Combined Use of Electroencephalography and Transcranial Electrical Stimulation: A Systematic Review

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Abstract

Objective. This article presents a systematic review of the combined use of electroencephalography (EEG) and transcranial electrical stimulation (tES) in clinical and healthy populations. The research questions address EEG's role in designing, monitoring, or assessing tES treatments. Furthermore, the review examines whether EEG responses to specific tES configurations are generalizable.

Methods. A systematic search was performed in Google Scholar, PubMed, Scopus, IEEE Xplore, ScienceDirect, and Web of Science using the query: "EEG AND (tDCS OR transcranial direct current stimulation OR tACS OR transcranial alternating current stimulation OR tRNS OR transcranial random noise stimulation OR tPCS OR transcranial pulsed current stimulation)" applied to article titles. Study quality was assessed using the Quality Assessment Tool for Quantitative Studies (QATQS).

Results. Among the 152 included studies, the majority employed EEG after tES to assess the neurophysiological effects of stimulation. Only a limited number utilized EEG to inform the design of stimulation protocols, and none implemented real-time EEG feedback to dynamically adjust tES parameters. A subset of studies integrated both approaches, using EEG data to design the stimulation setup and to evaluate post-stimulation outcomes. Considerable variability was observed in electrode configurations, stimulation parameters, and EEG features analyzed, which hinders cross-study comparisons of electrophysiological effects. In several cases, changes in EEG features did not reach statistical significance, despite standardized stimulation protocols and target populations. Overall, the methodological quality of most studies was rated as weak.

Conclusions. The identification of tES stimulation setups producing generalizable EEG outcomes across studies remains challenging. Contributing factors include incomplete reporting of stimulation parameters, variability in EEG features analyzed, and phenotypic heterogeneity among participants. A promising shift is emerging toward EEG-guided closed-loop protocols, with stimulation settings adapted to the effects observed in each individual, although without real-time adjustment to date.

Significance. This review highlights the critical role of EEG, when employed both before and after tES treatment, in supporting the transition toward a personalized approach to transcranial electrical stimulation.

Keywords: EEG, tES, personalisation, closed-loop,

1. Introduction

Transcranial Electrical Stimulation (tES) is a non-invasive neuromodulation technique able to deliver low-intensity electric currents (<4 mA) to the scalp [51].

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TES is applied by placing two or more electrodes on the target area to be stimulated. The delivered currents interact with the membrane potentials of neuronal cells, inducing multilayer effects on the brain and its related functions [57].

Generally, tES techniques are classified based on two main approaches : (i) Physical, referring to stimulation parameters such as waveform shape, amplitude, electrode montage, and timing of application; (ii) Intended use, including hypothesized mechanisms of action, anatomical targets, and expected outcomes [57, 18].

In recent years, there has been a marked increase in interest in tES as a therapeutic intervention for neurological and psychiatric disorders such as epilepsy, Alzheimer's disease, depression, chronic pain, and so on. These applications are mainly guided by evidence-based recommendations as outlined in the comprehensive guidelines provided by Lefaucheur et al. (2017) [88] or Antal et al. (2017) [9]. The former guideline evaluates the efficacy of the most common tES technique, namely transcranial direct current stimulation (tDCS) in various neurological conditions, offering structured protocols for its clinical use while the latter describes the application of different tES treatment supported by safety, ethical and regulatory guidelines.

However, they propose fixed stimulation setups, disregarding the specific characteristics or pathophysiological profiles of individual patients. This lack of personalization contrasts ongoing research on precision and adaptive medicine, aiming to tailor treatments based on the unique characteristics of each subject.

In this context, electroencephalography (EEG) has been proposed as a promising tool to guide treatment towards more precise and customised approaches. EEG offers a non-invasive assessment of brain activities, allowing the identification of EEG features associated with specific pathologies and the monitoring of changes induced by tES treatment [107]. The integration of EEG with tES techniques could be a significant step in adapting stimulation treatments to individual needs, thus enhancing therapeutic effects.

Several reviews have examined the efficacy of combining EEG and tES. In particular, Thut et al (2017) [147] and Ruffini et al. (2020) [131] have emphasized the potential of EEG biomarkers in predicting treatment outcomes, focusing on general applications and theoretical frameworks rather than systematically analyzing experimental studies evaluating EEG features in response to tES interventions. Beumer et al. (2022) [17] presented a personalized tDCS workflow for epilepsy based on imaging and EEG data, focusing on segmentation, source localization, and montage optimization. Their approach targets a single stimulation type and pathology, without detailing stimulation parameters, limiting insights into clinical efficacy. Similarly, Simula et al. (2022) [139] reviewed tES in epilepsy, considering only tDCS and tACS, without addressing broader stimulation strategies or parameter variability. Moreover, Yang et al. (2021) [161] offered a systematic overview of tES modalities and stimulation parameters in relation to EEG and fNIRS features across multiple disorders, but limited their analysis to a subset of commonly treated pathologies and excluded studies involving healthy subjects or baseline EEG activity.

In this context, the present review examines scientific contributions employing EEG to guide and monitor tES interventions in both clinical and healthy populations, with a focus on adapting stimulation protocols to individual neurophysiological profiles.

In particular, this review is structured around the following Research Questions (RQs):

- (RQ-I): Is transcranial electrical stimulation (tES) guided by EEG data?
- (RQ-II): Is EEG also used to guide tES in real time?
- (RQ-III): Are treatment outcomes assessed through EEG analysis?
- (RQ-IV): Do electroencephalographic outcomes of specific tES protocols generalize across individuals?

2. Methods

2.1. Search Strategy

A flow diagram of the database search process is presented in Figure 1, outlining the phases of identification, screening, eligibility, and inclusion. Articles were collected from Google Scholar, Pubmed, Scopus, IEEEXplore, Science Direct, and Web of Sciences databases by using the query "EEG AND (TDCS OR transcranial direct current stimulation OR TACS OR transcranial alternating current stimulation OR TRNS OR transcranial random noise stimulation OR TPCS OR transcranial pulsed current stimulation)", with the restriction to article title. Only peer-reviewed papers published in journals or conference proceedings and written in English or in Italian were included. No date limitations were applied. Subsequently, the screening process was carried out by combining the results from each source and excluding all duplicates and citations. Titles were

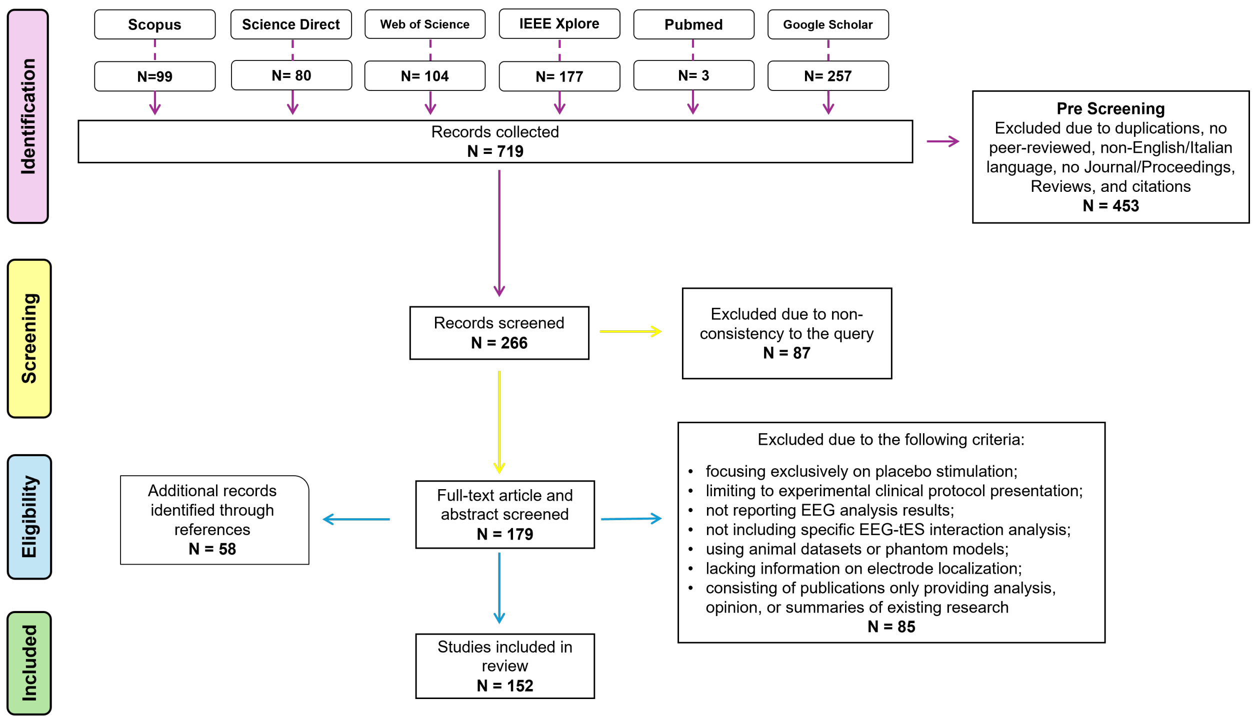

Figure 1: PRISMA-flow of articles selection process

manually screened to exclude papers deemed irrelevant or inconsistent with the query. Finally, during the eligibility phase, all remaining full-text papers and abstracts were screened based on the criteria outlined in the following section. Papers surviving this final phase were included in the review analysis to address the research questions.

2.2. Exclusion criteria

The present study was conducted in accordance with the PRISMA guidelines [89], incorporating the recommendations outlined in Kitchenham's guide [83]. All articles underwent a thorough screening process and were selected based on the following exclusion criteria for studies:

- focusing exclusively on placebo stimulation;
- limiting to experimental clinical protocol presentation;
- not reporting EEG analysis results;
- not including specific EEG-tES interaction analysis;
- using exclusively animals or phantom models for tES treatment analysis;
- lacking information on electrode localization or not reporting at least the anode position;
- being publications exclusively analyzing or commenting on experimental research (reviews, commentaries, editorials).

