Sleep Medicine Reviews 89 (2026) 102321

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

Clinical Review

Assessing the effects of non-invasive transcranial electrical stimulation (tACS and tDCS) on electrophysiological sleep parameters - a systematic review

Ramona Hauser [a,*], Flavio Fröhlich [b,c], Kaspar A. Schindler [a], Andrea Seiler [a]

[a] Department of Neurology, Inselspital, Bern University Hospital, University Bern, Bern, Switzerland
[b] Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
[c] Carolina Center for Neurostimulation, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

ARTICLE INFO

Keywords:
Electric stimulation therapy
Transcranial direct current stimulation
Polysomnography
Electroencephalography
Sleep stages
Brain waves
Sleep disorders

ABSTRACT

Transcranial electrical stimulation (tES), including transcranial direct current stimulation (tDCS) and transcranial alternating current stimulation (tACS), is considered a safe method to modulate cortical activity and endogenous brain oscillations. Given the therapeutic potential of tES across various clinical conditions and the central role of sleep in restoration and memory consolidation, numerous studies have investigated its effects on sleep and sleep-related parameters, yielding inconsistent results. This systematic review provides an up-to-date synthesis of 51 studies assessing the impact of tES on objectively measured electrophysiological sleep outcomes in both healthy individuals and clinical populations. The reviewed studies demonstrate heterogeneous effects, reflecting substantial variability in study designs. Nonetheless, consistent trends emerge, including reduced NREM1 and increases in total sleep time, NREM2, and NREM3 following tES. Moreover, slow-oscillatory tES increased slow-wave power during sleep.

Here we show that tES, particularly slow-oscillatory tES, may positively influence sleep architecture and continuity by modulating endogenous brain oscillations. However, due to heterogeneous stimulation protocols, inconsistent findings, the limited number of significant effects and substantial risk of bias the current evidence remains inconclusive. Well-designed, large-scale trials targeting specific sleep outcomes are needed to clarify the therapeutic potential of tES.

1. Introduction

Interest in non-invasive brain stimulation (NIBS) techniques to modulate human brain activity has grown in recent years, partly due to the safety and reversible fashion of these methods [1,2]. While NIBS is generally well-tolerated, ongoing research seeks to better elucidate its mechanism of actions [3,4]. Numerous studies have applied NIBS in individuals with psychiatric, neurological, and neurocognitive conditions, demonstrating promising therapeutic potential [2,5–11].

Two widely studied NIBS methods are transcranial magnetic stimulation (TMS) and transcranial electrical stimulation (tES), which influence neural activity through magnetic fields or low-intensity electrical currents applied to the scalp. Electrical currents can be delivered with fixed polarity - known as transcranial direct current stimulation (tDCS) - or in an oscillatory form with alternating polarity, referred to as transcranial alternating current stimulation (tACS). Depending on the polarity, tDCS can depolarize or hyperpolarize the resting membrane potential, thereby altering cortical excitability [1,4,12]. In contrast, tACS aims to entrain endogenous oscillatory brain activity by modulating neuronal excitability within specific frequency bands [1,11,13]. The choice of stimulation site depends on the desired neuromodulatory effects and the clinical indication. While the dorsolateral prefrontal cortex (DLPFC) is the most frequently targeted region for tES, other stimulation sites may include the prefrontal cortex, central regions, parieto-occipital areas, and, less commonly, the cerebellum [4,10,14]. To date, no standardized tES protocols have been established, and stimulation parameters vary considerably across studies [15].

Growing interest has emerged in exploring the effects of NIBS on sleep, given its fundamental role in brain homeostasis and the

* Corresponding author.
E-mail addresses: ramonasarahauser@gmail.com (R. Hauser), flavio_frohlich@med.unc.edu (F. Fröhlich), kaspar.schindler@insel.ch (K.A. Schindler), andrea.seiler@insel.ch (A. Seiler).

https://doi.org/10.1016/j.smrv.2026.102321
Received 14 July 2025; Received in revised form 1 June 2026; Accepted 3 June 2026
Available online 11 June 2026
1087-0792/© 2026 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Abbreviations

ARAS	ascending reticular arousal system
DLPFC	dorsolateral prefrontal cortex
EEG	electroencephalography
Hz	hertz
mA	milliampere
μA	microampere
min	minutes
n/a	not applicable
n/s	not specified
nse	no significant effects
NIBS	non-invasive brain stimulation
NREM sleep	non-rapid eye movement sleep
PSG	polysomnography
RoB2	Cochrane risk of bias tool 2
REM sleep	rapid eye movement sleep
RATES	Report Approval for Transcranial Electrical Stimulation
SD	standard deviation
SO	slow oscillation
so-tDCS	slow-oscillatory transcranial direct current stimulation
STAMPS	spatiotemporal amplitude-modulated pattern
SWS	slow-wave sleep
tACS	transcranial alternating current stimulation
tDCS	transcranial direct current stimulation
tES	transcranial electrical stimulation
TMS	transcranial magnetic stimulation

restoration of cardiovascular, metabolic, hormonal, and immune functions [16]. Sleep is essential for learning, memory consolidation, emotional regulation, and cognitive and physical development, particularly during childhood and adolescence [17–21]. Furthermore, sleep appears to play a pivotal role in the function of the recently discovered glymphatic system, which facilitates the clearance of interstitial metabolic waste from the brain and may thereby protect against neurodegeneration [22–24]. However, the underlying mechanisms, generalizability, and sleep dependency of the glymphatic theory remain subjects of ongoing debate [25,26].

Sleep disturbances are highly prevalent and associated with increased risks for cognitive decline, depression, obesity, and various cardiovascular and neurological disorders [16,17,21,27–31]. In the light of global population aging and the rising prevalence of neurodegenerative diseases such as dementia, the development of effective sleep-enhancing interventions is of pressing importance [30–32].

Arousal and sleep regulation involve complex interactions within the ascending reticular arousal system (ARAS), a network of interconnected nuclei in the brainstem's reticular formation. During wakefulness the ARAS activates the cortex via thalamic projections, while during sleep, this activity is suppressed, reducing cortical excitation via “bottom-up” mechanisms [16,33,34]. In parallel, a “top-down” pathway – arising from oscillatory interactions between centro-frontal cortical neurons and thalamocortical circuits influenced by the reticular thalamic nucleus - also contributes to sleep-wake coordination [33,35–38]. These bottom-up and top-downs pathways are now recognized as complementary components of sleep-wake regulation [16].

The concept that cortico-thalamic dynamics influence arousal and sleep continuity has led to increased interest in modulating cortical activity to affect sleep-wake cycles [35]. However, studies investigating the impact of NIBS on sleep have yielded mixed results, ranging from changes in sleep architecture and oscillatory activity to null findings [39–42]. Clarifying these effects is crucial given the critical role of sleep in regenerative and memory-related processes.

To date, only a limited number of reviews have addressed the effects of NIBS on sleep, either by combining various stimulation methods and sleep assessments [14,43,44] or by focusing solely tES and its impact on subjectively and objectively measured sleep [45,46]. The present review aims to synthesize current evidence on tDCS and tACS on sleep in both healthy and clinical populations, with a particular emphasis on studies using objective electrophysiological sleep measures, in order to address the heterogeneity of this emerging research field.

2. Methods

This systematic review followed the recommendation of the PRISMA 2020 reporting guidelines to ensure transparency and completeness in methodology and reporting [47]. The first author (RH) conducted the literature screening, data extraction, analysis, and risk of bias assessment under the guidance and supervision of the three co-authors (FF, KS, AS) to ensure accuracy and methodological quality.

2.1. Search strategy and inclusion criteria

A comprehensive literature search was performed in the MEDLINE database via PubMed using a combination of Medical Subject Headings (MeSH) and free-text terms (search term see Box 1). The search was limited to English-language publications with no date restriction until the last search on 25 September 2025. After removing duplicates, records were screened for eligibility based on their titles, abstracts and methods sections using the following predefined inclusion criteria guided by the PICOS framework: i) Population: healthy and clinical population of any age. ii) Intervention: application of non-invasive electrical brain stimulation using either tACS or tDCS. iii) Comparison: baseline and posttreatment sleep measurements; control condition (if available): sham stimulation and/or other tES protocols e.g. different stimulation polarity or frequencies. Studies with concomitant standard care were eligible provided that the comparative condition allowed attribution to tES (e.g., standard care + tES vs. standard care). iv) Outcomes: objective assessment of sleep and/or sleep-related brain activity using polysomnography (PSG) or electroencephalography (EEG). v) Study design: experimental (controlled and uncontrolled) and quasi-experimental studies were included. Case reports were also considered for the qualitative, hypothesis-generating insights. Only studies published in peer-reviewed journals were included. Review articles were excluded from the analysis to avoid duplicate data reporting.

