the evaluation of maximal velocity (e.g., in sets limited to three repetitions) to avoid confounding motivational factors related to maximizing total repetitions. Moreover, our reliance on velocity loss thresholds as the sole criterion for repetition validity did not account for subtle technical deviations (e.g., maintaining optimal lumbar curvature), which can affect force production and overall performance (149). Introducing standardized technique criteria and monitoring methods could reduce variability and provide a clearer understanding of how a-tDCS interacts with biomechanical and technical aspects of the exercise.

In conclusion, the application of a-tDCS exhibited the potential to enhance training volume in a velocity-based resistance exercise context, allowing individuals to complete a greater number of repetitions across multiple sets. However, this effect appeared subject to notable inter-individual variability and was not clearly associated with improvements in explosive performance or perceived exertion. To gain a more comprehensive understanding of the neuromodulatory mechanisms at play, future research should incorporate additional assessments—such as measures of voluntary or cortical activation—alongside performance-based outcomes. These combined approaches may help clarify the conditions under which a-tDCS confers ergogenic benefits and guide the development of more individualized and effective neuromodulation strategies in strength and conditioning contexts.

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VII – CONCLUSIONS

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The conclusions of the study are presented below. It should be considered that they are applicable only to people with characteristics similar to those of the present study.

General objective: to investigate the acute effects of anodal transcranial direct current stimulation (a-tDCS) on velocity-based squat training (VBT), focusing on changes in performance and perception under a 15% velocity loss threshold.

General conclusion: the application of a-tDCS appeared to have the potential to enable individuals to perform more reps at a higher velocity, thus completing multiple sets of VBT with a higher total rep across the entire sets. Nevertheless, this effect could interfere with inter-individual variability in the response to the tDCS, and did not seem to be associated with changes in the explosive and perceptual aspects of VBT performance. However, incorporating additional assessment (e.g., voluntary or cortical activation evaluation) into performance-based investigations might be valuable in clarifying the underlying mechanism of the ergogenic effects of tDCS.

Objective 1: to examinate how a-tDCS influences the number of repetitions performed, mean concentric movement velocity, and ratings of perceived exertion (RPE) during VBT in the back squat exercise, applying a 15% velocity loss criterion.

Conclusion 1: a-tDCS applied to M1, but not to DLPFC, appeared to increase the number of repetitions across multiple sets of VBT. Nevertheless, this benefit might be hindered by inter-individual response variability and appears to coincide with no changes in explosive or perceptual performance during VBT.

Objective 2: to determine whether stimulation over different cortical areas (M1 vs. DLPFC) results in distinct effects on explosive performance and subjective effort during VBT.

Conclusion 2: neither a-tDCS to M1 nor DLPFC induced a change in movement velocity and RPE during VBT, even when several tDCS receivers showed an improvement in the number of repetitions. The area-specific effects of tDCS on explosive or perceptual measure do not seem to be obvious during VBT.

Objective 3: to evaluate how a-tDCS-mediated changes in fatigue resistance, explosive strength, and perceived exertion interact, thereby providing insight into the neuromuscular mechanisms underlying any observed improvements in performance.

Conclusion 3: Even though an improvement in the number or repetitions across multiple set of VBT was observed after a-tDCS, the movement velocity and RPE remained unchanged throughout the VBT sets. The correlation between tDCS-induced changes in fatiguability and movement power output, or perceived effort during VBT, did not appear to be obvious.

VIII – LIMITATIONS AND FUTURE WORK

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Despite the potential confounding factors mentioned above, several limitations in our study might also have concealed the effect of tDCS and should be considered in future relevant studies. First, as there might be variability in the location of brain regions (e.g., M1 or DLPFC) among the individuals (146–148), neuroimaging technique (e.g., transcranial magnetic stimulation) could have been employed in our work to precisely locate the placement. Furthermore, during the stimulation, several of our participants noticed different stimulation placements. Therefore, a better blinding approach (e.g., placing the same number of electrode pads on each condition and activating only the ones targeting the area) could have been employed to avoid potential bias. Moreover, the feedback questionaries on the awareness of condition difference could have been included to examine blinding quality. Concerning our performance assessment, the explosive performance (e.g., velocity) was examined through the VBT that could be a dual-intentions exercise as mentioned earlier. Therefore, the maximal velocity in our study could have been examined with a specified repetition (e.g., 3 repetitions) in a separate set to prevent extra intentions (e.g., maximal reps). Moreover, each valid repetition was judged based on the VL amplitude without taking the technique into account, potentially leading to greater variability in performance, as technical factors (e.g., maintaining a slight lordotic curve in lumbar region) could alter squat's force of power (149). Therefore, a standard in technique should be established and included in repetition validation.

