Enhancement of Motor Learning and Corticospinal Excitability: The Role of Electroacupuncture and Motor Training in Healthy Volunteers

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Background: This study embarked on an innovative exploration to elucidate the effects of integrating electroacupuncture (EA) with motor training (MT) on enhancing corticospinal excitability and motor learning. Central to this investigation is the interplay between homeostatic and non-homeostatic metaplasticity processes, providing insights into how these combined interventions may influence neural plasticity and motor skill acquisition.

Material/Methods: The investigation enrolled 20 healthy volunteers, subjecting them to 4 distinct interventions to parse out the individual and combined effects of EA and MT. These interventions were EA alone, MT alone, EA-priming followed by MT, and MT-priming followed by EA. The assessment of changes in primary motor cortex (M1) excitability was conducted through motor-evoked potentials (MEPs), while the grooved pegboard test (GPT) was used to evaluate alterations in motor performance.

Results: The findings revealed that EA and MT independently contributed to enhanced M1 excitability and motor performance. However, the additional priming with EA or MT did not yield further modulation in MEPs amplitudes. Notably, EA-priming was associated with improved GPT completion times, underscoring its potential in facilitating motor learning.

Conclusions: The study underscores that while EA and MT individually augment motor cortex excitability and performance, their synergistic application does not further enhance or inhibit cortical excitability. This points to the involvement of non-homeostatic metaplasticity mechanisms. Nonetheless, EA emerges as a critical tool in preventing M1 overstimulation, thereby continuously fostering motor learning. The findings call for further research into the strategic application of EA, whether in isolation or with MT, within clinical settings to optimize rehabilitation outcomes.

Keywords: Electroacupuncture • Motor Cortex • Motor Skills • Neuronal Plasticity

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Introduction

Cortical neural plasticity, fundamental to the synaptic mechanisms underpinning learning and memory, manifests in distinct long-term potentiation (LTP) and long-term depression (LTD) characteristics, governed by Hebbian-type synapses [1]. Metaplasticity, the neural plasticity’s tunability in response to temporally separated stimuli, comprises either a dynamic capability for modulating plasticity (homeostatic metaplasticity) or a static response unaffected by subsequent experiences (non-homeostatic metaplasticity) [2]. The prevalent Bienenstock-Cooper-Munro model postulates a nonlinear interplay between priming events and subsequent treatments, elucidating the evolution of homeostatic metaplasticity. This model posits that the threshold for eliciting LTP/LTD varies within a sliding physiological spectrum, contingent upon prior synaptic activity [3]. Most synaptic excitation responses in humans align with the Bienenstock-Cooper-Munro model’s homeostatic metaplasticity regulation framework, while a minority exhibit non-homeostatic characteristics [4]. Notably, non-homeostatic metaplasticity has been shown to link alterations in neural excitability in the primary motor cortex (M1) and the enhancement of motor learning [5].

In the clinical context, motor training (MT) is integral to rehabilitation therapies for motor impairments, playing a pivotal role in enhancing voluntary movement [6,7]. MT is known to bolster synaptic strength and motor cortex excitability, leading to improved motor performance [8,9]. Specifically, voluntary movements of the first dorsal interosseus muscle alters motor output, demonstrating the direct impact of physical exercise on motor function [10]. Also, electrical stimulation of the first dorsal interosseus muscle leads to observable changes in motor-evoked potentials (MEPs), pointing to its ability to modulate motor cortex excitability in response to peripheral nerve stimulation [11]. Similarly, electroacupuncture (EA) has been shown to yield positive effects on motor dysfunction by modulating neural plasticity via passive somatosensory stimulation [12,13]. Furthermore, acupuncture at the Hegu acupoint (LI4), a recognized motor point for the first dorsal interosseous muscle, also modifies MEPs, underscoring the capacity of alternative therapies to affect neurophysiological responses [14,15]. Despite observations that combining MT with peripheral nerve stimulation promotes homeostatic metaplasticity, enhancing learning in specific motor tasks, such as finger opposition, the synergistic impact of EA when paired with MT on neural plasticity remains under-explored [16]. A previous study discovered that the integration of EA and MT synergistically elevates motor learning without modifying the excitability of M1[17]. This finding highlights a potential distinct pathway of neural modulation offered by the EA-MT combination, which may differ significantly from the effects observed with peripheral nerve stimulation.