In addition, the reference list of the included studies and review articles were manually screened to identify further eligible studies. The

Box 1
Search strategy (last search updated on 25 September 2025)

“Sleep" [Mesh] OR (sleep OR nocturnal) AND (“Transcranial Direct Current Stimulation" [Mesh] OR tDCS OR tACS OR transcranial alternating current stimulation OR tES OR transcranial electric stimulation) AND (EEG OR electroencephalography OR PSG OR polysomnography).

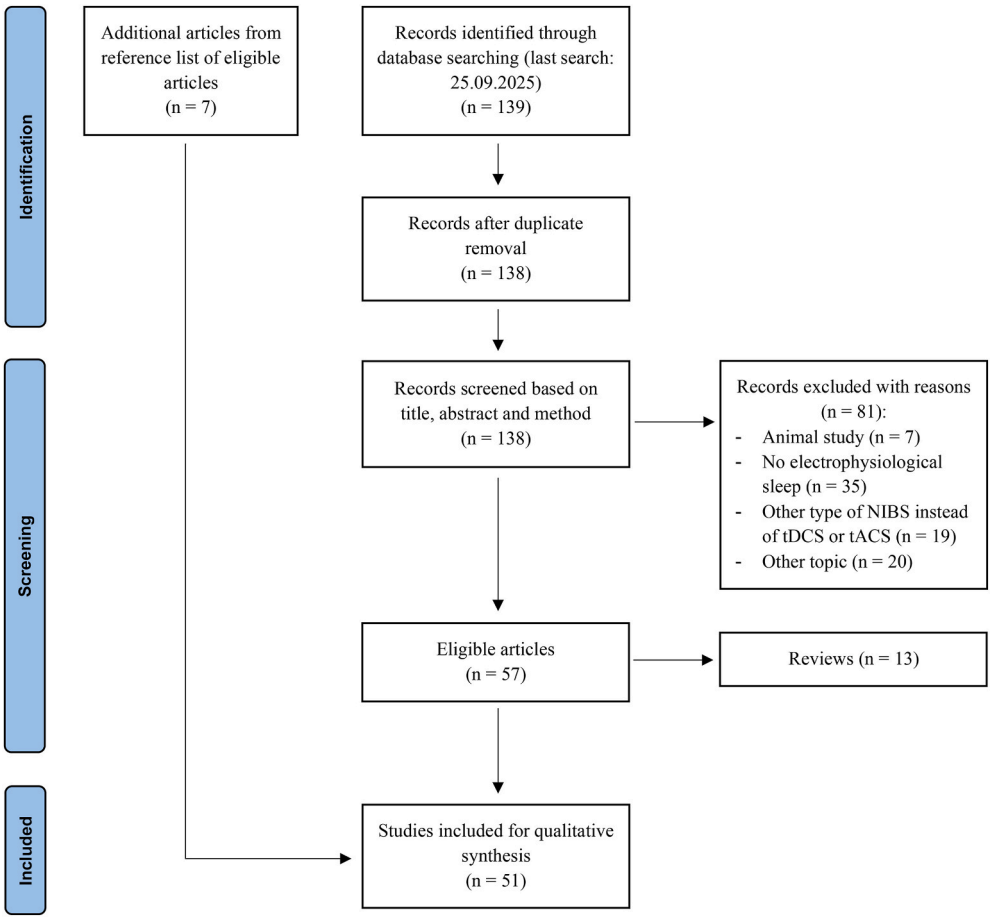


Fig. 1. Flowchart of the selection process.

study selection process is illustrated in the flow diagram (Fig. 1).

2.2. Study variables and data extraction

Data from eligible studies were extracted using a standardized Excel spreadsheet. The extracted variables were determined by employing all elements of the short version of the recently published Report Approval for Transcranial Electrical Stimulation (RATES) checklist [15]. Developed by Nejati et al. due to the need for standardized reporting criteria, this consensus-based checklist comprises a total of 26 elements to enhance transparency, comparability and quality assessment of tES studies. In addition, we defined variables to systematically extract data on electrophysiological sleep, resulting in the following extracted variables, which were grouped into different categories for each reference:

(a) Study characteristics: author, title, year of publication, journal, country, study design (parallel, crossover, uncontrolled), demographic characteristics (age, gender, education, handedness), sample size, study population (healthy or with clinical condition), participant eligibility criteria and whether sleep was the primary outcome (yes/no, if no, the primary outcome was specified); (b) Stimulation characteristics: type of tES, timing of stimulation, stimulation frequency, current intensity, current waveform, current amplitude, duration of stimulation, ramping up/down duration, number of sessions, electrode parameters (positioning, shape, size, number), montage, stimulator type, brain target area, duration until start of task performance (if applicable) and reported adverse events; (c) Objective sleep parameters (in accordance with the American Academy of Sleep Medicine (AASM) guidelines [48], if applicable): total sleep time, time in bed, sleep latency, sleep efficiency, arousal index, REM latency, slow-wave sleep latency, spindle events, apnea-hypopnea index, and both absolute and percentage values for sleep stages NREM1, NREM2, NREM3 and REM; (d) Spectral power analysis: frequency-specific power during sleep (definitions of frequency ranges for slow-wave activity, delta, theta, alpha, beta and gamma waves according to AASM guidelines [48]), while an additional range was defined for fast spindle activity (12-15 Hz), also known as the sigma range [49]. When sleep staging followed the classification of Rechtschaffen and Kales [50], stages S3 and S4 were considered equivalent to NREM3 in accordance with the AASM guidelines [48], reflecting slow-wave sleep.

Given the considerable heterogeneity among the study methods, populations, and outcomes, a meta-analysis was not conducted. Therefore, the findings are presented in a narrative format.

2.3. Risk of bias assessment

Methodological quality of the included studies was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool [51]. Each study was evaluated across five domains of bias, or six domains for crossover trials: randomization process; deviations from intended interventions; missing outcome data; measurement of the outcome; selection of the reported result; and, for crossover trials, bias arising from period and carryover effects. In accordance with the RoB2 guidelines, a judgment of « some concerns » or « high » risk of bias for any individual domain resulted in the overall assessment being classified as « some concerns » or « high » risk of bias. The overall judgements proposed by the RoB2 algorithm were reviewed and verified by the first author (RH). As RoB 2 was originally developed for randomized controlled trials, its use in assessing uncontrolled trials (n = 2 [52,53]) should be interpreted with caution. However, applying it in this context aligns with methodologies adopted in other systematic reviews [54,55].

Table 1
Study characteristics.