2.3. Quality Assessment Strategy

The papers were evaluated using the Quality Assessment Tool for Quantitative Studies (QATQS) [58], developed by researchers from Canada's Efficient Public Health Practice Project (EPHPP).

Specifically, the six components of the QATQS were considered: (i) selection bias, (ii) study design, (iii) confounders, (iv) blinding, (v) data collection methods, and (vi) withdrawal and dropouts. These components incorporate the criteria outlined in the Cochrane Collaboration and PRISMA declaration guidelines concerning bias issues [89] [123]. Each component was rated by assigning a quality score ranging from 1 to 3. The individual component ratings were first assessed, and an overall score was then calculated for each article. Papers were classified as strong when no component received a score of 3. A single component with a score of 3 led to a moderate classification. Articles with two or more components scoring 3 were classified as weak.

The initial evaluation was conducted by the second author, adhering to the QATQS protocol guidelines. Subsequently, the third author independently reassessed the papers. In cases of disagreement, all authors participated in discussions to reach a consensus. A QATQS dictionary was used to ensure consistency and standardization of the results. After further discussions, the authors confirmed the absence of discrepancies in outcome interpretation.

3. Results

A comprehensive literature search was conducted through Google Scholar, Scopus, PubMed, ScienceDirect, Web of Science, and IEEE Xplore, resulting in the identification of 719 records. Of these, 453 records were excluded due to duplication or non-compliance with the predefined pre-screening criteria. Subsequently, during the screening phase, an additional 87 articles were excluded due to irrelevance to the established search query. Furthermore, 85 articles were removed during the eligibility assessment as they did not meet the predefined inclusion criteria. Manual screening of reference lists from studies meeting the eligibility criteria ensured more comprehensive coverage of the literature. This approach yielded 58 additional relevant records not captured by the initial database search. Overall, 152 studies were ultimately included in the review analysis. The distribution of the selected articles by year of publication is illustrated in Fig. 2.

The included articles were systematically categorized and analyzed according to the specific research questions they addressed. For each category, a detailed table was constructed, containing information on the clinical use case, waveform type, anode and cathode positioning, the number of tES electrodes employed, the QATQS index, sample size, the EEG features analyzed, and the data analysis methods applied.

The clinical populations examined in the reviewed studies demonstrate significant heterogeneity, encompassing a wide range of neurological disorders, including epilepsy, Alzheimer's disease, Parkinson's disease, stroke, as well as psychiatric and neurodevelopmental disorders. The majority of studies focus on tDCS, with a smaller subset examining tACS, and a clear minority exploring alternative waveform modalities such as transcranial Random Noise Stimulation (tRNS) or transcranial Pulsed Current Stimulation (tPCS). Electrode placements, both anodal and cathodal, vary significantly across studies, frequently targeting the left or right Dorsolateral Prefrontal Cortex (DLPFC) or the epileptogenic focus (EF) in research involving epileptic patients.

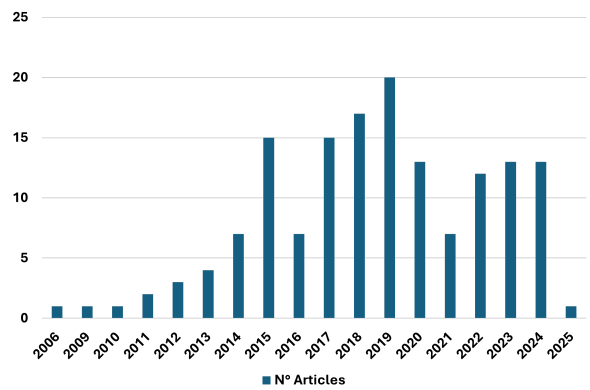

Figure 2: Temporal trend in publication years of the included studies

Quality assessment reveals a predominance of studies rated as "weak," while only a limited number receive "moderate" or "strong" ratings, often associated with relatively small sample sizes. From an analytical perspective, most studies continue to use traditional statistical methods, with only a few employing Machine Learning or Deep Learning approaches for EEG analysis.

3.1. Results for Research Question I (RQ-I)

Tab. 1 presents the articles addressing the first research question, namely whether EEG is used to guide tES treatment. The clinical conditions investigated include epilepsy [59, 133], Alzheimer's disease [5], and chronic tinnitus [39], and a study involving a cohort of healthy subjects [108]. The majority of the studies focus on the use of tDCS, with only one employing tACS. The QATQS evaluation indicates one study as "strong," and the remaining studies categorized as "moderate" or "weak". Conventional statistical approaches are predominantly used, with Machine Learning techniques applied in only one instance for EEG data analysis.

The table illustrates how EEG primarily helps in setting either the electrode placement or the stimulation frequency. For example, parameters such as functional connectivity and the localization of the epileptic focus are used to determine the optimal stimulation site, while Peak Alpha Frequency (PAF) is employed to identify the ideal stimulation frequency for tACS. This approach proposes a methodological shift, wherein the stimulation protocol is customized to the individual's specific neurophysiological characteristics, rather than relying on standardized placements or general models, thus enabling more precise and potentially more effective stimulation.

3.2. Results for Research Question III (RQ-III)

Tab. 2 presents the 131 studies addressing the third research question, namely whether the assessment of the treatment outcomes is based on EEG analysis.

The table reveals a predominant use of spectral power, particularly in alpha (seventeen articles), theta (fourteen articles), and beta bands (twelve articles) as a neurophysiological metric in evaluating the effects of transcranial stimulation on brain activity. Event-Related Potentials (ERPs) were reported in eleven studies, indicating a frequent use of time-locked measures to cognitive or sensory stimuli. Furthermore, Event-Related Desynchronization/Synchronization (ERD/ERS) parameter, observed in six and five studies respectively, reflect a less common focus on the dynamic analysis of induced brain activity. More complex and less frequently employed measures, such as Global/Local Mean Field Power (GMFP/LMFP), connectivity indices (e.g., Phase Locking Value (PLV), Lagged Phase Synchronization (LPS)), and Approximate Entropy (ApEn) were reported in isolated cases.

The table includes 86% of the reviewed studies, highlighting the predominant use of EEG to assess the effects of tES treatments based on standardized setups.

3.3. Results Relevant to Both Research Questions I (RQ-I) and III (RQ-III)

Tab. 3 presents the studies simultaneously meeting the criteria set by the first and third Research Questions, namely whether EEG is used both to design stimulation parameters and to assess tES effects. Regarding the RQ-I, three main EEG feature extraction approaches are generally employed in the design of tES treatments focused on: (i) spectral parameters, such as abnormal patterns of absolute or relative power in theta and alpha bands, (ii) epileptogenic foci in epilepsy-related studies, or (iii) cortical sources localized by exploiting techniques like Low Resolution Brain Electromagnetic Tomography (LORETA). The spectral domain is particularly relevant in the context of tACS, where the stimulation frequency is typically determined based on spectral EEG features. In particular, the Individual Alpha Frequency (IAF), the Individual Theta Frequency (ITF), or the EEG band exhibiting the highest relative power are the mostly used spectral features.

Among the articles presented in Tab. 3, Del Felice et al. [41] and Rocha et al. [128] studies reported the highest QATQS score. The former used the EEG within an intra-subject framework to personalize tACS parameters on each PD patient. For each patient, the relative power in the delta, theta, alpha, and beta bands is compared to thresholds derived from data acquired in a control group, allowing for the identification of cortical regions exhibiting significant deviations. The frequency and localization of tACS are then determined based on the extent of deviation from the normative condition. Specifically, 4 Hz-tACS is applied when fast frequencies predominate, whereas 30 Hz-tACS is used in the presence of higher relative power in slow frequencies. Regarding localization, electrodes are positioned over the scalp region showing the greatest deviation from normative values in the predominant frequency band, with the return electrode placed on the ipsilateral mastoid. Post-treatment EEG acquisition is performed at two different time points, namely right after (T1) and 4-weeks after (T2) tACS treatment. TES effectiveness is assessed by comparing the relative powers of six Region of Interest (three for each emisphere) with respect to the pre-treatment values. Patients exhibiting excessive beta power showed a significant reduction in beta activity following 4 Hz-tACS over the sensorimotor and left parietal areas at T1, and over the right sensorimotor and left frontal areas at T2. In contrast, 30 Hz-tACS produced no significant effects. TThese results suggest effective modulation of pathological high-frequency activity in Parkinson's disease patients through low-frequency tACS. However, there is no evidence supporting the efficacy of high-frequency tACS in patients with predominant low-frequency abnormalities.

Similarly, in Rocha et al. [128] EEG was employed to identify the optimal cortical target for tDCS aimed at enhancing shooting performance. EEG recordings during a target shooting task from skilled shooters showed the highest cortical activation over the right DLPFC. For this reason, this region is then selected for anodal tDCS in unskilled participants. After tDCS, EEG showed increased beta PSD in the left DLPFC and bilateral parietal cortices, and increased low-gamma PSD in the right DLPFC, interpreted as markers of improved visuospatial attention and working memory. Behavioral data confirmed improvements in both accuracy and shot grouping, linking neurophysiological and behavioural changes. The other articles offer valuable insights into the adaptation of TES parameters and their subsequent evaluation using electroencephalographic data , despite receiving low scores according to the QATQS indices. For instance, the study by Akturk et al. (2022) [2] does not account for potential confounding variables nor does it clearly report the level of blinding applied in the experimental protocol. Nevertheless, it is notable for including the largest sample size among the studies included in the table and for proposing an interesting adaptive stimulation setup.In particular, the stimulation frequency is set at ITF -1 Hz, based on the hypothesis of improved memory capacity in healthy participants through *theta–gamma coupling*, obtained by slowing the theta frequency and allowing the integration of multiple gamma cycles within each theta cycle. The result was an increase in resting-state theta coherence around the stimulation site (F3–P3), associated with improved memory performance.