The distinct mechanisms of EA and peripheral nerve stimulation highlight the importance of further examining the unique contributions of EA. Peripheral nerve stimulation predominantly targets group I and II afferent fibers, influencing the motor cortex through the direct stimulation of deep proprioceptors and cutaneous receptors [18-20]. Conversely, EA activates alternative neural pathways, primarily involving A-δ and C fibers, thereby modulating cerebral activity via the spinal-thalamic or spino-limbic-cortical pathways [21-24]. Investigating the specific mechanisms and effects of EA not only deepens our understanding of neural modulation strategies but may also broaden the spectrum of therapeutic options for treating motor dysfunction.

In this study, we investigated EA combined with MT, as well as MT followed by EA, on the excitability of M1 and overall motor function. This included assessing the amplitude of MEPs and performance in the grooved pegboard test (GPT) to ascertain whether homeostatic or non-homeostatic metaplasticity is the underlying mechanism driving the outcomes of these interventions. It is our hope to provide valuable insights into the application of EA and MT in therapeutic approaches for motor dysfunction, potentially offering novel and effective treatment modalities.

Material and Methods

Participants

Twenty healthy, right-handed volunteers aged 18 to 50 years were recruited and assigned to 2 experiments. All participants were evaluated to ensure no contraindications for undergoing transcranial magnetic stimulation (TMS) [25]. This trial was registered in the Chinese Clinical Trial Registry (NO. ChiCTR2000039910), and the register time was November 11, 2020.

The trial received ethical approval from the Ethics Committee of Guangdong Provincial Hospital of Chinese Medicine (Approval No: BF2020-228-01) and was performed in accordance with the Declaration of Helsinki. Before starting the trials, written informed consent was secured from each participant.

Experimental Design

This investigation was an exploratory study conducted with healthy participants, diverging from traditional clinical efficacy trials. Consequently, in estimating the sample size, the study did not adhere to conventional clinical trial methodologies. Instead, it drew inspiration from the design approaches of similar studies, resulting in the recruitment of 20 healthy participants to undergo 4 intervention measures, with each intervention group comprising 20 participants [17,26].
The present study encompassed 2 self-comparative experiments. Experiment 1 involved 20 healthy participants who were subjected to the following 2 groups in a random order. One group received EA while the other received MT. This experiment aimed to determine whether each intervention independently enhanced cortical excitability and improved motor function. Experiment 2 involved 2 additional groups of 20 participants each: 1 group was EA primed with MT, and the other was MT primed with EA. The objective was to investigate the combined effect of EA and MT, to assess whether there was an additive impact on cortical stimulation and motor function. A 1-week washout period was instituted between each intervention group to mitigate potential carryover effects. The detailed experimental procedures and group assignments are depicted in Figure 1.

**Figure 1. Experimental protocol.** The experimental protocol was structured into 2 distinct experiments, each comprising outcome measurement testing phases (T0, T1, T2, and T3) and intervention phases. The outcome measurement testing phases involved acquiring transcranial magnetic stimulation (TMS) outcomes and conducting the grooved pegboard test (GPT) to evaluate the participants’ M1 excitability and motor performance, respectively. In experiment 1, the outcome measurements were taken at 4 different times: at baseline (T0), immediately after the intervention (T1), 30 min after intervention (T2), and 45 min after intervention (T3). The interventions in this experiment were electroacupuncture (EA) for the EA group and motor training (MT) for the MT group. Experiment 2 followed a similar structure for outcome measurements, with the phases conducted at baseline (T0), immediately after the primary (primer) intervention (T1), immediately following the secondary (subsequent) intervention (T2), and 30 min after the secondary intervention (T3). In this experiment, one group received EA as the primer intervention followed by MT (EA-priming group), while the other group received the interventions in the reverse order, with MT as the primer followed by EA (MT-priming group). This figure was produced using Adobe Illustrator 2023 by Adobe.
needles were adjusted subtly to elicit the de qi sensation, a traditional Chinese medicine term indicating effective needle placement. Subsequently, the needles were connected to a Han’s Acupoint Nerve Stimulator (model: HANS-200A), set to a frequency of 2 Hz. The current intensity was adjusted to a level sufficient to induce a minor muscle twitch [27]. This EA treatment session lasted for a duration of 30 min. The identification, codename, and exact locations of the Hegu acupoint were in accordance with the standards set by the World Health Organization [28].