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
Li et al. [63]	2025	China	Double-blind randomized, controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 39.1 ± 13.5 (active 42.5 ± 11.13; sham 35.89 ± 14.91); gender: 29:26 (active 15:14; sham 14:12); education: active 13.19 ± 4.26; sham 17.29 ± 9.34; handedness: n/s Sample size: 55 (active: 27; sham: 28) Population: chronic insomnia	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal Duration: 30 min Time: daytime, awake Number of Sessions: 10 sessions (1x/d for 10 consecutive days) Adverse Events: none Montage: anode over Fz (over DMPFC), surrounded by four cathodes over FPz, F3, Cz, F4 Stimulated brain area: DMPFC	Yes (Primary + secondary outcome: Sleep questionnaires (PSQI, FFS) Tertiary outcome: sleep architecture)
Wang et al. [65]	2025	China	Double-blind, randomized, controlled, crossover design. Condition 1: non-individualized frequency tACS Condition 2: individualized frequency tACS Base condition: sham stimulation	Demographic characteristics: age: 38.4 ± 12.4; gender: 20:18 (healthy 4:4; subsyndromal insomnia 10:8; insomnia 6:6); education: n/s; handedness: n/s Sample size: 38 (healthy: 8; subsyndromal insomnia: 18; insomnia: 12) Population: healthy, subsyndromal insomnia, insomnia	tACS	Intensity: 520 μA Frequency: non-individualized: 10 Hz (Wake), 5 Hz (NREM1), 0.8 Hz (NREM2) individualized: alpha band (Wake stage), theta band (NREM1), delta band (NREM2), sinusoidal current Polarization: n/a Duration: stimulation until three consecutive NREM2 sleep stages or reaching 1 h Time: nocturnal, sleep onset process Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: 9-channel high definition transcranial electrical stimulator; occipito-parietal (Wake), parieto-central (NREM1), fronto-central (NREM2) Stimulated brain area: occipito-parietal (Wake), parieto-central (NREM1), fronto-central (NREM2)	Yes
Thom et al. [72]	2025	UK	Single-blind, randomized, controlled, crossover, repeated-measures design. Condition 1: anodal tDCS over right motor cortex Condition 2: anodal tDCS over left motor cortex	Demographic characteristics: age: 25 ± 4.37; gender: 10:9; education: n/s; handedness: 19:0 Sample size: 19 Population: healthy	tDCS	Intensity: 1 mA Frequency: n/a Polarization: anodal Duration: 20 min Time: daytime, awake Number of Sessions: 2 (5 d over contralateral motor cortex) Adverse Events: n/s Montage: anode over C3 or C4, cathode over contralateral supraorbital cortex Stimulated brain area: left or right motor cortex	Yes
Shi et al. [73]	2025	China	Single-blind, randomized, controlled, crossover design Control condition: sham stimulation	Demographic characteristics: age: 23.7 ± 1.88 (23.81 ± 1.68; 23.59 ± 2.09); gender: 16:17 (8:8; 8:9); education: 81.8% college or higher (87.5%; 94.1%); handedness: 33:0 Sample size: 33 (group A: 16; group B: 17) Population: healthy	tACS	Intensity: 0.75 mA Frequency: 5 Hz Polarization: n/a Duration: 40 min Time: daytime, awake Number of Sessions: 1 Adverse Events: scalp tingling (8.3%). Montage: anodes over F3/F4, cathodes over M1/M2. Stimulated brain area: frontal cortex	Yes
Tabarak et al. [74]	2024	China	Single-blind, randomized, controlled, crossover design Control condition: sham stimulation	Demographic characteristics: age: 19.27 ± 1.41; gender: 11:10; education: n/s; handedness: n/s Sample size: 21 Population: healthy	tACS	Intensity: 2-3 mA Frequency: 60 Hz, sinusoidal current Polarization: n/a Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2	No (memory performance)

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
						Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F5 and F6, mastoid return Stimulated brain area: frontal cortex	
Ladenbauer et al. [75]	2023	Germany	Single-blind, randomized, controlled, crossover design. Control condition: standardized so-tDCS (0.75 Hz) or sham stimulation.	Demographic characteristics: age: 65.3 ± 8.5; gender: 13:15; education: 15.3 ± 3.1; handedness: n/s Sample size: 28 (30 s: standard frequency: 26, eigenfrequency: 21, sham: 28; 2 min: 24:23:28; 5 min: 23:21:22) Population: healthy	so-tDCS	Intensity: 0-262.5 μA Frequency: 0.75 Hz or eigenfrequency between 0.5 and 1 Hz, sinusoidal current Polarization: anodal Duration: 5 × 30 s, 2 min and 5 min with stimulation free intervals (at least 90s) Time: daytime nap, after 4 min of NREM2 Number of Sessions: 7 (each stimulation condition, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	Yes
Motamedi et al. [64]	2023	USA	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 50 ± 10; gender: 1:9; education: n/s; handedness: n/s Sample size: 9 Population: chronic insomnia	tACS	Intensity: 0.75 mA Frequency: 0.75 Hz, sinusoidal current Polarization: n/a Duration: 5 min Time: nocturnal, sleep onset process Number of Sessions: start tACS upon lights out with 5 min stimulation - 1 min pause- repeat of circle until patient entered NREM2 sleep Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	Yes
Wang et al. [41]	2023	China	Open-label, randomized, controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 22.1 ± 1.7 (control 22.38 ± 2.16; tDCS 21.85 ± 3.47); gender: 34:0; education: n/s; handedness: 34:0 Sample size: 34 (tDCS: 17; sham: 17) Population: healthy	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 30 min Time: daytime, awake, just before bedtime Number of Sessions: 2 (repeat the next morning with same duration of stimulation) Adverse Events: n/s Montage: anode at F3, cathode at FP1 Stimulated brain area: DLPFC	Yes
Li et al. [76]	2022	China	Double-blind, randomized, controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 44.8 ± 15.25; gender: 15:22 (tDCS 6:13; sham 9:9); education: n/s; handedness: 37:0 Sample size: 37 (tDCS: 19; sham: 18) Population: major depressive disorder (MDD)	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 20 min Time: daytime, awake Number of Sessions: 10 (5x a week) Adverse Events: none Montage: anode at F3, cathode at F4 Stimulated brain area: frontal/prefrontal cortex	Yes
Robert et al. [77]	2022	USA	Single-blind, randomized, controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 20.67 ± 2.93; gender: 14:22; education: n/s; handedness: 36:0 Sample size: 36 (tACS: 15;	tACS	Intensity: 1.5 mA Frequency: individual dominant frequency in the SWO band (0.5–1.2 Hz) Polarization: n/a Duration: 5 × 5 min with 1 min	No (memory; object location memory task)