In epilepsy-related studies, only San-Juan et al. (2016) [133] applied tACS treatment, with a stimulation frequency of 3 Hz to match the patient's spike–slow-wave activity and targeting the stimulation site based on the most active epileptiform zone identified through visual EEG inspection. In this case, the intervention led to a clinical worsening, with a 75% increase in seizure frequency. In contrast, the remaining studies employed cathodal tDCS and consistently reported clinical improvement. These studies used EEG for identifying the EF, serving as the basis for selecting the optimal stimulation site for each patient. Overall, cathodal tDCS was associated with a significant reduction in the frequency or amplitude of Epileptiform Discharges (EDs) in the stimulated cortical area [55, 11, 90, 146].

A singular case is presented by Dallmer-Zerbe et al. [36], involving ADHD subjects with reduced amplitude of P300 in Pz. In this context, tACS current is set with respect to both frequency and stimulation timing to promote an increase in P300 amplitude measured during a visual oddball task. Specifically, the stimulation frequency was individually tailored to match each participant's P300 oscillatory frequency, averaged approximately 3 Hz across the subjects. Furthermore, the stimulation timing is synchronized to keep the current in phase with the P300 latency. The results show a significant increase in P300 amplitude and a reduction in errors during the cognitive task. In conclusion, the studies presented in the table reflect an active phase of research on non-real-time closed-loop protocols, highlighting the potential of systems based on direct interaction between EEG and tES. These systems aim to enhance both cognitive performance in healthy individuals and clinical outcomes in patients, developing highly personalized treatment strategies.

3.4. Results for Research Question IV (RQ-IV)

The most recurrent stimulation paradigm reported in the literature was identified to address RQ-IV, with the aim of enhancing the statistical power of cross-study comparison. In this review, a homogeneous stimulation cluster was defined when three parameters coincided: current waveform, anode placement, and cathode placement.

Among the 152 articles included in this review, the most commonly used stimulation protocol involved a direct current waveform, with the anode positioned over F3 and the cathode over Fp2 according to the 10/20 International EEG system. Thirteen studies adopted this configuration, but the EEG features assessed varied considerably, including for example absolute power and Event-Related Synchronization (ERS%), among others. Moreover, for the same EEG feature, different adjacent channels were considered. For this reason, the analysis focused on regional effects rather than specific EEG channels to enable meaningful comparisons across studies.

The comparative analysis summarized in Tab. 4 highlights the absolute power in delta, theta, alpha, and gamma frequency bands as the most commonly assessment parameter adopted to evaluate the effects of tES across different populations. Specifically, absolute gamma power in the frontal area has frequently been assessed, typically showing increased activity following stimulation. For example, Boudewyn et al. (2018) [19] report increases in electrodes FC1, Fz, and FC2, Andrade et al. (2023) [5] identify changes in Fc1 and F8 among responders, and Boudewyn et al. (2020) [20] observe widespread frontal gamma power enhancement, particularly in F3, F7, and FC5. Although all studies consistently focused on the frontal area, the different spatial resolution of EEG limits the precision in localizing neural sources, leading to variability in the specific electrode sites identified. This methodological constraint must be considered when interpreting the apparent consistency across findings, as the regions showing increased gamma power do not fully overlap in terms of electrode selection. In contrast, the P300 component has been explored in only a few studies, including O'Neil-Pirozzi et al. (2017) and Rassovsky et al. (2018) [119, 127], using electrodes placed at Cz and Fz, respectively. These studies reported divergent findings, with only one showing a statistically significant effect.

Another scarcely used parameter is ERS%, analyzed in both Murphy et al. (2023) and Hoy et al. (2015) [112, 70]. In both studies, Event-Related Synchronization (ERS%) was specifically evaluated at the F3 electrode, although within broader analyses. Murphy et al. (2023) assessed ERS% and ERD% across multiple frequency bands including theta, upper alpha, and gamma across all recorded channels, while Hoy et al. (2015) focused more narrowly on gamma ERS% during correct trials at F3. Despite examining ERS% at the same channel location, the two studies reported different sig-

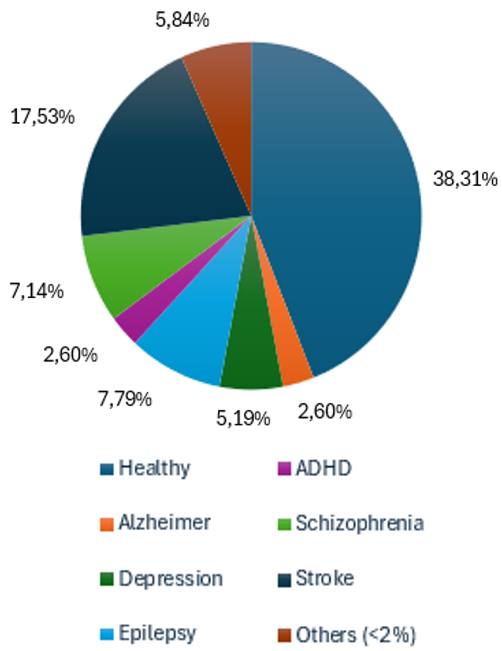

Figure 3: Percentage distribution of participant categories across studies.

nificant effects. Murphy et al. (2023) found an increase in upper alpha ERS% in parieto-occipital regions 5 minutes after stimulation, and later increases in the left frontal and lateral parieto-occipital areas 25 minutes after stimulation. In contrast, Hoy et al. (2015) observed a significant increase in gamma ERS% at F3 40 minutes following 2 mA stimulation, along with a significant decrease in gamma ERS% in the sham condition at the same time point.

These results underscore how far the field remains from identifying generalizable EEG effects of specific tES setups. Progress in this direction may depend on the adoption of standardized stimulation protocols and homogeneous EEG feature extraction methods to evaluate stimulation outcomes. These observations highlight the need for greater standardization in channel selection and feature computation to enable more robust cross-study comparisons. Furthermore, the presence of non-significant findings in some studies emphasizes the need for additional research to clarify the neurophysiological impact of left frontal stimulation [91, 127]. Notably, even when stimulation parameters are fixed, statistically significant outcomes are not consistently observed across participants.

3.5. Distribution of participant categories

Analysis of the sample distribution as shown in Fig. 3 reveals a predominance of studies conducted clinical populations accounting for 61.69% of the total, while the remaining 38.31% involves healthy subjects. In particular, patients affected by stroke represent the second largest group at 17.53%, indicating strong interest in neuromodulation for post-stroke rehabilitation. Epilepsy (7.79%) and depression (7.14%) follow, with promising outcomes in modulating dysfunctional cortical activity. Patients diagnosed with schizophrenia account for 5.19%, while those with Attention-Deficit/Hyperactivity Disorder (ADHD) and Alzheimer's Disease (AD) make up 2.60% each. Lastly, 5.84% falls under the "Others" category, each with an incidence below 2% such as Dementia, Affective disorder, Fibromyalgia or Burnout syndrome. Across different pathological conditions, studies adopt various stimulation setups, based on literature indicating how each disorder affects specific brain areas, often identified through EEG features. In studies involving healthy subjects, stimulation typically targets the prefrontal cortex, reflecting a focus on cognitive processes, particularly memory-related functions.

3.6. Distribution of current waveform

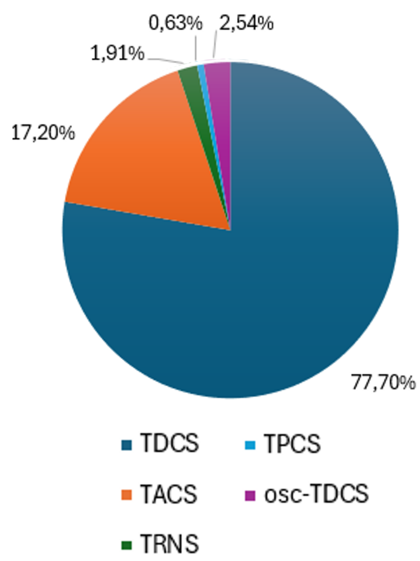

Figure 4: Percentage distribution of current waveforms applied across studies. Expanded acronyms are reported in Tab. 5 in the Appendix Section.

Analysis of the collected data indicates tDCS as the most frequently employed type of stimulation, accounting for approximately 77.70% of cases as reported in

Fig. 4. This predominance results from its relative technical simplicity, allowing use over many years, with development of documents and guidelines supporting application in healthcare, also based on consistent preliminary outcomes across clinical and cognitive domains. The literature has provided substantial evidence supporting its effectiveness in modulating cortical activity, facilitating widespread use in both experimental and therapeutic contexts [134]. In tDCS, anodal stimulation is now well established to increase neuronal excitability through a depolarizing shift in membrane potential, facilitating action potential initiation [116]. In contrast, cathodal stimulation induces hyperpolarization, leading to inhibition of action potential initiation [117]. The tACS emerges as the second most utilized approach, with a prevalence of 17.20%. Despite its lower adoption compared to tDCS, tACS is gaining interest due to its ability to selectively influence neural oscillatory activity at physiologically relevant frequencies. Its more limited application is related to reduced standardization and the increased complexity of protocols, requiring an additional parameter, namely stimulation frequency, to produce appropriate spectral shifts toward greater balance. Less conventional modalities, including tRNS (0.63%), tPCS (1.91%), and osc-tDCS (2.54%), show minimal usage. This low prevalence may results from their still-exploratory nature, limited protocol validation, and a lack of robust evidence of clinical efficacy.

The evident imbalance in usage across techniques suggests a need for broader methodological exploration and increased openness to alternative experimental protocols. The predominance of tDCS reflects its perceived effectiveness, but also limits a comprehensive understanding of the potential benefits offered by alternative approaches such as tPCS and tRNS.