In the MT group, participants were instructed to perform a 25-hole GPT. This task required them to precisely and sequentially insert 25 pegs into corresponding holes on the pegboard using their left hands [29]. The orientation of the GPT apparatus was randomly altered, resulting in 4 distinct training sets. To prevent muscle fatigue, a 2-min rest interval was implemented between each set. The entire duration of this training session was 30 min.

Experiment 2

In the EA-priming group, participants first underwent the EA-priming session, followed by MT. The procedures for both EA and MT were identical to those used in experiment 1.

In the MT-priming group, participants first underwent the MT-priming session, followed by EA.

Outcome Measurements

In both experiments, the time taken to complete the GPT and the outcomes of TMS were recorded. For experiment 1, the measurements were taken at 4 specific time points: before the intervention (T0), immediately after the intervention (T1), 30 min after the intervention (T2), and 45 min after the intervention (T3). Similarly, in experiment 2, measurements were conducted at 4 time points: before the intervention (T0), immediately following the primer intervention (T1), immediately after the subsequent intervention (T2), and 30 min after the subsequent intervention (T3).

Completion Time of GPT

GPT is a well-established tool for assessing fine motor function, effectively measuring motor skill levels [30]. In this test, a stopwatch was used to record the duration taken by participants to complete the task, from the moment they picked up the first peg to the time they inserted the last one. Prior to the actual trial, participants were instructed to practice the GPT multiple times until their completion time stabilized, ensuring consistency in performance on a single day.

TMS Outcomes

This assessment involved measuring various outcomes of TMS, including the amplitude of MEPs, the resting motor threshold, and the latency of MEPs. For each of these parameters, an average value was computed.

TMS Protocols

1) For TMS measurements, participants were seated comfortably in a chair. The central point on the scalp and the right M1 point, located 5 cm to the right of the central point, were marked. Subsequently, a “9-palace” grid, with 1-cm horizontal and vertical spacing, was drawn over the M1 point.

2) Single-pulse TMS was administered using a figure-8 coil with a 70-mm diameter, starting at 60% of the stimulus intensity. The coil was positioned tangentially to the scalp at a 45° angle relative to the anterior-posterior midline, with the handle directed backwards. To maintain consistency in the stimuli, a minimum interval of 3 s was maintained between each pulse.

3) Surface electromyography was conducted on the left first dorsal interosseous muscle. This involved using pairs of silver/silver chloride electrodes connected to the EMG machine. The active electrode was placed over the muscle belly, the reference electrode over the proximal interphalangeal joint of the thumb (short pastern bone joint), and the ground electrode on the ulnar malleolus.

4) The “9-palace” grid on the right M1 was sequentially stimulated using single-pulse TMS. The minimum stimulus intensity required to elicit a valid MEPs amplitude (≥0.05 mV) on 3 or more occasions was recorded as the resting motor threshold. A stimulus intensity of 120% of the resting motor threshold was then used to evoke MEPs at each grid point. The latency of the MEPs was noted as the time from stimulus application to the appearance of the evoked potential.

Randomization Methods

The study used a straightforward randomization strategy. The “Complete Random Design” function of the PEMS 3.1 for Windows software was used to generate random sequences of numbers representing different intervention orders for experiments 1 and 2. These sequences were then transcribed onto cards and enclosed within sealed envelopes. Enrolled participants proceeded to open these envelopes in sequential order as they entered the trial, subsequently receiving the interventions as dictated by the order outlined on the cards.

Statistical Analysis

The data obtained from the experiments were processed using IBM SPSS Statistics software. To ascertain the normal
distribution of the data, the Shapiro-Wilk test was used while the Mauchly test was used to assess sphericity.

In experiment 1, one-way repeated measures ANOVA was conducted to analyze differences in TMS outcomes and GPT completion times between the EA group and MT group. Here, TIME served as the within-subjects factor with 4 levels: baseline (T0), immediately after intervention (T1), 30 min after intervention (T2), and 45 min after intervention (T3).

For experiment 2, two-way repeated measures ANOVA was used to investigate the interaction effects between EA and MT. This analysis used GROUP as the between-subjects factor, with 2 levels (EA-priming group and MT-priming group), and TIME as the within-subjects factor, also encompassing 4 levels: baseline (T0), immediately after primer intervention (T1), immediately after subsequent intervention (T2), and 30 min after subsequent intervention (T3). Bonferroni post hoc tests were conducted to further explore any significant interactions that emerged.