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
				sham: 21) Population: healthy		stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 1 Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	
Yu et al. [78]	2022	China	Double-blind, randomized-controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 54.8 ± 9.65 (tDCS: 52.4 ± 10.3 with sleep cycles (sc); 47.8 ± 8.5 without sc; sham: 52.4 ± 10.3 with sc; 61.0 ± 8.7 without sc); gender: 32:1; education: n/s; handedness: n/s Sample size: 33 (tDCS: 17 (10 with sc +7 without sc); sham: 16 (10 with sc + 6 without sc)) Population: vegetative state (VS), with or without sleep cycles (sc) or minimally conscious state (MCS)	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 20 min Time: daytime, unknown Number of Sessions: 10 (1x/d for 10 d) Adverse Events: n/s Montage: anode over left DLPFC (F3), cathode over right supraorbital region (Fp2) Stimulated brain area: DLPFC	Yes
Zhou et al. [53]	2022	China	Case report	Demographic characteristics: age: 52; gender: 0:1; education: n/s; handedness: n/s Sample size: 1 Population: chronic insomnia	tDCS TMS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 20 min Time: daytime, awake, after TMS Number of Sessions: 20 (1x/d for 20 consecutive days) Adverse Events: transient skin redness Montage: anode over left prefrontal cortex (F3-Fp1), cathode over right prefrontal cortex (F4-Fp2) Stimulated brain area: PFC	Yes
Charest et al. [79]	2021	Canada	Single-blind, randomized, controlled, repeated-measures parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 21.1 ± 2.1 (tDCS 22.07 ± 1.83; sham 20.13 ± 2.03); gender: 15:15 (tDCS 8:7; sham 7:8); education: n/s; handedness: n/s Sample size: 30 Population: healthy, sleep difficulties	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal Duration: 20 min Time: daytime, awake, 90 min before bedtime Number of Sessions: 2 (1 d) Adverse Events: n/s Montage: Anode over Fpz, cathode over Pz Stimulated brain area: PFC	Yes
Dehnavi et al. [80]	2021	Iran, Germany	Single-blind, pseudo-randomized, controlled, crossover design. Control condition: sham stimulation or no stimulation (no memory tasks, undisturbed sleep).	Demographic characteristics: age: 22.4 ± 2.12; gender: 10:15; education: n/s; handedness: 25:0 Sample size: 25 Population: healthy	so-tDCS	Intensity: 0-300 μA Frequency: individual frequency between 0.7 and 1.2 Hz (effective stimulation 0.86 ± 0.02 Hz), trapezoid shape Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 3 (7d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	Yes
Hathaway et al. [81]	2021	USA, Argentina	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 42; gender: 7:6; education: n/s; handedness: n/s Sample size: 13 Population: healthy	tACS	Intensity: 520 μA Frequency: 0.5 Hz, sinusoidal Polarization: n/a Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
						Number of Sessions: 1 Adverse Events: n/s Montage: frontopolar (electrodes near Fp1 and Fp2) and inferofrontal returns (electrodes near F9 and F10) Stimulated brain area: anterior ventral limbic site	
Hubbard et al. [82]	2021	USA	Randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: n/s; gender: n/s; education: n/s; handedness: 18:0 Sample size:18 Population: healthy	tDCS STAMPS tACS STAMP	Intensity: 2.5 mA total, 150 μA −1.5 mA Frequency: 40 Hz, sinusoidal Polarization: cathodal Duration: entire duration of encoding resp. nocturnal sleep Time: daytime (while encoding) and nocturnal (half of participants received STAMP during subsequent night), STAMP delivered during up-states of SWO detected by an algorithm Number of Sessions: 4 (2 experimental, 2 sham, repeat the following night respectively 8 d apart) Adverse Events: n/s Montage: all over the head with a 64-channel-neoprene cap montage with 32 stimulation channels Stimulated brain area: cortex	No (metamemory sensitivity)
Xie et al. [83]	2021	China	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 22.1 ± 3.8; gender: 18:12; education: n/s; handedness: 30:0 Sample size: 30 Population: healthy	tACS	Intensity: 1.4 mA Frequency: 5 Hz, sinusoidal current Polarization: n/a Duration: 15 min (10 min steady stimulation, last 5 min stimulus current gradually weakened) Time: daytime, during wakefulness and sleep onset process Number of Sessions: 2 (1stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F7-T3 and F8-T8 Stimulated brain area: DLPFC	Yes
Noreika et al. [84]	2020	Finland, UK	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 26.8 ± 4.4; gender: 4:6; education: n/s; handedness: 10:0 Sample size: 10 (5:5) Population: healthy	tDCS	Intensity: 1 mA Frequency: n/a Polarization: anodal and cathodal Duration: 10 min Time: nocturnal, during REM sleep, start with second sleep cycle Number of Sessions: 6 (2-3 sessions in one night, few hours resp. 1 week) Adverse Events: n/s Montage: anode over right senosmotoric cortex (C4), cathode over left sensomotoric cortex (C3) Stimulated brain area: sensomotoric cortex	No (dream movements, subjective dream experience)
Pilly et al. [85]	2020	USA	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.96 ± 6.08; gender: 10:15; education: n/s; handedness: 25:0 Sample size: 25 Population: healthy	tDCS STAMPS tACS STAMP	Intensity: 2.5 mA total, 150 μA −1.5 mA Frequency: 40 Hz, sinusoidal Polarization: cathodal Duration: entire duration of encoding resp. nocturnal sleep Time: daytime (while encoding) and nocturnal (half of participants received	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
						STAMP during subsequent night), STAMP delivered during up-states of SWO detected by an algorithm Number of Sessions: 4 (2 experimental, 2 sham, repeat the following night respectively 8 d) Adverse Events: itching, burning, tingling during active stimulation Montage: all over the head with a 64-channel-neoprene cap montage with 32 stimulation channels Stimulated brain area: cortex	
Zhou et al. [71]	2020	China	Double-blind, randomized, controlled, parallel design. Control condition: sham stimulation.	Demographic characteristics: age: 42.3 ± 9.94 (tDCS 43.91 ± 11.20; sham 40.45 ± 8.31); gender: 30:60 (tDCS 16:31, sham 14:29); education: n/s; handedness: n/s Sample size: 90 (tDCS: 47; sham: 43) Population: major depressive disorder (MDD) and insomnia	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 30 min Time: daytime, awake Number of Sessions: 20 (on consecutive days excluding weekends) Adverse Events: n/s Montage: anode over left DLPFC, cathode over right DLPFC Stimulated brain area: DLPFC	Yes
Bueno-Lopez et al. [39]	2019	Germany	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.2 ± 1.9; gender: 10:13; education: n/s; handedness: n/s Sample size: 23 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: anodes at F3 and F4, cathodes at M1 and M2 Stimulated brain area: frontal/prefrontal cortex	Yes
Cellini et al. [59]	2019	Italy, USA	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 32.24 ± 8.06; gender: 11:6; education: college 5; associate degree 6; Bachelor's degree 4; graduate degree 2; handedness: n/s Sample size: 17 Population: healthy	so-tDCS	Intensity: 2 mA Frequency: 0.75 Hz, trapezoid current Polarization: anodal Duration: stimulation cycle of 30 s with 4 s of active stimulation and 4 s recovery of EEG + 22 s of artifact-free EEG data Time: daytime nap, after min 5 s after detected SO Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: Anodes anterior and posterior of F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; declarative memory task)
D'Atri et al. [86]	2019	Italy	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 24.3 ± 3.1; gender: 9:8; education: n/s; handedness: n/s Sample size: 17 Population: healthy	tACS	Intensity: 0.6 mA Frequency: 5 Hz, sinusoidal current Polarization: n/a Duration: 10 min Time: daytime, awake Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: Electrodes between F7-T7 and F8-T8	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
Frase et al. [67]	2019	Germany	Single-blind, controlled, quasi-randomized, crossover, repeated-measures design. Conditions: anodal tDCS, cathodal tDCS or sham stimulation.	Demographic characteristics: age: 43.8 ± 15.1; gender: 6:13; education: n/s; handedness: n/s Sample size: 19 Population: chronic insomnia	tDCS	Stimulated brain area: frontotemporal cortex Intensity: 2 mA Frequency: n/a Polarization: bilaterally anodal and cathodal Duration: 2 × 13 min anodal stimulation 2 × 9 min cathodal stimulation with 20 min stimulation free interval Time: daytime, awake, 60 min before bedtime Number of Sessions: 6 (2/night respectively 1/week) Adverse Events: prickling, itching, headache, burning were reported, but both in sham as well as in stimulation group Montage: bifrontal prefrontal, electrodes at Fp1 und Fp2 Stimulated brain area: PFC	Yes
Ketz et al. [87]	2018	USA	Single-blind, randomized, controlled, crossover, repeated-measures design. Conditions: object target/people target, active stimulation, sham stimulation.	Demographic characteristics: age: 22.25 ± 4.96; gender: 13:3; education: n/s; handedness: 16:0 Sample size: 16 Population: healthy	tACS	Intensity: 1.5 mA Frequency: individual dominant frequency in the SWO band (0.5–1.2 Hz), sinusoidal current Polarization: n/a Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (n/s) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; target detection task)
Koo et al. [88]	2018	Germany	Single-blind, pseudo-randomized, controlled, crossover design. Control condition: sham stimulation or no stimulation (no memory tasks, undisturbed sleep).	Demographic characteristics: age: 22.4 ± 2.12; gender: 10:15; education: n/s; handedness: 25:0 Sample size: 25 Population: healthy	so-tDCS	Intensity: 0-300 μA Frequency: individual frequency between 0.7 and 1.2 Hz (effective stimulation 0.86 ± 0.02 Hz), trapezoid shape Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 3 (1 stimulation, 1 sham, 1 task, 7 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; declarative and procedural memory tasks)
Johnson et al. [61]	2018	UK	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 20.73 ± 0.3; gender: 5:10; education: n/s; handedness: 15:0 Sample size: 15 Population: healthy	so-tDCS	Intensity: 0.4 mA Frequency: 0.75 Hz or 5 Hz, square current Polarization: cathodal Duration: 25 min Time: nocturnal, start after 4 min of continuous REM sleep Number of Sessions: 3 (1 stimulation 0.75 Hz, 1 stimulation 0.75 Hz, 1 sham, 48h) Adverse Events: none Montage: cathode at F4, anode at contralateral forearm Stimulated brain area: right hemisphere	No (memory; declarative and emotional memory