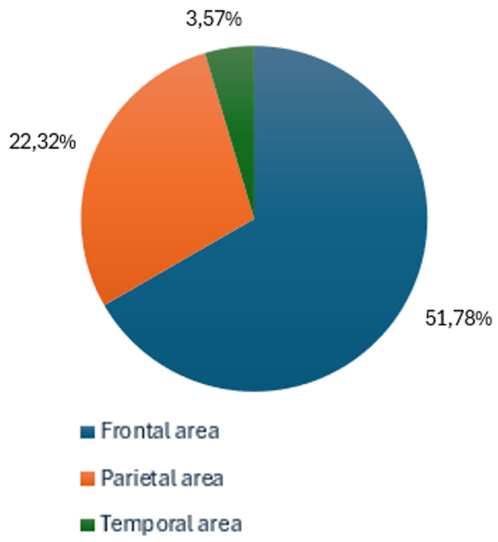

Figure 5: Percentage distribution of anode placement across studies.

3.7. Distribution of anode and cathode position

The literature analysis revealed a marked predominance in the use of specific cortical sites for the placement of the anode electrode during tES. As shown in Fig. 5, more than half of the reviewed studies (51.78%) positioned the anodal electrode over the frontal area. This finding reflects the growing interest in the role of the frontal lobe, particularly the Dorsolateral Prefrontal Cortex (DLPFC), in cognitive functions including attention, working memory, and emotional regulation [156]. The high frequency of stimulation in this area suggests continued preference for targeting the frontal cortex in investigations of cognitive and therapeutic effects of tES. The parietal area was also commonly targeted, each accounting for 22.32% of the studies. Parietal stimulation is often associated with research on spatial attention, multisensory integration, and body awareness [122]. In contrast, the temporal lobe appeared in only 3.57% of the reviewed articles, indicating a significantly lower usage. However, given the temporal lobe's involvement in auditory processing, language, and episodic memory, its relevance may increase as tES applications expand into these cognitive domains [98]. Overall, the distribution of stimulation sites highlights a clear trend toward frontal and motor areas, likely due to stronger empirical support, anatomical accessibility, and established protocols. Nevertheless, a broader exploration of less frequently targeted regions remains crucial to fully characterize the neuromodulatory potential of tES across both clinical and experimental contexts.

The distribution of cathode placements across studies reveals consistent trends in methodological choices for tES protocols. In most cases, as illustrated in Fig. 6, the cathode is positioned over cortical areas, specifically over the frontal region in 62.82% of studies, the parietal region in 25.64%, and the occipital region in 2.56%. The remaining 8.67% of studies opt for extracerebral placements, including sites such as the shoulder or mastoid. In these cases, the cathode serves as a reference electrode. This strategy aims to minimize unintended cortical effects from the reference and to isolate stimulation effects at the active site [118].

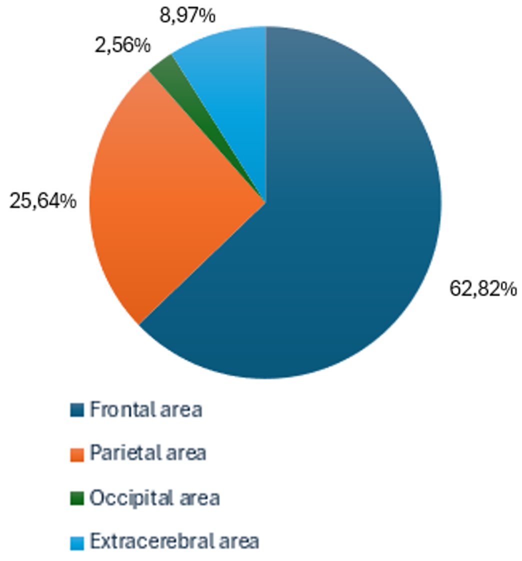

Figure 6: Percentage distribution of cathode placement across studies.

4. Discussion

This systematic review provides an updated synthesis of current practices and challenges in the integration of EEG with transcranial electrical stimulation (tES). Four key findings emerge in response to the predefined research questions. First (RQ-I), only a limited number of studies (3%) employed EEG solely to design stimulation parameters, such as electrode placement or stimulation frequency, using individual neurophysiological markers (e.g., peak alpha frequency [108] or functional connectivity [39]). Second (RQ-II), no studies implemented real-time EEG-guided modulation of stimulation parameters, indicating that latency remains a key unresolved barrier in the development of adaptive closed-loop protocols. The absence of real-time feedback integration may reflect both the technical complexity of synchronizing stimulation with ongoing brain dynamics and the current lack of methodological frameworks for such systems. Third (RQ-III), the majority of studies (86 %) used EEG to assess the effects of tES. Most analyses focused on spectral power changes in alpha, theta, and beta bands, or on event-related potentials (ERPs). Although standardized stimulation configurations are frequently employed, indicating a general convergence in protocol design, the resulting electrophysiological effects remain highly heterogeneous across subjects [30, 6, 69, 53, 25]. Fourth (RQ-IV), studies applying identical stimulation protocols (e.g., tDCS with anode over F3 and cathode over Fp2) reported highly variable EEG outcomes. This variability persisted even when targeting the same EEG features using consistent stimulation settings, and often resulted in non-significant statistical effects inserisci citazioni degli studi confrontabili. Such findings underscore the considerable inter-individual variability of EEG responses to tES.

A non-negligible 10 % of the included articles combined both uses of EEG, relying on pre-treatment EEG recordings to define stimulation parameters and subsequently assessing treatment impact. In most of these studies, the EEG baseline condition was found to predict specific responses to stimulation. Three major themes emerge from the reviewed literature: (i) inter-individual variability, often linked to phenotypic heterogeneity; (ii) inconsistent reporting of stimulation parameters; and (iii) an emerging, yet underdeveloped, transition toward EEG-informed closed-loop approaches. With respect to variability, most tES studies defined stimulation protocols based solely on clinical diagnosis, without integrating neurophysiological features such as resting-state EEG patterns, age, or sex—all of which can influence current distribution and neural response. A critical limitation to the generalization of EEG findings across patients with the same diagnosis is the phenotypic heterogeneity within clinical populations. While this issue has not yet been thoroughly addressed, the work by Dagnino et al. (2023) [35] offers a compelling demonstration. Using unsupervised clustering on resting-state EEG data collected prior to tES in a healthy pediatric sample, the authors identified distinct EEG phenotypes associated with different behavioral responses to tDCS. This supports the idea that EEG phenotyping may account for inter-individual variability and improve prediction of tES outcomes.

Another critical barrier to progress in EEG–tES research lies in the lack of methodological transparency. As shown in Fig. 7, nearly half of the reviewed studies failed to fully report key stimulation parameters, such as electrode material, size, current intensity, and stimulation duration. A more detailed analysis, represented in the bar chart in Fig. 8, nearly half of the reviewed studies failed to fully report key stimulation parameters, such as electrode material, size, current intensity, and stimulation duration. The most frequently omitted information concerned the electrode material and size. These omissions compromise the reproducibility of protocols and the interpretability of neurophysiological results, especially considering that variations in electrode characteristics significantly influence the distribution and magnitude of the electric field. For instance, in three studies, only the anode location was reported without specifying the cathode position [132, 86, 171],

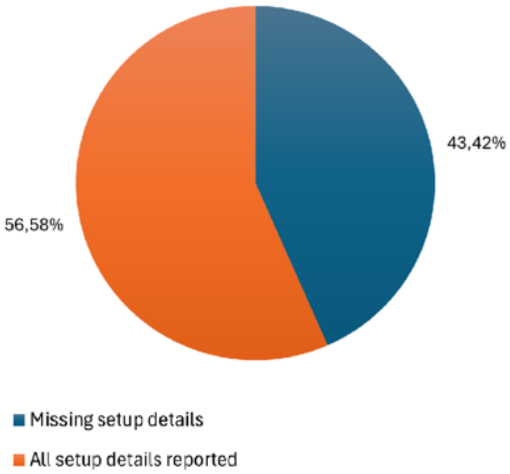

Figure 7: Percentage distribution of articles reporting complete versus incomplete information on tES setup. Incomplete information is considered when at least one of subsequent parameters is missing: stimulation time, current amplitude, electrodes material, or electrodes size.

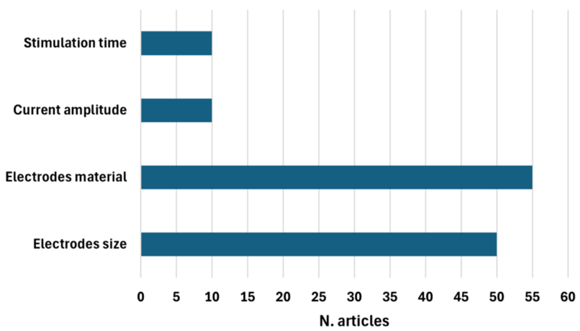

Figure 8: Bar chart showing, for each parameter of the stimulation setup, the number of articles not reporting it.

thus preventing accurate estimation of current density and the overall montage configuration. Incomplete reporting obstructs comparison across studies and limits the generalization of findings. A further limitation of the current literature is the lack of correlation analyses linking neurophysiological changes induced by tES to clinical or cognitive outcomes. In several cases, EEG was recorded pre- and post-intervention, but these changes were not statistically modeled alongside behavioral data. Integrating EEG features, stimulation parameters, and clinical outcomes into unified statistical frameworks could provide a more comprehensive understanding of tES efficacy. In conclusion, the integration of EEG and tES within a precision medicine framework remains in its early stages. To unlock the full therapeutic potential of tES, future studies should prioritize: (i) EEG-based phenotypic stratification, (ii) standardized and transparent reporting of stimulation protocols, and (iii) the implementation of real-time closed-loop EEG–tES systems. Addressing these gaps will be crucial for advancing both the understanding of tES mechanisms of action and its clinical efficacy.