IX – REFERENCES

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X – APPENDIXES

X - APPENDIXES

Appendix 1. Informed Consent form

CONSENTIMIENTO INFORMADO

Yo, ..., con DNI:.....

DECLARO:

Haber sido informado/a del estudio y procedimientos de la investigación del Proyecto titulado: El efecto de la estimulación transcraneal por corriente directa sobre el cambio en la velocidad de ejecución durante la sentadilla.

Los investigadores que van a acceder a mis datos personales y a los resultados de las pruebas son: Taichih Chen y Salvador Romero Arenas.

Asimismo, he podido hacer preguntas del estudio, comprendiendo que me presto de forma voluntaria al mismo y que en cualquier momento puedo abandonarlo sin que me suponga perjuicio de ningún tipo.

CONSIENTO:

1.-) Someterme a las siguientes pruebas exploratorias:

Estimulación transcraneal por corriente directa durante 20 minutos, cálculo de una repetición máxima en el ejercicio de sentadilla, control de la velocidad de ejecución durante el ejercicio de sentadilla.

2.-) El uso de los datos obtenidos según lo indicado en el párrafo siguiente:

En cumplimiento del Reglamento (UE) 2016/679 del Parlamento Europeo y del Consejo, de 27 de abril de 2016 y Ley Orgánica 3/2018, de 5 de diciembre, de Protección de Datos Personales y Garantía de los Derechos Digitales, le comunicamos que la información que ha facilitado y la obtenida como consecuencia de las exploraciones a las que se va a someter pasará a formar parte del fichero automatizado INVESALUD, cuyo titular es la FUNDACIÓN UNIVERSITARIA SAN ANTONIO, con la finalidad de INVESTIGACIÓN Y DOCENCIA EN LAS ÁREAS DE CONOCIMIENTO CIENCIAS EXPERIMENTALES Y CIENCIAS DE LA SALUD. Tiene derecho a acceder a esta información y cancelarla o rectificarla, dirigiéndose al domicilio de la entidad, en Avda. de los Jerónimos de Guadalupe 30107 (Murcia). Esta entidad le garantiza la adopción de las medidas oportunas para asegurar el tratamiento confidencial de dichos datos.

En Guadalupe (Murcia) a ..... de ..... de 20

El investigador,

Fdo:..... Fdo:.....

Appendix 2. tDCS screening questionnaire (205).

Es importante que responda todas las preguntas siguientes con sinceridad. Si alguna de las preguntas / términos de este formulario no está clara, o si no está seguro de cómo responderlas, no dude en preguntar al investigador del estudio.		
	Si	**No**
¿Ha tenido convulsiones alguna vez?		
¿Alguna vez ha tenido una lesión en la cabeza que haya provocado la pérdida del conocimiento?		
¿Tiene actualmente un diagnóstico médico de una condición psicológica o neurológica?		
¿Tiene algún metal en la cabeza (fuera de la boca) como metralla o clips quirúrgicos?		
¿Tiene algún dispositivo implantado (por ejemplo, marcapasos cardíaco, estimulador cerebral)?		
¿Tiene una afección cutánea en el cuero cabelludo? (por ejemplo, psoriasis)		
¿Tiene una herida en la cabeza que no ha sanado por completo?		
¿Ha tenido una reacción adversa al tDCS/TMS?		
Para las mujeres participantes: ¿Existe la posibilidad de que esté embarazada?		
¿Está tomando actualmente algún medicamento?		
He comprendido la información que antecede y que me ha sido explicada satisfactoriamente		
Fdo: El voluntario: Nombre: …….. DNI……		