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
Ladenbauer et al. [89]	2017	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 70.6 ± 8.9; gender: 9:7; education: 15.2 ± 3; handedness: 16:0 Sample size: 16 (8:8) Population: mild cognitive impairment	so-tDCS	Intensity: 0-262.5 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: daytime nap, start after 4 min of NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 14 d) Adverse Events: one patient tingling perception Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Lafon et al. [52]	2017	USA	Open-label, non-randomized, uncontrolled single group design.	Demographic characteristics: age: 33.5 ± 10.4; gender: 5:3; education: n/s; handedness: 8:0 Sample size: 8 Population: medication-refractory epilepsy and patients undergoing surgical evaluation with iEGG monitoring	tACS	Intensity: 0.3-2.5 mA Frequency: 0.75 Hz or 1 Hz, sinusoidal current, trapezoidal shape Polarization: n/a Duration: 5-10 min, up to 4 blocks of stimulation Time: nocturnal, during sleep (4 daytime nap, 3 nocturnal sleep) Number of Sessions: 1 Adverse Events: none (one patient woke up due to itching during a stimulation block with 2.5 mA) Montage: frontopolar F3 and F4 and occipital poles return Stimulated brain area: full cortex	Yes
Frase et al. [90]	2016	Germany	Single-blind, quasi-randomized, controlled, crossover, repeated-measures design. Conditions: anodal tDCS, cathodal tDCS or sham stimulation.	Demographic characteristics: age: 53.7 ± 6.9; gender: 6:13; education: n/s; handedness: 19:0 Sample size: 19 Population: healthy	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: 2 × 13 min anodal stimulation 2 × 9 min cathodal stimulation Time: daytime, awake, 60 min before bedtime Number of Sessions: 6 (2/night respectively 1/week) Adverse Events: some skin sensations, some headache without any differences between the tDCS conditions superficial skin lesion due to unintended direct contact of cathode and anode Montage: bifrontal frontopolar, electrodes at Fp1 und Fp2 Stimulated brain area: frontopolar cortex	Yes
Ladenbauer et al. [91]	2016	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 65.1 ± 1; gender: 8:10; education: 15.6 ± 0.6; handedness: 18:0 Sample size: 18 (9:9) Population: healthy	so-tDCS	Intensity: 0-262.5 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: daytime nap, start after 4 min of NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 14d) Adverse Events: one patient tingling perception Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
Lustenberger et al. [70]	2016	USA	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 22 ± 4; gender: 16:0; education: n/s; handedness: 16:0 Sample size: 16 Population: healthy	tACS	Intensity: 1 mA Frequency: 12 Hz, sinusoidal current Polarization: n/a Duration: 1.5 s stimulation followed by 6.5 s time out total stimulation time with repeats between 504 and 1647 s Time: nocturnal, during sigma activity (start when individualized sigma activity threshold exceeded) Number of Sessions: 2 (1 stimulation, 1 sham, 5 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Passmann et al. [56]	2016	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 65 ± 1; gender: 11:13; education: 17 ± 4; handedness: n/s Sample size: 24 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 14-21 d) Adverse Events: none (two participants: tingling perception during sham) Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Del Felice et al. [92]	2015	Italy	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 34.2 ± 15.4; gender: 4:8; education: 15.7 ± 2.5; handedness: n/s Sample size: 12 Population: pharmaco-resistant mesial temporal lobe epilepsy and mesial sclerosis	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 6 × 5 min block with 1 min stimulation free intervals Time: daytime, awake, 1 h before daytime nap (for 60 min or Time in Bed 1h 45 min) Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: anode at F7/T7 or F8/T8 (over affected temporal lobe), cathode over ipsilateral mastoid Stimulated brain area: frontotemporal cortex	Yes
Garside et al. [93]	2015	UK	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 21 ± 0.93; gender: 4:4; education: n/s; handedness: n/s Sample size: 8 Population: healthy	tACS	Intensity: 550 μA Frequency: 0.75 Hz, sinusoidal current Polarization: n/a Duration: 5 × 5 min with 1 min stimulation free intervals Time: daytime nap, after 4 min of NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, n/s) Adverse Events: none Montage: electrodes at F3 and F4 Stimulated brain area: frontal/ prefrontal cortex	Yes
Munz et al. [57]	2015	Germany	Double-blind, randomized, controlled, crossover design.	Demographic characteristics: age: 12.3 ± 1.39; gender: 14:0; education: n/s; handedness: n/s	so-tDCS	Intensity: 0-250 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
			Control condition: sham stimulation.	Sample size: 14 Population: ADHD		stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	
Saebipour et al. [66]	2015	Iran	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 34 ± 7; gender: 4:2; education: n/s; handedness: n/s Sample size: 6 Population: primary insomnia	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, trapezoid current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: one participant with mild transient headache Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Sahlem et al. [40]	2015	USA	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 25 ± 2.3; gender: 3:9; education: n/s; handedness: n/s Sample size: 12 Population: healthy	so-tDCS	Intensity: 0-600 μA Frequency: 0.75 Hz, square current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 14 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	No (memory; declarative and non-declarative memory tests)
Westerberg et al. [94]	2015	USA	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 73.4 (65-85); gender: 3:16; education: 15.4 (12-20); handedness: n/s Sample size: 19 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F7 and F8 Stimulated brain area: frontal cortex	No (memory; declarative and non-declarative memory tests)
Prehn-Kristensen et al. [68]	2014	Germany	Double-blind, randomized, controlled, crossover design. Control condition: Sham stimulation.	Demographic characteristics: age: 12.1 ± 1.4 (ADHD), 11.9 ± 1.4 (healthy); gender: 24:0; education: n/s; handedness: n/s Sample size: 24 (12 ADHD, 12 healthy) Population: healthy, ADHD	so-tDCS	Intensity: 0-250 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	No (memory; declarative memory test)
Voss et al. [60]	2014	Germany	Double-blind, randomized, controlled, crossover	Demographic characteristics: age: 18-26; gender: 12:15; education: n/s; handedness: n/	tACS	Intensity: 250 μA Frequency: 2 Hz, 6 Hz, 12 Hz, 40 Hz, 70 Hz, 100 Hz,	No (dream report)

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
			design. Control conditions: sham stimulation, tACS at different frequencies (2 Hz, 6 Hz, 12 Hz, 25 Hz, 70 Hz, 100 Hz)	s Sample size: 27 Population: healthy		sinusoidal current Polarization: n/a Duration: 30 s Time: nocturnal, start after 2 min of continuous REM sleep Number of Sessions: 4 (different frequencies over 4 nonconsecutive nights) Adverse Events: none Montage: electrodes at F3 and F4, mastoid return (close to TP9 and TP10) Stimulated brain area: frontal/prefrontal cortex	
Antonenko et al. [58]	2013	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.4 ± 1.9; gender: 8:7; education: similar (high school); handedness: 15:0 Sample size: 15 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 6-8 x 4 min stimulation, separated by 1 min stimulation free intervals Time: daytime nap, after 4 min of NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 4 w) Adverse Events: none Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; declarative memory task)
Eggert et al. [95]	2013	Germany	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 69.1 ± 7.7; gender: 10:16; education: n/s; handedness: n/s Sample size: 26 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none (one participant reported nocturnal perceptions of current flow during sham condition) Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; declarative and procedural memory task)
Göder et al. [96]	2013	Germany	Pseudo-randomized, controlled crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 33 ± 8; gender: n/s; education: n/s; handedness: n/s Sample size: 14 Population: paranoid schizophrenia	so-tDCS	Intensity: 0-300 μA Frequency: 0.75 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 10 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/prefrontal cortex	No (memory; declarative, procedural and short-term memory tasks)
Reato et al. [62]	2013	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.8 (20-28); gender: 6:7; education: n/s; handedness: 13:0 Sample size: 13 Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 0.75 Hz, trapezoid current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none	No (power and spatial coherence changes in EEG data)

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
						Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	
Jakobson et al. [97]	2012	Australia	Experiment 1: Double-blind, controlled, crossover, repeated-measures design. Control conditions: low tDCS or sham stimulation. Experiment 2: Double-blind, controlled, crossover, repeated-measures design. Control conditions: anodal tDCS, cathodal tDCS or sham stimulation.	Demographic characteristics: age: 23.2 ± 5.1 (experiment 1), 25.7 ± 5.6 (experiment 2); gender: 5:12 (experiment 1), 2:8 (experiment 2); education: n/s; handedness: 17:0 (experiment 1), 10:0 (experiment 2) Sample size: 17 (experiment 1); 10 (experiment 2) Population: healthy	tDCS	Intensity: Experiment 1: condition: 2 mA (or 1.5 mA if not tolerated), control condition: 1 mA Experiment 2: always 2 mA Frequency: n/a Polarization: anodal and cathodal Duration: Experiment 1: condition: 181 s, control condition: 41 s Experiment 2: always 181 s Time: nocturnal Number of Sessions: experiment 1: 3, experiment 2: 4 (n/s, within the same night) Adverse Events: n/s Montage: Experiment 1: anode at P4, cathode at Fpz Experiment 2: conditions 1-3: anode at P4, cathode at Fpz condition 4: anode at Oz, cathode at Fpz Stimulated brain area: frontal and right posterior parietal cortex	No (dream report)
Marshall et al. [98]	2011	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.9 (18-35, NREM), 24.3 (21.3-30.6, REM); gender: 10:14 (NREM), 8:8 (REM); education: n/s; handedness: 25:0 (NREM), 16:0 (REM) Sample size: 25 (NREM); 16 (REM) Population: healthy	so-tDCS	Intensity: 0-260 μA Frequency: 5 Hz, sinusoidal current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal experiment 1: tDCS after 4 min of continuous NREM sleep experiment 2: tDCS after 4 min of REM sleep after 3:00 a.m. Number of Sessions: 2 (1 stimulation, 1 sham, 10 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Nitsche et al. [99]	2010	Germany	Single-blind, randomized, controlled, crossover, repeated-measures design Test condition: anodal tDCS or cathodal tDCS Control condition: sham stimulation.	Demographic characteristics: age: 35.6 ± 11.9 (36.6 ± 10.6; 33.7 ± 11.7; 36.7 ± 13.5); gender: 4:16 (2:5; 2:5; 0:6); education: n/s; handedness: 20:0 Sample size: 20 (7:7:6) Population: healthy	tDCS	Intensity: 1 mA Frequency: n/a Polarization: anodal and cathodal Duration: 15 min Time: nocturnal, during second REM sleep period Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: anode/cathode 3 cm anterior to C3, return electrode above the contralateral right orbit Stimulated brain area: premotor cortex	Yes
Roizenblatt et al. [69]	2007	Brasil	Double-blind, randomized, controlled, parallel design. Conditions: tDCS over DLPFC, tDCS over M1 or sham stimulation.	Demographic characteristics: age: 53.4 ± 8.9 (54.2 ± 7.4 DLPFC tDCS; 54.8 ± 9.3 M1 tDCS; 50.8 ± 10.2 sham); gender: 0:32; education: n/s; handedness: n/s Sample size: 32 (11 DLPFC tDCS; 11 M1 tDCS; 10 sham) Population: fibromyalgia	tDCS	Intensity: 2 mA Frequency: n/a Polarization: anodal Duration: 20 min Time: daytime, awake Number of Sessions: 5 (5 consecutive days) Adverse Events: similar adverse effects across three treatment groups, type of adverse effects not specified	Yes