5. Conclusion

This systematic review examines the combined use of EEG and tES in both clinical and healthy populations, focusing on EEG's role in protocol design (RQ-I), monitoring (RQ-II), assessment (RQ-III), and the generalizability of EEG responses to specific tES configurations (RQ-IV). A systematic search across Google Scholar, PubMed, Scopus, IEEE Xplore, ScienceDirect, and Web of Science identified 152 articles and abstracts, later evaluated in relation to the four research questions. Most studies applied EEG post-stimulation to assess neurophysiological effects (RQ-III), while only a few used EEG to guide protocol design (RQ-I), and none implemented real-time feedback for dynamic adjustment of stimulation parameters (RQ-II). Heterogeneity in stimulation setups, EEG features analyzed, and participant characteristics hindered cross-study comparisons and the identification of generalizable EEG responses (RQ-IV). Despite promising theoretical frameworks, the majority of studies rely on standardized electrode placements and neglect inter-individual neurophysiological variability. EEG is mostly employed for post-hoc assessment rather than protocol customization. The lack of consistent reporting on stimulation parameters and poor methodological standardization reduce reproducibility and limit the clinical translation of findings. Future research should prioritize real-time

EEG-tES integration, transparent protocol documentation, and correlation of EEG features with functional outcomes. Such efforts will be essential to advance toward adaptive, phenotype-informed neuromodulation strategies.

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Table 1: Articles addressing the first research questions. Expanded acronyms are reported in Tab. 5 in the Appendix Section.

Articles	Clinical use case	Waveform	A position	C position	N° electrodes	QATQS (sample size)	Feature EEG	Data Analysis method
Fregni et al (2006) [59]	epilepsy	tDCS	silent area	EF	2	moderate (19)	EF	S
De Ridder et al. (2012) [39]	chronic tinnitus	tDCS	(a) right DLPFC, (b) based on functional connectivity	(a) left DLPFC (b) based on functional connectivity	2	weak (675)	theta and gamma functional connectivity	S
San-Juan et al. (2017) [133]	epilepsy	tDCS	silent area	EF	2	weak (28)	EF	S
Marinho Andrade et al. (2023) [5]	AD	tDCS	(a) F5, CP5, F4, (b) F3, P4, P5	contralateral supraorbital	4	strong (70)	PSD in all bands	ML
Mokhtarinejad et al. (2024) [108]	healthy	tACS	Oz	Cz	2	moderate (24)	PAF and PAP	S

Table 2: Articles addressing the third research question. Expanded acronyms are reported in Tab. 5 in the Appendix Section. n.a.: not available.

Articles	Clinical use case	Waveform	A position	C position	N° electrodes	QATQS (sample size)	Feature EEG	Data Analysis method
Palm et al (2009) [120]	MDD	tDCS	left DLPFC (F3)	right supraorbital area	2	weak (1)	absolute and relative power in delta, theta, alpha	S
Zaehle et al (2011) [167]	healthy	tDCS	left DLPFC (F3)	ipsilateral left mastoid	2	weak (16)	ERP and ERSP	S
Zaehle et al (2011) [166]	healthy	tDCS	(a) T7 or Cp5, (b) contralateral supraorbital	(a) contralateral supraorbital, (b) T7 or Cp5	2	weak (14)	ERP	S
Kasashima et al (2012) [78]	stroke	tDCS	M1 of affected hemisphere	opposite supraorbital region	2	weak (6)	ERD	S
Kongthong et al (2013) [84]	healthy	tDCS	right temporal area (T6)	left DLPFC (F3)	2	weak (14)	LPC and ERP	S
Rütsche et al (2013) [132]	healthy	tDCS	left PPC	n.a.	2	weak (26)	ERS/ERD	S
Lazarev et al (2013) [87]	healthy	HD-tDCS	C3	5cm to C3	5	weak (15)	amplitude spectra	S
Mangia et al. (2014) [101]	healthy	tDCS	right PPC	ipsilateral deltoid muscle	2	weak (10)	PSD in theta, alpha, beta and gamma	S
Romero Lauro et al. (2014) [86]	healthy	tDCS	right PPC	left supraorbital area	2	weak (14)	GMFP and LMFP	S
Roy et al. (2014) [129]	healthy	HD-tDCS	between the C3 and CP3	left sensorimotor cortex	5	weak (8)	ERS; ERD	S
Crivelli et al (2014) [32]	healthy	tDCS	right DLPFC	cephalic area	2	weak (22)	ERP	n.a.
von Mengden (2014) [150]	healthy	tACS	F3 and F4	mastoid	2	weak (1)	PSD in theta, alpha and beta	S
Powell et al. (2014) [124]	affective disorder	tDCS	left DLPFC (F3)	F8	2	weak (18)	relative power in alpha and theta; ERP	S
Dominguez et al (2014) [43]	stroke	tDCS	left frontal area	rigth contralateral area	2	weak (1)	absolute power and coherence in delta, theta, alpha, and beta	S
Miller et al. (2015) [106]	healthy	tDCS	AFz	under the chin	2	weak (8)	frontal–midline theta amplitude	S
D'Atri et al. (2015) [37]	healthy	osc-tDCS	(a) Fz, (b) right deltoid muscle	(a) right deltoid muscle (b) Fz	2	weak (20)	EEG oscillatory components	S
Jindal et al (2015) [75]	stroke	tDCS	left DLPFC (F3)	Cz	2	weak (5)	MEP and log-transformed mean-power	S

Sood et al (2015) [142]	stroke	tDCS	DLPFC (F3 and F4)	Cz	3	weak (5)	log-transformed mean-power in (0.5Hz-11.25Hz)	S
Amatachaya et al. (2015) [4]	ASD	tDCS	left DLPFC (F3)	right shoulder	2	weak (24)	Peak alpha frequency	S
Cosmo et al. (2015b) [30]	ADHD	tDCS	left DLPFC (F3)	right DLPFC (F4)	2	strong (60)	functional cortical network	S
Del Felice et al (2015) [41]	epilepsy	so-tDCS	frontal-temporal (F7-T3 or F8-T8)	ipsilateral mastoid	2	weak (12)	spindle frequency and cortical sources	S
Hoy et al (2015) [70]	schizophrenia	tDCS	frontal cortex (F3)	right supraorbital	2	weak (16)	gamma ERS and correlation	S
Dutta et al (2015) [49]	stroke	tDCS	motor cortex (Cz)	left supraorbital notch	2	moderate (4)	log-transformed mean-power in (0.5Hz-11.25Hz)	S
Kasashima-Shindo et al (2015) [78]	stroke	tDCS	primary sensorimotor cortex	controlateral supraorbital area	2	weak (18)	ERD	S
Wu et al (2015) [158]	stroke	tDCS	left posterior peri sylvian	unaffected shoulder	2	weak (12)	ApEn	S
Jindal et al (2015) [76]	stroke	tDCS	motor cortex (Cz)	frontal cortex (F3 or F4)	2	weak (29)	log-transformed mean-power in (0.5Hz-11.25Hz); relative power in all bands	S
Ang et al (2015) [6]	stroke	tDCS	M1 of the ipsilesional hemisphere	contralesional M1	2	moderate (19)	ERD	n.a.
Ulam et al (2015) [148]	TBI	tDCS	left DLPFC (F3)	right supraorbital area (Fp2)	2	strong (26)	relative power in delta, theta, alpha, and beta	S
Impey et al (2015) [72]	healthy	tDCS	left auditory cortex (between C5 and T7)	contralateral forehead	2	strong (12)	ERP (MMN)	S
Cappon et al (2016) [24]	healthy	tACS	Fz (electrode area centroid)	C5 (electrode area centroid)	2	weak (18)	ERS/ERD	S
Caldiroli et al (2016) [23]	healthy	tDCS	right supraorbital region	left DLPFC (F3)	2	weak (30)	ERP	S
Marceglia et al (2016) [102]	AD	tDCS	bilateral temporal-parietal-area	tight deltoid muscle	3	weak (7)	absolute power and coherence in all bands	S
Liu et al. (2016) [91]	epilepsy	tDCS	left DLPFC (F3)	right supraorbital area	2	weak (37)	relative power in alpha and theta	S
Dunn et al (2016) [48]	schizophrenia	tDCS	DLPFC (Fp1 and Fp2)	right upper arm	3	weak (36)	ERP (P300)	S
D'Agata et al (2016) [34]	stroke	tDCS	perilesional M1(C3 or C4)	controlesional M1	2	weak (34)	ERP (P300, N200)	S
Ashikhmin et al. (2017) [10]	healthy	tDCS	over T3 area	over A2 lead	2	weak (10)	relative power in all bands	n.a.
Angulo-Sherman et al. (2017) [7]	healthy	tDCS	(a) in front of C3, (b) between Cz and FC1	inion level (3 cm to the left hemisphere)	2	weak (5)	absolute power in (9-30 Hz)	S
Angulo-Sherman et al. (2017) [8]	healthy	HD-tDCS	(a) C3, (b) Cz	(a) FC1, FC5, CP1, and CP5, (b) FC1, CP1, FC2 and CP2.	5	weak (2)	ERS	S
Grande et al (2017) [65]	healthy	tACS	parietal cortex (P3/P4)	parietal cortex (P4/P3)	2	weak (19)	ERP (N200)	S
Donaldson et al (2017) [45]	healthy	tDCS	right TPJ	right TPJ		weak (n.a.)	ERP (N170, P300)	n.a.
Berger et al (2017) [15]	healthy	tACS	parietal cortex (P3/P4)	parietal cortex (P4/P3)	2	weak (15)	relative power in alpha	S