The results from these statistical analyses are presented as mean±SD, and the threshold for statistical significance was set at P<0.05 for all tests, with a 2-sided 5% significance level and a power of 80%.

Results

Baseline Characteristics

All 20 participants (aged 21.05±2.72, M/F=6/14) completed the whole experiment, without reporting adverse events.

Experiment 1

Completion Time of GPT

There was a significant effect of TIME in the EA group (F (2.11, 40.16)=15.73, P<0.001). Compared with T0, the completion time of GPT in T2 and T3 was reduced by 3.37±0.70 s (95% CI: 1.30-5.43, P<0.01) and 4.02±0.85 s (95% CI: 1.52-6.51, P<0.01) respectively, while 1.87±0.43 s (95% CI: 0.60-3.14, P<0.01) in T2 and 2.52±0.61 s (95% CI: 0.72-4.32, P<0.01) in T3 compared with T1. No statistically significant difference was found in T1 compared with T0 and T3 compared with T2.

There was a significant effect of TIME in the MT group (F (1.88, 35.80)=46.00, P<0.01). Compared with T0, the completion time of GPT in T1, T2, and T3 was reduced by 5.10±0.60 s (95% CI: 3.35-6.82, P<0.01), 5.00±0.68 s (95% CI: 3.04-7.03, P<0.01) and 5.00±0.67 s (95% CI: 3.04-6.96, P<0.001) respectively. No statistically significant difference was found among T1, T2, and T3. (Figure 2)
There was a significant effect of TIME in the EA group (F(3, 57)=7.21, P<0.01). Increasing levels of MEPS amplitude were observed by 0.38±0.09 mV (95% CI: 0.12-0.64, P<0.01), 0.64±0.06 mV (95% CI: 0.46-0.82, P<0.01) and 0.72±0.08 mV (95% CI: 0.49-0.95, P<0.01) in T1, T2 and T3, compared with T0, respectively, while 0.34±0.10 mV (95% CI: 0.41-0.65, P<0.05) in T3 compared with T1. No significant difference was found in T3 compared with T2 and T2 compared with T1.

There was a significant effect of TIME in the MT group (F(3, 57)=44.84, P<0.01). Increasing levels of MEPS amplitude were observed by 0.47±0.05 mV (95% CI: 0.31-0.63, P<0.01), 0.53±0.06 mV (95% CI: 0.35-0.71, P<0.01) and 0.59±0.06 mV (95% CI: 0.41-0.76, P<0.01) in T1, T2 and T3 compared with T0, respectively, while no significant difference was found among T1, T2, and T3 (Figure 3).

**Experiment 2**

**Completion Time of GPT**

The completion time of GPT between the EA-priming group and MT-priming group showed a significant TIME*GROUP interaction (F(1.79, 34.03)=17.62, P<0.01) but no significant difference in T0, T2, and T3. Also, the simple effect of TIME in the EA-priming group (F(1.59, 30.25)=11.98, P<0.01) and MT-priming group (F(1.79, 34.03)=20.43, P<0.01) were statistically significant. In the EA-priming group, a significant reduction was observed in T2 (6.27±1.44 s, 95% CI: 2.03-10.51, P<0.01) and T3 (7.52±1.61 s, 95% CI: 2.79-12.25, P<0.01) compared with T0, and in T2 (4.06±1.04 s, 95% CI: 1.00-7.12, P<0.01) and T3 (5.31±1.16 s, 95% CI: 1.89-8.73, P<0.01) compared with T0. No significant differences were observed by 0.47±0.05 mV (95% CI: 0.31-0.63, P<0.01), 0.53±0.06 mV (95% CI: 0.35-0.71, P<0.01) and 0.59±0.06 mV (95% CI: 0.41-0.76, P<0.01) in T1, T2 and T3 compared with T0, respectively. This figure was produced using GraphPad Prism 10 by GraphPad Software.
found in T1 compared with T0 and T3 compared with T2. While in the MT-priming group, a significant reduction was observed in T1 (5.87±1.17 s, 95% CI: 2.42-9.31, P<0.001), T2 (5.38±1.00 s, 95% CI: 2.45-8.31, P<0.01) and T3 (5.63±1.12 s, 95% CI: 2.34-8.92, P<0.01) compared with T0. No statistically significant difference was found among T1, T2, and T3 (Figure 4).