(*continued on next page*)

Table 1 (*continued*)

Authors	Publication year	Country	Study design	Study sample[a]	Type of tES	Stimulation characteristics	Sleep as primary outcome[b]
						Montage: condition 1: primary M1, anode at C3, cathode over contralateral supraorbital area condition 2: left DLPFC, anode at F3, cathode over contralateral supraorbital area Stimulated brain area: M1, DLPFC	
Marshall et al. [100]	2006	Germany	Single-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.8 (20-28); gender: 6:7; education: n/s; Handedness: 13:0 Sample size: 13 Population: healthy	so-tDCS	Intensity: 0-260 μA (current density 0.517 mA/cm2) Frequency: 0.75 Hz, trapezoid current Polarization: anodal Duration: 5 × 5 min with 1 min stimulation free intervals Time: nocturnal, start after 4 min of continuous NREM2 Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: none Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	Yes
Marshall et al. [42]	2004	Germany	Double-blind, randomized, controlled, crossover design. Control condition: sham stimulation.	Demographic characteristics: age: 23.8 (19-28); gender: 18:0; education: n/s handedness: n/s Sample size: 18 Population: healthy	tDCS	Intensity: 260 μA Frequency: n/a Polarization: anodal Duration: 30 min (15 s on, 15 s off) Time: experiment 1: nocturnal, start after 30 s of NREM3/4 sleep experiment 2: daytime, awake Number of Sessions: 2 (1 stimulation, 1 sham, 7 d) Adverse Events: n/s Montage: electrodes at F3 and F4, mastoid return Stimulated brain area: frontal/ prefrontal cortex	No (memory; declarative and procedural memory task)

Abbreviations: n/s = not specified, n/a = not applicable, tES = transcranial electrical stimulation, tDCS = transcranial direct current stimulation, tACS = transcranial alternating current stimulation, so-tDCS = slow-oscillatory transcranial direct current stimulation, TMS = transcranial magnetic stimulation, STAMPS = spatiotemporal amplitude-modulated patterns, NREM = non-rapid eye movement sleep, REM = rapid eye movement sleep, PFC = prefrontal cortex, DLPFC = dorsolateralprefrontal cortex, DMPFC = dorsomedialprefrontal cortex, M1 = primary motor cortex, SO = slow oscillation, ADHD = attention-deficit/hyperactivity disorder.

[a] Age in years [mean ± SD] or [mean (range)], gender (m:f), education (years/%), handedness (right:left).

[b] If sleep was not the primary outcome, indication of primary outcome.

3. Results

The literature search yielded 139 articles. After removal of duplicates, 138 articles remained for screening for eligibility. Of these, 81 articles were excluded based on predefined criteria (see Fig. 1 for details). Following the exclusion of review articles, 44 articles studied fulfilled the eligibility criteria. An additional seven studies [56–62] were identified through reference list screening, resulting in total of 51 studies included in the final analysis.

3.1. Study characteristics

The included studies were published between 2004 and 2025. Most employed a crossover design (n = 41), while fewer used a parallel design (n = 8), one study [52] had an uncontrolled, single-group design and one study [53] was a case report. Of the 51 studies, 13 (25.5%) investigated the effects tACS, 15 (29.4%) focused on tDCS, two (3.9%) examined spatiotemporal amplitude-modulated patterns (STAMPs) of tES, and 21 (41.2%) assessed slow-oscillatory tDCS (so-tDCS).

Electrophysiological sleep was the primary outcome in 32 studies (62.7%) and a secondary or tertiary outcome in 19 studies (37.3%). Among the latter, memory consolidation was the primary endpoint in 13 studies (25.5%).

The majority of studies (n = 35, 68.6%) were conducted in healthy populations, while 16 articles (31.4%) studied clinical populations, with insomnia being the most frequently studied condition (n = 6 [53, 63–67]).

Apart from two studies [57,68] involving children with attention-deficit/hyperactivity disorder (ADHD), all studies examined adult participants aged between 19.3 and 73.4 years. Over half of the studies (n = 30, 58.8%) focused on younger adults (mean age <35 years) and 12 articles (23.5%) investigated elderly populations with a mean age of ≥50 years. Most studies showed an overall balanced female- male ratio. Notable exceptions included one study focusing exclusively on women with fibromyalgia [69], and five studies that enrolled only male participants [41,42,57,68,70].

Sample sizes were generally small: 27 studies (52.9%) included fewer than 20 participants, and only two studies [63,71] enrolled more than 50 participants.

Geographically, most studies were conducted in Germany (n = 19, 37.3%), followed by the United States (n = 10, 19,6%), China (n = 10, 19.6%), United Kingdom (n = 3, 5.9%) and Italy (n = 3, 5.9%). An overview of the individual study characteristics is provided in Table 1.

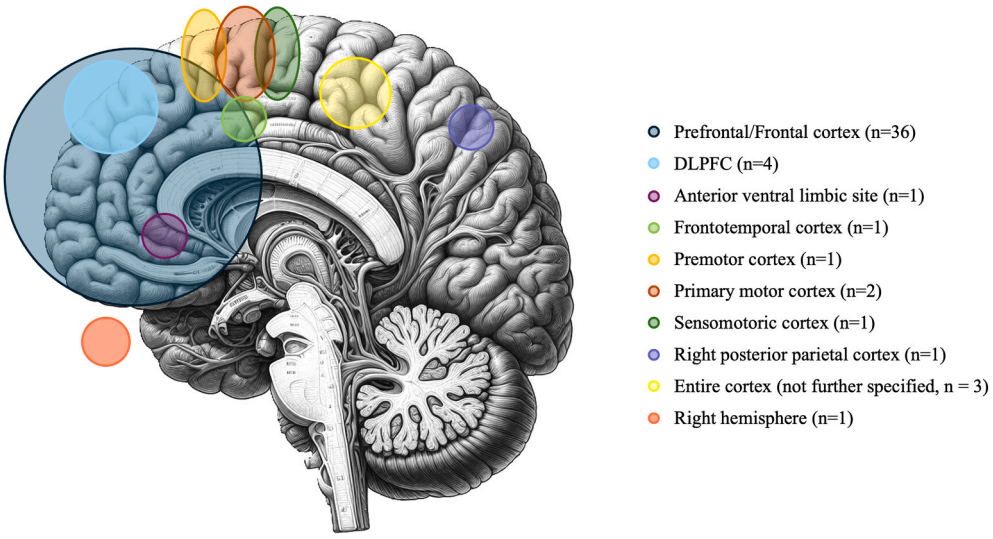


Fig. 2. Representation of the brain areas stimulated with non-invasive electrical brain stimulation. Figure created using *Microsoft Designer's AI Image Generator*.