Cortes et al (2017) [29]	healthy	tDCS	motor cortex (Cz)	Fpz	2	weak (4)	total EEG power in all bands	S
Romero Lauro et al (2017) [86]	healthy	tDCS	rigth PPC	n.a.	n.a.	weak (14)	GMFP and LMFP on mean TEP	S
Ladenbauer et al (2017) [85]	MCI	so-tDCS	prefrontal cortex (F3-F4)	ipsilateral mastoid	3	moderate (16)	absoluter power in (0.5-1 Hz) e fast spindles (12-15 Hz)	S
Impey et al (2017) [73]	schizophrenia	tDCS	left auditory or left frontal cortex	controlateral forehead	2	weak (12)	ERP	S
Naros and Gharabaghi (2017) [113]	stroke	tACS	ipsilesional sensorimotor cortex	controlesionally forehead	2	weak (20)	relative power and ERD in beta	S
Yuan et al (2017) [164]	stroke	tDCS	M1	controlateral shoulder		weak (9)	ApEn	S
O'Neil-Pirozzi et al (2017) [119]	TBI	tDCS	left DLPFC	right supraorbital	2	weak (8)	auditory ERP (P300) and absolute power in alpha and theta	S
Boudewyn et al. (2018) [19]	healthy	tDCS	left DLPFC	right supraorbital	2	weak (20)	absolute power in gamma	S
Kang et al. (2018) [77]	ASD	tDCS	DLPFC	right supraorbital	2	weak (13)	MER	S
Mane et al. (2018) [99]	chronic stroke	tDCS	the ipsilesional M1	contralesional M1	2	weak (19)	PRI, Delta-Alpha Ratio, Theta-Beta Ratio, Theta-Alpha Ratio, Theta-Beta-Alpha Ratio, pdBSI, Rbsi	S
Cucik et al. (2018) [33]	healthy	tDCS	left motor cortex	contralateral eyebrow	2	weak (16)	MSS and SV	S
Friedrich et al (2018) [60]	healthy	tDCS	contralateral orbit parallel to the eyebrow	somatosensory cortex (C3)	2	weak (17)	ERP	S
Mondini et al. (2018) [109]	healthy	tDCS	(a) left motor cortex (C3), (b) right supraorbital (Fp2)	(a) right supraorbital (Fp2), (b) left motor cortex (C3)	2	weak (20)	alpha-ERD and relative power in theta and alpha	S
Holgado et al (2018) [68]	healthy	tDCS	DLPFC	shoulder	2	weak (36)	absolute power in all bands	S
Berger et al (2018) [16]	healthy	tACS	P3	P4	2	weak (24)	relative power in alpha	S
Ferrucci et al (2018) [56]	dementia	tDCS	fronto-temporal (F7-F8)	Right deltoid muscle	3	moderate (13)	absolute power in alpha and beta	S
Shahsavar et al (2018) [138]	depression	tDCS	left DLPFC (F3)	right DLPFC (F4)	2	weak (7)	ERP and alpha average energy	S
Meiron et al. (2018) [104]	epilepsy	HD-tDCS	PO3-P6-AF3-F6-FC4-O1 CP3-C1-FC8-C6-FCz-FC3 O4-F2-CP4 PO4-O2 AF8-C2	C2, TP8,CP8, O3, T8	24	weak (1)	mean number spikers, mean peak amplitude, mean absolute power	S
Rassovsky et al (2018) [127]	schizophrenia	tDCS	DLPFC (F3)	right supraorbital (Fp2)	2	weak (38)	ERP (P300 and N170)	S
Hordacre et al (2018) [69]	stroke	tDCS	M1	controlateral orbit	2	weak (10)	connectvity in delta, theta, alpha, beta, and gamma	S
Nicolo et al (2018) [114]	stroke	tDCS	ipsilesional supraorbital region	controlesionally M1	2	moderate (41)	effective and functional connectivity	S

Straudi et al. (2019) [144]	MCS	tDCS	M1	M1	n.a.	weak (10)	parietal site EEG upper alpha bandwidth	S
D'Atri et al. (2019) [50]	healthy	tACS	right fronto-temporal area	left fronto-temporal area	2	moderate (20)	relative power in all bands	S
Dondè et al. (2019) [46]	healthy	tRNS	right-DLPFC (F4)	left-DLPFC (F3)	2	Strong (19)	beta/alfa power ratio	S
Donaldson et al. (2019) [44]	healthy	tDCS	rTPJ	rTPJ	n.a.	weak (n.a.)	ERP (P300)	n.a.
Dowsett et al (2019) [47]	healthy	tACS	Cz	O2	2	weak (30)	SSVEP	S
Bueno-Lopez et al (2019) [22]	healthy	so-tDCS	prefrontal positions (F3-F4)	ipsilateral mastoids (M1-M2)	4	moderate (23)	relative power in all bands	S
Handiru et al (2019) [67]	stroke	tDCS	ipsilesional M1	controlesional M1	n.a.	weak (19)	beta coherence	S
Willms et al (2019) [157]	healthy	tDCS	left DLPFC	right DLPFC	n.a.	weak (n.a.)	power in alpha	S
Mastakouri et al (2019) [103]	healthy	HD-tACS	M1 (C3)	Cz, F3, T7, and P3	5	moderate (19)	absolute power in beta	S
Emonson et al (2019) [53]	MCI	tDCS	DLPFC (F3)	controlateral supraorbital (Fp2)	2	weak (49)	ERP and TEP	S
Cespòn et al (2019) [26]	AD	tDCS	left DLPFC (F3)	right shoulder	2	moderate (26)	ERP, absolute power in theta, alpha and beta	S
Alexander et al. (2019) [3]	MDD	tACS	left/right DLPFC (F3/F4)	Cz	2	strong (32)	absolute power in alpha	S
Meiron et al. (2019) [105]	epilepsy	HD-tDCS	frontal-parietal cortex (AF8, F2, C2, PO4)	C6	5	weak (1)	relative power in theta, alpha, beta; delta-ERD	S
Ahn et al (2019) [1]	schizophrenia	tACS and tDCS	prefrontal cortex (between F3 and Fp1)	TPJ (between T3 and P3)	2	moderate (22)	alpha oscillations, PSD, functional connectivity	S
Singh et al (2019) [140]	schizophrenia	tPCS	cerebellar vermis	right shoulder	2	weak (9)	relative power in delta and theta	S
Schoellmann et al (2019) [135]	PD	tDCS	left sensorimotor (C3)	right frontal area (FP2)	2	moderate (21)	relative power and coherence in all bands	S
Mane et al (2019) [100]	stroke	tDCS	ipsilesional M1	controlesionally M1	2	weak (19)	PSD and relative power in delta, theta, alpha, beta; PRI; rBSI	S
Bao et al (2019) [13]	stroke	HD-tDCS	ipsilesional M1 (C3)	frontal-parietal cortez (F1,F5,P1,P5)	5	weak (30)	coherence and PSD in alpha, beta, and gamma	S
Luna et al. (2020) [95]	healthy	HD-tDCS	(a) right PPC, (b) right DLPFC	(a) right PPC, (b) right DLPFC	5	moderate (92)	absolute and relative power in alpha	S
El-Hagrassy (2020) [52]	healthy	tDCS	left DLPFC	(a) right shoulder, (b) right DLPFC	2	weak (24)	PSD in delta, theta, alpha, beta, and gamma	S
de Melo et al. (2020) [38]	fibromyalgia	tDCS	left M1 (C3)	right supraorbital	2	strong (31)	absolute power in (0.5-30 Hz)	S
Sergiou et al (2020) [136]	substance dependence	HD-tDCS	Fpz	AF3, AF4, F3, Fz and F4	6	weak (50)	LPP	S
Pross et al (2020) [125]	schizophrenia	tDCS	DLPFC	DLPFC	n.a.	weak (40)	alpha-activity	n.a.
Gangemi et al (2020) [61]	AD	tDCS	left frontotemporal lobe (F7-T3)	right frontal lobe (Fp2)	2	moderate (26)	alpha/beta/theta rhythm	S
Nikolin et al (2020) [115]	depression	tDCS	left DLPFC (F3)	right shoulder	2	weak (20)	PSD in alpha and theta; ERP	S