MEPs Amplitude

The MEPS amplitude between the EA-priming group and MT-priming group showed no TIME*GROUP interaction. The main effect of GROUP showed no statistically significant difference (F (1, 19)=3.50, P>0.05), indicating that no significant difference in MEPS amplitude was found in the intervention sequence of EA and MT. A statistically significant difference was observed in the main effect of TIME (F (2, 39.72)=13.36, P<0.01), with asymptotic effects observed in T1 (0.38±0.051 mV, 95% CI: 0.23-0.53, P<0.01), T2 (0.35±0.08 mV, 95% CI: 0.11-0.60, P<0.01), and T3 (0.49±0.09 mV, 95% CI: 0.22-0.76, P<0.01), compared with T0.

In the EA-priming group (F (3, 57)=9.66, P<0.01), the results revealed that T1 (0.39±0.88 mV, 95% CI: 0.14-0.72, P<0.01), T2 (0.40±0.11 mV, 95% CI: 0.09-0.72, P<0.01) and T3 (0.60±0.11 mV, 95% CI: 0.28-0.93, P<0.01) had asymptotic effects on MEPS amplitude, compared with T0. In MT-priming group (F (1.97, 37.45)=6.22, P<0.01), the results of one-way repeated measures ANOVA revealed that T1 (0.36±0.07 mV, 95% CI: 0.16-0.56, P<0.01), T2 (0.30±0.09 mV, 95% CI: 0.31-0.57, P<0.01), and T3 (0.38±0.11 mV, 95% CI: 0.07-0.69, P<0.01) had asymptotic effects on MEPS amplitude compared with T0. No representative changes were observed among T1, T2, and T3 in both groups (Figure 5).

Resting Motor Threshold

In experiment 1, no significant change in the resting motor threshold was observed in either group. In experiment 2, no significant difference between TIME*GROUP interaction and the main effects of TIME and GROUP was observed.

Latency of MEPS

In experiment 1, no significant change in the latency of MEPS was observed in either group. In experiment 2, no significant difference between TIME*GROUP interaction and the main effects of TIME and GROUP was observed.

Discussion

The primary aim of this study was to investigate the combined effects of EA and MT on corticospinal excitation and motor learning, specifically focusing on the EA-priming and MT-priming groups. In the first experiment, we found that both EA and MT, when applied independently, led to a significant increase in MEPS amplitude and a decrease in the time required to complete CPT, compared with baseline measurements. This indicates that each intervention alone can enhance corticospinal excitability and motor performance. The second experiment revealed differing outcomes for the EA-priming and MT-priming groups. In the MT-priming group, participants showed no significant changes in corticospinal excitability or motor performance following EA treatment. However, in the EA-priming group, while EA prior to MT did not significantly alter corticospinal excitability, it did lead to improved motor performance in subsequent tests. These findings imply that the combined approach of EA and MT does not exert clear excitatory or inhibitory effects on cortical plasticity. Instead, the results hint at the potential involvement of non-homeostatic metaplasticity in the observed changes, particularly in the context of motor performance enhancement.

Metaplasticity, a crucial element of neuroplasticity, affects synaptic changes in direction, magnitude, and duration [2]. The characteristics of initial treatments (excitatory or inhibitory) significantly influence corticospinal excitability outcomes [31]. Typically, metaplasticity operates homeostatically, balancing excitatory and inhibitory synaptic transmissions within a flexible physiological range [5].

Key evidence for homeostatic metaplasticity was provided by Hamada et al, who demonstrated bidirectional shifts in motor cortical plasticity following different NIBS protocols [32]. For example, an excitatory NIBS protocol primed with an inhibitory one enhances LTD-like effects, while an inhibitory protocol primed with an excitatory one enhances LTP-like effects. Similar findings have been reported in studies combining various NIBS protocols or integrating MT with NIBS [33-42].

Molecularly, plasticity changes are linked to ionotropic glutamate receptors, particularly NMDA receptors, which regulate intracellular calcium levels [43,44]. Metabotropic glutamate receptors also contribute by modulating the sliding threshold (θm) for plasticity [45]. Additionally, GABA levels influence θm bidirectionally [46]. An alternative hypothesis suggests that θm is related to presynaptic excitatory rates rather than a fixed postsynaptic LTD/LTP threshold [47]. However, the specific mechanism responsible for determining the θm remain unclear.