3.2. Safety and stimulation methods

Details regarding the stimulation characteristics can be found in Table 1 and additionally in Table S1 of the supplementary material. The included references did not consistently include the crucial elements required for reporting in tES studies according to the RATES checklist. Most missing items were details of stimulator brand, electrode shape and size, information about ramping procedures, and safety monitoring. Furthermore, some studies lacked reporting all relevant data on participants, specifically information about handedness and education. However, items relating to electrodes and current characteristics were generally reported and the following observations were made: In 35 studies, brain stimulation was administered during sleep, typically during NREM stage 2 or deeper stages. Of these, stimulation was applied during daytime naps in 6 studies and during nocturnal sleep in 29 studies. Stimulation durations varied widely, ranging from a few seconds to 30 min. The most commonly used protocol (n = 20) consisted of five 5-min stimulation blocks, each separated by a 1-min stimulation-free interval. Single-session protocols were predominant (n = 36), while a subset of studies (n = 8) implemented repeated daily sessions over 2 to 20 consecutive days. In six studies, stimulation was applied more than once during the night or included an additional session on the following morning. One study [53] combined repetitive TMS over the primary motor cortex with tDCS over the prefrontal cortex.

Among the 21 studies utilizing so-tDCS, most adopted the protocol initially introduced by Marshall et al. [100], featuring bifrontal electrode placement with mastoid return electrodes, anodal stimulation at 0.75 Hz, current intensities ranging from 0 to 260 μA, and a variety of waveforms (sinusoidal, trapezoidal, square). Five studies applied so-tDCS at individually adjusted frequencies based on algorithmic determinations [75,77,80,87,88]. In tACS protocols, stimulation intensities ranged from 0.3 to 2.5 mA, with frequencies between 0.75 and 100 Hz, while sinusoidal waveforms were the most used. tDCS protocols generally applied 2 mA stimulation using either anodal or cathodal polarization. To reduce sensation caused by the simulation most studies applied a ramp up/down period lasting a few seconds.

All studies followed the International 10-20 EEG system for electrode montage. In 42 studies, stimulation targeted the frontal or prefrontal cortex, most often the DLPFC, typically employing bilateral frontal electrodes with return electrodes placed at the mastoids. Other targeted regions included the right hemisphere (n = 1 [101]), the primary motor cortex (n = 2 [69,72]), the sensorimotor cortex (n = 1 [84]), the right posterior cortex (n = 1 [97]), the anterior ventral limbic site (n = 1 [66]), and unspecified cortical sites (n = 3 [52,82,85]). An overview of stimulation targets across protocols is provided in Fig. 2.

All controlled studies (n = 49) included sham stimulation as a control condition typically with the same set-up, but without stimulation applied. Other control conditions included stimulation at different frequencies, reverse polarization or other targets.

Adverse events were reported in only 11 studies and were predominantly mild, including skin sensations or headaches. These effects occurred both in stimulation and sham groups, with no significant difference in frequency. No serious adverse events were reported, except for one study [90] that reported a superficial skin lesion due to unintended direct contact between the cathode and anode. Adverse effects were observed in three tDCS protocols with stimulation intensities of ≥1 mA [67,69,90], five so-tDCS protocols (0-260 μA at 0.75 Hz) [56,66,89,91,95], and once each in a STAMP [85] and tACS protocol (1.5 mA at 5 Hz) [73]. A relevant number of studies (n = 25) did not explicitly indicate information regarding safety monitoring.

3.3. Effect of tES on electrophysiological sleep

Key findings on the effects of tES on sleep architecture and EEG spectral power are summarized in Table 2. Given the limited informative value due to the small number of results, variables reported in fewer than 50% of the reviewed studies are not discussed in the following section but are available in the Supplementary Material S2. These include time in bed, arousal index, REM latency, slow-wave sleep latency, spindle events, and the apnea–hypopnea index.

3.3.1. Effect of tES on sleep architecture

Significant changes in TST were reported in only five out of 28 studies (17.9%): Three studies [71,78,92] involving clinical populations reported prolonged TST following prefrontal or frontotemporal tDCS or so-tDCS. In contrast, one study [67] found a decrease of approximately 25 min in TST in healthy participants after anodal tDCS administered before sleep onset, compared to cathodal or sham stimulation, while another article [74] reported a reduction of about 23 min in TST following tACS relative to sham.

Findings on SL were inconsistent: four studies [63,65,83,92] reported a decrease in SL after stimulation, whereas three others [69,80,88] reported prolonged SL in the stimulation group. These conflicting results may reflect heterogeneity in study populations, stimulation modalities, and protocol parameters.

Similarly, findings on SE were heterogenous, with significant effects

Table 2
Significant effects of transcranial electrical brain stimulation on sleep architecture and EEG power spectrum.

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
Li et al. [63]	tDCS	nse	Decrease in SL in stimulation group (27.68 ± 23.45 min before vs. 18.16 ± 12.41 min after treatment, $p = 0.023$).	Increase in SE in stimulation group ($72.82 \pm 13.97\%$ before vs. $79.91 \pm 13.48\%$ after treatment, $p = 0.025$).	nse	nse	nse	nse	n/a
Wang et al. [65]	tACS	nse	Decrease in SL in individualized frequency tACS compared to baseline in insomnia patients (25.26 ± 6.76 min vs 16.92 ± 5.81 min, $p = 0.027$).	Increase in SE in both tACS groups compared to baseline in insomnia ($71.31 \pm 4.9\%$ (baseline) vs. $80.59 \pm 4.65\%$ (NIF-tACS) vs. $84.85 \pm 3.60\%$ (IF-tACS), $p < 0.001$) and subsyndrome insomnia patients ($79.13 \pm 3.57\%$ (baseline) vs. $86.51 \pm 5.07\%$ (NIF-tACS) vs. $88.19 \pm 2.64\%$ (IF-tACS), $p < 0.001$).	Decrease in NREM1 in both tACS groups compared to baseline in insomnia ($9.37 \pm 1.75\%$ (baseline) vs. $4.13 \pm 1.85\%$ (NIF-tACS) vs. $4.73 \pm 2.69\%$ (IF-tACS), $p < 0.001$) and subsyndrome insomnia patients ($8.14 \pm 4.72\%$ (baseline) vs. $4.21 \pm 2.06\%$ (NIF-tACS) vs. $4.23 \pm 3.07\%$ (IF-tACS), $p = 0.004$).	nse	Increase in NREM3 in IF-tACS ($15.24 \pm 2.51\%$) compared to baseline ($10.29 \pm 3.68\%$, $p = 0.003$) and NIF-tACS ($10.58 \pm 3.59\%$, $p = 0.006$).	nse	nse
Thom et al. [72]	tDCS	nse	nse	n/a	nse	nse	nse	nse	nse/n/a
Shi et al. [73]	tACS	nse	nse	nse	nse	nse	nse	nse	Increase in delta power and SWA during NREM3 in tACS group compared to sham ($p < 0.05$). Decrease in alpha power during NREM3 and in sigma and beta power during NREM2 and NREM3 in tACS group compared to sham ($p < 0.05$).
Tabarak et al. [74]	tACS	Decrease in TST in tACS condition (411.88 ± 48.42 min) compared to sham (434.20 ± 44.36 min), $p < 0.001$.	nse	n/a	nse	Decrease in NREM2 in tACS condition (187.78 ± 68.31 min) compared to sham (219.62 ± 51.86), $p < 0.001$.	nse	nse	Decrease in SO-spindle coupling signals in tACS condition compared to sham ($p < 0.05$).
Ladenbauer et al. [75]	so-tDCS	nse	n/a	n/a	nse	nse	nse	nse	Increased coupling of sleep spindles with SO in 0.75 Hz so-tDCS ($p < 0.05$) vs. no effect in eigenfrequency stimulation.
Yu et al. [78]	tDCS	Prolonged TST (95% CI: 14.387-283.527,	n/a	n/a	nse	Prolonged NREM2 (95% CI 3.16-246.17,	nse	nse	Increase in alpha, beta and gamma power ($p = 0.005$) in VS