Breitling et al (2020) [21]	ADHD	tDCS/ HD-tDCS	right inferior frontal gyrus (F8)	controlateral supra-orbital	2 (5 for HD)	weak (15)	ERP (N-200 and P-300)	S
Boudewyn et al (2020) [20]	schizophrenia	tDCS	left DLPFC (F3)	right supraorbital (Fp2)	2	moderate (37)	relative power in gamma	S
Jahshan et al (2020) [74]	schizophrenia	tDCS	central occipital cortex	right shoulder	n.a.	weak (27)	VEP	n.a.
Zhang et al (2020) [170]	TBI	tDCS	left DLPFC (F3)	neck / F4	2	weak (10)	ApEn; C-ApEn	S
Grasso et al. (2021) [66]	healthy	tDCS	left PPC	upper part of the right arm	2	moderate (32)	ERP and TEP	S
Hasballah (2021) [168]	post-stroke	tDCS	left-DLPFC (F3)	right-DLPFC (F4)	2	weak (23)	absolute and relative power, delta-theta-alpha-beta and delta-alpha ratio	S
Ghin et al (2021) [63]	healthy	hf-tRNS	PO3/P04	PO4/PO3	2	weak (16)	PSD; VEP	S
Mostafavi et al (2021) [111]	OUD	tDCS	(a) left DLPFC (F3), (b) right DLPFC (F4)	(a) right DLPFC (F4), (b) left DLPFC (F3)	2	moderate (30)	absolute power, amplitude and coherence in all bands	S
Mai et al (2021) [96]	healthy	tDCS	left/right auditory cortex (T7/T8)	contralateral forehead	2	strong (90)	EFR	S
Wang et al (2021) [151]	stroke	tDCS	(a)/(c) ipsilesional M1 (C3 or C4), (b) lateral supraorbital	(a) lateral supraorbital, (b)/(c) controlateral M1	2	weak (19)	PSD and relative power in delta, theta, alpha and beta	S
Hu et al (2021) [71]	healthy	tACS	DLPFC (F3/F4)	DLPFC (F4/F3)	2	weak (44)	absolute power in alpha; ERP	S
Ghafoor et al. (2022) [62]	healthy	HD-tACS/HD-tDCS	FpZ	left and right PFC	5	weak (15)	relative power in alpha and beta	S
Wang et al. (2022) [152]	ischemic stroke	tDCS	(a)/(c) ipsilesional M1, (b) lateral supraorbital	(a) lateral supraorbital, (b)/(c) controlateral M1	2	moderate (32)	PSD and relative power in delta, theta, alpha, and beta	S
Liu et al. (2022) [92]	UWS	tDCS	(a) prefrontal area, (b) left FTPC, (c) right FTPC, (d) left DLPFC	(a) neck, (b)/(c) back of the opposite shoulder d. F4	2	strong (85)	c-ApEn	S
Kim et al. (2022) [80]	PTSD	tDCS	left DLPFC (F3)	right-DLPFC (F4)	2	weak (48)	PSD in delta, theta, alpha, and beta	S+ML
Westwood et al. (2022) [155]	ADHD	tDCS	F8	right supraorbital (Fp1)	2	moderate (29)	PSD in alpha, theta and beta	S
Maimon et al (2022) [97]	DOC	tDCS	left DLPFC (F3)	right supraorbital (Fp2)	2	weak (6)	MMN, ERP, VC9 activity; theta relative power	S+ML
Ayub et al (2022) [12]	healthy	tDCS	Cz	Cp1	2	weak (10)	ERDs	S
Palmisano et al (2022) [121]	AD	tACS	6 locations covering 4 lobes in both hemispheres	6 locations covering 4 lobes in both hemispheres	n.a.	weak (15)	spectral power in all bands; theta, alpha and beta activity	S
Cheng et al (2022) [28]	OCD	tDCS	AF8, AF4, AFZ, and FPZ	right supraorbital (Fp2)	5	weak (51)	TEP (N45, P60, N100, and P200)	S
Wang et al (2022) [152]	stroke	tDCS	left DLPFC (F3)	right DLPFC (Fp2)	2	moderate (4)	relative power in delta, theta, alpha, and beta	S
de Souza Moura et al (2022) [40]	head and neck cancer	tDCS	F4	C5	2	weak (2)	PLI; PSD at 4/8/16/24 Hz	S

Mosayebi-Samani et al. (2023) [110]	healthy	tDCS	(a) C3, (b) F3	contralateral supraorbital	2	moderate (18)	TEP; TMS-evoked oscillations; MEP	S
Yeh et al. (2023) [162]	schizophrenia	tACS	(a) F1, F5, AF3, FC3, (b) P1, P5, CP3, PO3	(a) CPz, (b) FCz	10	strong (35)	LPS, connectivity	S
Dagnino et al (2023) [35]	healthy	tDCS	(a) left DLPFC (F3, AF3, AF7), (b) frontal gyrus (FC6, F8)	(a) Fp2 and T7, (b) Fp2, T8 and C6	5	strong (56)	relative power in all bands	S+ML
Sergiou et al (2023) [137]	substance dependence	HD-tDCS	Fpz	vmPF (AF3, AF4, F3, Fz and F4)	6	moderate (50)	beta activity; alpha and beta synchronicity	S
Kim et al (2023) [81]	PTSD	tDCS	F3	F4	2	weak (48)	EEG spectrogram	DL
Roy et al (2023) [130]	healthy	tDCS	DLPFC	DLPFC	n.a.	weak (72)	ERP	S
Liu et al (2023) [94]	stroke	tDCS	ipsilesional M1	ipsilesional M1	2	weak (15)	PSD in all bands	S
Fabio et al (2023) [54]	PD	tDCS	left DLPFC (F7)	right supraorbital area	2	weak (30)	PDS and absolute power in alpha and beta; ERP (P300 latency)	S
Chan et al (2023) [27]	ASD	tDCS	right DLPFC (Fp2)	left DLPFC (F3)	2	moderate (60)	theta E/I balance	S
Murphy et al (2023) [112]	MDD	tDCS/tRNS	left DLPFC (F3)	right supraorbital	2	moderate (49)	ERS/ERD	S
Wang et al (2023) [153]	healthy	tACS	left DLPFC (F3)	right DLPFC (F4)	2	moderate (40)	brain activity trajectories	S
Wang et al. (2024) [154]	DoC	HD-tDCS	Pz	parietal cortex	5	weak (8)	PSD and relative power in all bands; spectral entropy, spectral exponent	S
Tarantino et al. (2024) [145]	DoC	tDCS	left DLPFC	right supraorbital	2	weak (19)	alpha/theta power ratio	S
Vimolratana et al. (2024) [149]	stroke	tDCS	lesioned hemisphere (C3/C4)	controlateral supraorbital	2	moderate (34)	absolute power in delta, theta, alpha, and beta	S
Singh et al. (2024) [141]	MDD	tDCS	left DLPFC (F3)	left FTPC and FCPC	5		PSD in all bands; functional connectivity	S
Couto et al (2024) [31]	comorbid anxiety-depression	tDCS	(a) rVLPFC (F6), (b) vmPFC and anterior cingulate cortex (AF3)	(a) contralateral (Fp1), (b) contralateral mastoid (TP1)	2	weak (20)	absolute power in all bands; functional connectivity; alpha activity	S
Liu et al. (2024) [93]	stroke	tDCS	ipsilesional M1 (C3/C4)	contralesional site (FP1/FP2)	2	moderate (36)	absolute power in alpha	S
Wynn et al (2024) [159]	healthy	tACS	AF4 and P5	Cz	3	weak (54)	absolute power and peak frequency in theta and gamma	S
Yeh et al. (2024) [163]	schizophrenia	tDCS	left DLPFC (F3)	Fp1, Fz, C3 and F7	5	moderate (59)	delta DMN connectivity and LPS	S
Zhou et al (2024) [171]	healthy	tDCS	motor cortex	n.a.	2	weak (29)	relative power in alpha and beta	S
Zhang et al (2024) [169]	healthy	tDCS	Oz	Cz	2	weak (13)	SSVEP	S
Xiao et al (2025) [160]	bipolar depression	tDCS	left DLPFC (F3)	right DLPFC (F4)	2	weak (20)	absolute power in all bands; PLV	DL

Table 3: Articles addressing the first and third research questions. Expanded acronyms are reported in Tab. 5 in the Appendix Section. n.a.: not available.

Articles	Clinical use case	Waveform	A position	C position	N° electrodes	QATQS (sample size)	Feature EEG (RQ1)	Feature EEG (RQ3)	Data Analysis method
Zaehle et al (2010) [165]	healthy	tACS	PO9/PO10	PO10/PO9	2	weak (20)	IAF	absolute power in alpha	S
Faria et al (2012) [55]	epilepsy	tDCS	CPF (FP1, FPz, FP2)	CP6 or CP5	4	weak (17)	EF	average number of EDs	S
Auvichayapat et al (2013) [11]	epilepsy	tDCS	controlateral shoulder area	EF	2	weak (36)	EF	average number of EDs	S
San-Juan et al. (2016) [133]	epilepsy	tACS	frontal cortex (Fp1/Fp2)	frontal cortex (Fp2/Fp1)	2	weak (1)	EF	average number of spike-low, poli spiker-slow, slow rhythmic waves	n.a.
Stecher et al. (2017) [143]	healthy	tACS	Cz	Oz	2	weak (33)	IAF	alpha absolute power	S
Khayyer et al. (2018) [79]	MDD	tDCS	left/right DLPFC (F3/F4)	Cz	2	weak (9)	LORETA EEG source localization	absolute power in alpha	S
Lin et al. (2018) [90]	epilepsy	tDCS	controlateral shoulder	EF	2	weak (9)	EF	PLI in delta, theta, alpha and beta	n.a.
Tecchio et al (2018) [146]	epilepsy	tDCS	opposite homologous	EF	2	weak (6)	EF	functional connectivity	S
P.-De Koninck et al (2019) [14]	healthy	tACS	(a) PO7/PO8, (b) F3/F4	(a) PO8/PO7, (b) F4/F3	2	weak (12)	IAF or ITF	absolute power in alpha	S
Del Felice et al. (2019) [42]	PD	tACS+ tRNS	based on power spectral difference	ipsilateral mastoid	2	moderate (15)	relative power difference	relative power in delta, theta, alpha, and beta	S
Rocha et al (2020) [128]	healthy	tDCS	contralateral supraorbital area	right DLPFC (F4)	2	moderate (60)	EEG activity	PSD in beta and gamma	S
Dallmer-Zerbe et al. (2020) [36]	ADHD	tACS	motor-parietal cortex (C3, C4, CP3, CP4, P3, P4)	temporal-parietal (T7-T8-TP7-TP8-P7-P8)	12	weak (18)	P300 and ERSP maximum	ERP (P-300)	S
Aktürk et al. (2022) [2]	healthy	tACS	frontal region (F3)	parietal region (P3)	2	weak (46)	ITF	theta absolute power and theta connectivity-based ERP	S
Radecke et al (2023) [126]	healthy	tACS	parietal cortex	parietal cortex	6	weak (22)	maximal lateralization of alpha power	ERP	S
Gòral-Pòlrola et al (2024) [64]	burnout syndrome	tDCS	left frontal cortex (F7)	n.a.	2	weak (1)	alpha rythm	EEG spectra and ERP	S
Kim et al (2024) [82]	healthy	tACS	DLPFC (F5 or Fpz)	DLPFC (F7, F3, AF7 or Afz, Fz, FCz)	4	weak (24)	ITF	absolute power in theta	S

Table 4: Articles including the same type of stimulation (tDCS), anode placed on F3 and cathode placed on Fp2 are reported. Expanded acronyms are reported in Tab. 5 in the Appendix Section.