Overall, homeostatic metaplasticity aligns with the principles of the Bienenstock-Cooper-Munro model. It implies that priming with an excitatory treatment raises the threshold for LTP-like plasticity, thereby reducing the likelihood of LTP-like plasticity and increasing the potential for LTD-like plasticity, and vice versa [48].
Our study’s findings diverge from the typical patterns of homeostatic metaplasticity, as neither priming with EA followed by MT, nor the reverse, significantly affected corticospinal excitability. This suggests the involvement of non-homeostatic metaplasticity in these processes.

Non-homeostatic metaplasticity has been observed in various studies. For instance, MT priming led to a saturation of motor learning, showing temporary resistance to change in response to an excitatory protocol [49]. Additionally, anodal transcranial direct current stimulation (tDCS) prior to MT reduced the duration of the corticospinal silent period, hinting at unexpected corticospinal connections from excitatory priming [50]. This might involve GABAergic neuron targeting by the MT protocol, demonstrating a potential non-homeostatic mechanism. Other studies have implicated glycogen synthase kinase-3 inhibition in non-homeostatic metaplasticity [51]. Furthermore, 5-Hz repetitive transcranial magnetic stimulation (rTMS) followed by excitatory or inhibitory protocols abolished later plasticity effects [52].

Interestingly, certain combinations of interventions have shown prolonged LTP/LTD-like effects, such as excitatory protocols followed by excitatory tDCS [53], or repetitive application of the same protocol [54]. Likely, priming and treatment with inhibitory protocols resulted in extended LTD-like effects [55]. The timing between interventions appears crucial, with shorter intervals favoring non-homeostatic metaplasticity due to the residual effects of the first intervention, whereas longer intervals might lead to homeostatic responses [55]. However, it is also likely that the appropriate time interval for inducing non-homeostatic plasticity varies among different protocols [54].

In our study, particularly in the EA-priming group, a noteworthy observation was the improved motor task performance following the subsequent MT intervention. This suggests that priming with EA might effectively raise the threshold for plasticity saturation, thereby augmenting the potential for synaptic excitability and further learning [56]. This effect is potentially indicative of non-homeostatic metaplasticity, where the balance between synaptic excitation and inhibition is not merely maintained, but actively shifted to optimize learning and recovery [57-59]. The results align with the concept of non-homeostatic plasticity due to the residual effects of the first intervention, whereas longer intervals might lead to homeostatic responses [55]. However, it is also likely that the appropriate time interval for inducing non-homeostatic plasticity varies among different protocols [54].

In clinical applications, especially for stroke rehabilitation, the separate application of EA and MT could offer significant benefits. Each therapy, due to its unique interaction with non-homeostatic metaplasticity, could independently enhance motor cortex excitability and performance [63]. Given the complexities of neural plasticity, further research is needed to refine these therapies, focusing on patient-specific needs and recovery stages [9]. Tailored protocols for EA and MT could significantly improve rehabilitation effectiveness for stroke patients, optimizing the distinct advantages of each therapy while avoiding potential interference from combined use.

**Study Limitations**

The study had several limitations that must be acknowledged. First, the sample size was relatively small and may not have provided enough statistical power to generalize the findings. This limitation is crucial in interpreting the study’s results and understanding its applicability to a wider population. Second, the research only involved healthy volunteers, which means the results may not accurately reflect the responses in individuals with neurological disorders or other health conditions. This restricts the study’s relevance to clinical applications, particularly for patients with specific neuropsychiatric or motor disorders. Third, the use of MEPs as a measure shows considerable inter-individual variability. This variability can affect the consistency of the results and introduce a level of uncertainty in the data interpretation. The individual differences in MEPs can be influenced by numerous factors, such as age, neural anatomy, and physiological state, thereby affecting the reliability of MEPs as a standardized measure across different participants. Lastly, the study lacked a control group that did not receive EA or MT. This makes it challenging to clearly attribute improvements in motor activity and M1 activation to the interventions themselves, rather than placebo effects or natural fluctuations.

**Conclusions**

The study’s findings indicate that the phenomenon of combining MT with EA not significantly altering cortical excitability is a manifestation of non-homeostatic metaplasticity, indicating a nuanced modulation in neural plasticity mechanisms. Furthermore, EA seems to play a role in preventing plasticity saturation within motor learning circuits. These observations underscore the necessity for continued research into the applications of EA, both as a standalone intervention and in conjunction with MT, particularly for the treatment of motor dysfunctions.

**Declaration of Figures’ Authenticity**

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