Author	Sample type	EEG features	Results
Boudewyn et al. (2018) [19]	20 healthy, 17 female, mean age 21, range (18-30 y.o.)	Absolute power in low gamma and high gamma frequency bands in Frontal (FC1, Fz, FC2), Central (CP1, Cz, CP2) and Posterior (PO3, Pz, PO4) regions.	Increased frontal gamma power for B cues
Andrade et al. (2023) [5]	70 AD, sex and age not reported	Absolute power of the delta, theta, alpha, beta, and gamma frequency bands in Fc1, Fc2, Fc5, Fc6, Fp1, Fp2, F3, F4, F7, F8, FT9, FT10, C3, C4, CP1, CP2, CP5, CP6, T7, T8, P3, P4, P7, P8, O1, and O2.	Increased absolute power in Fc1, F8, CP5, Oz e F7 in responder patients

Boudewyn et al. (2020) [20]	37 schizophrenia, 12 female, mean age 22.76 ± 3.65, range (18 - 30 y.o.)	Absolute power in gamma band in Left Frontal (F3, F7, FC5), Mid Frontal (AF4, AF3, Fz), Right Frontal (F4, F8, FC6), Central (FC2, Cz, CP2, FC1, CP1), Left Posterior (P3, CP5, P7), Mid Posterior (O1, Oz, O2), and Right Posterior (P4, CP6, P8) regions.	Increased absolute gamma power compared to the sham condition in all clusters except Left Posterior and Mid Posterior when sham is done before active stimulation.
Liu et al. (2016) [91]	37 epilepsy, sex not reported, range (18 - 70 y.o.)	Averaged absolute power values in delta, theta, low alfa, high alfa, beta and low gamma across frontocentral (Fp1, Fp2, F3, F4, C3, C4), left temporal (F7, T3, T5, A1), right temporal (F8, T4, T6, A2), and occipital (O1, O2) regions.	No statistically significant results
Palm et al. (2009) [120]	1 66-year-old female MD (major depression) patient	Averaged absolute and relative power in delta, theta, alpha and beta for frontal (Fp1, Fp2, F3, FC1, F4, FC2, FC5, F7, F8, FC6, Fz), central (T3, T4, CP5, CP6, C3, C4, Cz) and posterior (T5, T6, P3, P4, Pz, O1, O2)	Decreased absolute power in delta band in frontal area, decreased absolute power in alfa band in frontal and central areas. Decreased relative power in delta and theta bands in frontal area and in alfa band in frontal and central areas post - tES treatment
Wang et al. (2022) [152]	24 PSEI (post-stroke executive impairment), 7 female, mean age 54.08 ± 10.53	Averaged absolute power in delta, theta, alpha, and beta in left prefrontal (Fp1, AF3, F3, and F7), left central (C3), left occipital (O1), right prefrontal (Fp2, AF4, F4, and F8), right central (C4), right occipital (O2), prefrontal (Fp1, AF3, F3, F7, Fp2, AF4, F4, F8, and Fz), central (C3, C4, and Cz), and occipital (O1, O2, and Oz) regions.	Higher theta band absolute power after stimulation in the left central region than before stimulation
Maimon et al. (2022) [97]	6 DOC, 1 female, range (24 - 81 y.o.)	Frontal MMN N1 peak amplitudes, frontal theta VC9 biomarker activity and mean prefrontal theta-band power.	2 patients with significant difference between standard tone N1-amplitudes and deviant tone N1-amplitudes before Tdcs treatment, and three patients exhibited a significant MMN post-tDCS treatment. Absolute frontal theta power increased in 4 patients, decreased in 1. VC9 activity significantly increased in 3 patients, decreased in 1
Emonson et al. (2019) [53]	20 younger adults, 10 female (mean age 24.50 ± 4.48); 20 older adults, 11 female (mean age 65.47 ± 5.62); 9 MCI, 4 female (mean age 72.11 ± 5.75)	For TEP at rest: P30/N40, P60, N100, and P200 in F1, FZ, F2. ERP analysis for 2-back task: N100, P150, N250, and P300 in posterior and frontal regions.	In the young, P30 and P60 reduced post-tES amplitude, N250 increased post-tES amplitude; in the elderly, N250 increased post-tES amplitude
Rassovsky et al. (2018) [127]	38 schizophrenia, 32% females, mean age 42.7 ± 8.57, range (23 – 55 y.o.)	MMN in Fz using a passive attention auditory duration deviant paradigm, P300 in Pz using an active attention auditory oddball paradigm. N170 in P7 and P8 during another task.	No statistically significant results
Murphy et al. (2023) [112]	49 MDD, 29 females, mean age = 28.46 ± 6.12, range (18 - 65 y.o.)	Event-related synchronisation (ERS%) and Event-related desynchronisation (ERD%) within the theta, upper alpha, and gamma frequency bands in all acquisition channels.	Increase in upper alpha ERS% on parieto-occipital regions 5 min post tES and on left frontal and lateral parieto-occipital regions 25 min post tES. tDCS > Sham in both conditions.
Hoy et al. (2015) [70]	18 schizophrenia, 6 females, mean age 42.17 ± 11.04	ERS% for correct trials only in the gamma band during the active interval and the reference interval in F3.	Significant ERS% increase in gamma at 40 min post-stimulation 2 mA for tES. Significant decrease in gamma at 40 min post-stimulation for sham tES.
O'Neil-Pirozzi et al. (2017) [119]	4 Neurotypical, one male, mean age = 51.6, range (44 - 59 y.o.); 4 TBI, two males, mean age = 43, range (35 - 53 y.o.)	P300 in Cz, absolute power in theta and alpha bands from each electrode in frontal, parietal, and occipital areas.	Increased P300 amplitude after anodal stimulation compared to sham only in TBI.

Ulam et al. (2015) [148]	26 TBI, 4 females, mean age = 33.52 ± 12.25	Relative power Z scores in delta, theta, alpha, beta, high beta at 6 different time points in F3 (anode) and Fp2 (cathode).	Active tDCS group had greater delta at Fp2 than the sham group for EEG1 and EEG 2. Greater delta at F3 for the active tDCS group compared to the sham group, at EEG3. Greater total delta for the active tDCS group at EEG 2 and 3 compared to the sham group in Fp2. a significant decrease in theta between EEG 2 and EEG3 for the active tDCS group in F3. a significant decrease in delta between EEG 1 and 6 for the active tDCS group in F3 and Fp2. a significant increase in alpha from EEG 1 to 6 for the active tDCS group in F3 and in Fp2. a significant difference was identified between the active tDCS and sham groups at EEG 6, with the active group having greater alpha relative power than the shams in F3 and Fp2.

Appendix

Table 5: Expanded acronyms for Clinical Use Case, Stimulation electrode position, EEG Features, Stimulation Types, and Data Analysis Methods

Abbreviation	Meaning
Clinical Case	
MDD	Major Depressive Disorder
ADHD	Attention Deficit Hyperactivity Disorder
ASD	Autism Spectrum Disorder
PD	Parkinson's Disease
AD	Alzheimer's Disease
OUD	Opioid Use Disorder
DOC / DoC	Disorder of Consciousness
TBI	Traumatic Brain Injury
PTSD	Post-Traumatic Stress Disorder
MCS	Minimally Conscious State
MCI	Mild Cognitive Impairment
OCD	Obsessive Compulsive Disorder
UWS	Unresponsive Wakefulness Syndrome
Stimulation electrode position	
DLPFC	Dorsolateral Prefrontal Cortex
PFC	Prefrontal Cortex
rVLPFC	ventrolateral Prefrontal cortex
vmPFC	ventromedial Prefrontal cortex
M1	Primary Motor Cortex
PPC	Posterior Parietal Cortex
TPJ	Temporo-Parietal Junction
EF	Epileptogenic Focus
EEG feature	
ERP	Event-Related Potentials
ERSP	Event-Related Spectral Perturbations
ERS / ERD	Event-Related Synchronization / Desynchronization
PSD	Power Spectral Density
GMFP / LMFP	Global / Local Mean Field Power
ApEn / C-ApEn	Approximate Entropy / Cross Approximate Entropy
TEP	Transcranial Evoked Potentials
MEP	Motor Evoked Potentials
SSVEP/VEP	Steady-State Visual Evoked Potential/Visual Evoked Potentials
MMN	Mismatch Negativity
EFR	Envelope Following Response
PLV	Phase-Locking Value
PAF	Peak Alpha Frequency
LPC	Late Positive Component
LP	Late Potential
DMN	Default Mode Network
MER	Maximum Entropy Ratio
PRI	Power-Ratio Index
pdBSI	pairwise-derived Brain Symmetry Index
rBSI	revised Brain Symmetry Index
MSS	Mean State Shift
SV	State Variance

Abbreviation	Meaning
LPP	Late Positive Potential
PAP	Peak Alpha Power
EF	Epileptogenic Focus
IAF	Individual Alpha Frequency
ITF	Individual Theta Frequency
EDs	Epileptiform Discharges
LORETA	Low Resolution Brain Electromagnetic Tomography
PLI	Phase Lag Index
Stimulation Type	
tDCS	Transcranial Direct Current Stimulation
tACS	Transcranial Alternating Current Stimulation
tRNS	Transcranial Random Noise Stimulation
so-tDCS	Slow Oscillatory Transcranial Direct Current Stimulation
HD-tDCS	High-Definition Transcranial Direct Current Stimulation
HD-tACS	High-Definition Transcranial Alternating Current Stimulation
tPCS	Transcranial Pulsed Current Stimulation
hf-tRNS	High-Frequency Transcranial Random Noise Stimulation
osc-tDCS	Oscillatory Transcranial Direct Current Stimulation
Data Analysis Method	
S	Statistical analysis
ML	Machine Learning
DL	Deep Learning

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