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
		$p = 0.013$) in the VS group with sleep cycles.				$p = 0.04$) in the VS group with sleep cycles.			group with sleep cycles after tDCS compared to VS group without sleep cycles. Increased coherence in all frequency bands (δ, θ, α, β, γ) in VS patients with sleep cycles after a 10 d tDCS ($p < 0.05$).
Robert et al. [77]	tACS	nse	nse	nse	n/a	n/a	n/a	n/a	n/a
Zhou et al. [53]	tDCS	Increase in TST after 10 d (431 min), 20 d (502.5 min), and 50 d (521 min) compared to baseline.[a]	Decrease in SL after 10 d (35.9 min), 20 d (21.5 min) and 50 d (27 min) compared to baseline (41 min).[a]	Increase in SE after 10 d (67.8%), 20 d (82.9%) and 50 d (87.7%) compared to baseline (65.4%).[a]	Decrease in percentage of NREM1 after 10 d (5.5%), 20 d (10%), and 50 d (5.4%) compared to baseline (15.1%).[a]	Increase in percentage of NREM2 after 10 d (80.9%) and 50 d (81.7%) compared to baseline (70.2%).[a]	Decrease in percentage of NREM3 after 10 d (1.4%), 20 d (2.2%), and 50 d (4.7%) compared to baseline (6.1%).[a]	Increase in percentage of REM after 10d (12.3%) and 20d (21.9%) compared to baseline (8.62%).[a]	n/a
Li et al. [76]	tDCS	nse	nse	n/a	nse	nse	nse	nse	n/a
Motamedi et al. [64]	tACS	n/a	nse	nse	nse	nse	nse	nse	n/a
Xie et al. [83]	tACS	n/a	Sleep onset process is accelerated in stimulation group.	n/a	nse	Higher amount of NREM2 during 15 min daytime nap in stimulation group compared to sham.[a]	Higher amount of NREM3 during 15 min daytime nap in stimulation group compared to sham.[a]	n/a	Higher increase of delta-theta power (0.5 -7 Hz) in stimulation group compared to sham, particularly during NREM3 ($p \leq 0.05$). Higher alpha (8-12 Hz) and sigma activity (12-16 Hz) in stimulation group compared to sham ($p \leq 0.05$). Higher beta activity in the sham group compared to the stimulation group ($p \leq 0.05$).
Dehnavi et al. [80]	so-tDCS	nse	Longer SL in sham (18.7 ± 2.1 min) and stimulation condition (19.6 ± 2.3 min) compared to control condition (11.5 ± 1.1 min, $p < 0.05$).	nse	nse	nse	nse	nse	Increase in fast spindle power ($p = 0.013$) and fast spindle density (per 30 s, $p = 0.047$) in stimulation condition compared to sham during 150 s of poststimulation NREM3 sleep.
Hathaway et al. [81]	tACS	nse	n/a	n/a	nse	nse	Increase in mean and percent time spent in NREM3 during stimulation night (63.75 min/13.2%) than during sham	nse	nse

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
							night (56.35 min/ 11.5%, $p < 0.05$).		
Noreika et al. [84]	tDCS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Decrease in interhemispheric coherence in high beta range (21-31 Hz) between parietal electrodes P3-P4 during a 1 min stimulation-free period before awakening ($p = 0.01$, uncorrected).
Zhou et al. [71]	tDCS	Increase in TST on day 28 compared to day 1 in both sham and stimulation group, higher difference in stimulation group (87.5 ± 70.5 min vs. 52.05 ± 69.9 min, $p = 0.016$).	nse	Increase of SE after treatment in stimulation group (73.04 ± 6.98 min before vs. 81.26 ± 12.71 min after treatment, $p < 0.001$).	nse	nse	nse	nse	n/a
D'Atri et al. [86]	tACS	nse	nse	n/a	nse	nse	nse	nse	Increase in SWA (0.5-4.75 Hz) in the frontal, central and parietal sites in stimulation group compared to sham ($p = 0.037$).
Charest et al. [79]	tDCS	nse	nse	nse	nse	nse	nse	nse	n/a
Bueno-Lopez et al. [39]	so-tDCS	nse	n/a	n/a	nse	nse	nse	nse	nse
Frase et al. [67]	tDCS	nse	nse	nse	n/a	nse	nse	nse	Slight decrease in frontal delta power (0.1-1.5 Hz) with anodal and cathodal stimulation compared to sham ($p = 0.036$).
Ketz et al. [87]	tACS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Increase in 0.5-4.7 Hz power in stimulation group compared to sham during first 5 s poststimulation ($p = 0.036$). Marginally increase in coupling between SW-band and amplitude of high spindles with stimulation ($p = 0.05$). Decrease in 0.5-47.2 Hz power in stimulation group compared to sham from 5 to 10 s

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
									poststimulation ($p < 0.05$).
Koo et al. [88]	so-tDCS	nse	Longer SL in both stimulation condition (19.6 ± 2.3 min) and sham (18.7 ± 2.1 min) compared to control (11.5 ± 1.1 min, $p < 0.05$).	nse	nse	nse	nse	n/a	Increase in centroparietal fast spindle fast spindle power in stimulation group as compared to sham or control group ($p < 0.05$).
Lafon et al. [52]	tACS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	nse
Ladenbauer et al. [89]	so-tDCS	nse	n/a	n/a	nse	nse	nse	nse	Increase in SO power in stimulation condition (frontal $p < 0.001$, centroparietal $p = 0.006$). Increase in slow spindle power in frontal areas in stimulation condition ($p = 0.001$) Increase in fast spindle power (frontal $p < 0.001$, centroparietal $p = 0.041$) in stimulation group compared to sham. Increase in centroparietal fast spindle power during the SO up-phase (phase amplitude coupling) ($p < 0.05$)
Lustenberger et al. [70]	tACS	nse	nse	nse	nse	nse	nse	nse	Decrease in theta power during NREM2 and NREM3 in stimulation condition compared to sham ($p < 0.05$). Increase in spindle activity (11-16 Hz) in NREM2 in stimulation condition compared to sham ($p < 0.05$). Increase specifically in the fast spindle frequencies 15-16 Hz in the motor memory responders compared to non-responders ($p < 0.05$).

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
Ladenbauer et al. [91]	so-tDCS	nse	n/a	nse	nse	nse	nse	nse	Increase in SO power in stimulation condition compared to sham during 5 × 1 min poststimulation blocks ($p = 0.029$). Increase in fast spindle power in stimulation condition compared to sham in frontal areas ($p = 0.003$) and strong trend in centroparietal areas ($p = 0.054$).
Frase et al. [90]	tDCS	Decrease in TST (25 min) in anodal (387.4 ± 44.5 min) compared to cathodal stimulation (414 ± 28.3 min) and sham (412.6 ± 27.7 min, $p = 0.017$). Decrease in TST for second quarter (2h intervals) of the night ($p = 0.021$).	nse	Decrease in SE in anodal stimulation (81 ± 9.3%) compared to cathodal stimulation (86.3 ± 5.7%) and sham (86.3 ± 6%, $p = 0.014$).	n/a	nse	nse	nse	Higher beta power (16-24 Hz) in cathodal stimulation compared to sham ($p = 0.012$) and anodal stimulation ($p = 0.021$).
Westerberg et al. [94]	so-tDCS	n/a	nse	n/a	nse	nse	nse	nse	Significant increase in the frontal SO power (0.5-1 Hz) in stimulation condition during the 1 min-off-intervals ($p < 0.05$).
Saebipour et al. [66]	so-tDCS	nse	nse	n/a	Decrease in NREM1 in stimulation condition compared to sham (difference 22 ± 17.7 min, $p = 0.028$).	Decrease in probability of transition from NREM2 to wake ($p \leq 0.01$).	Increase in NREM3 in stimulation condition compared to sham (difference 33 min ± 26 min longer, $p = 0.026$).	nse	Increase in average power at frequencies <1 Hz in stimulation condition compared to sham during the first 1 min EEG after stimulation ($p \leq 0.05$).
Del Felice et al. [92]	so-tDCS	Increase in TST in stimulation condition (54.6 ± 5.8 min) compared to sham (41.9 ± 6.1 min, $p = 0.01$).	Reduced SL in stimulation condition (13 ± 5.8 min) compared to sham (18 ± 6.1 min, $p = 0.01$).	n/a	nse	nse	nse	nse	n/a
Sahlem et al. [40]	so-tDCS	nse	n/a	nse	nse	nse	Marginal increase in NREM3 during first hour following stimulation compared to sham (10.1 ± 7.1 min vs. 6.1 ± 4.3 min, $p = 0.04$)	nse	nse
Garside et al. [93]	tACS	nse	nse	n/a	nse	nse	nse	n/a	Reduction of delta power (1-4 Hz) in stimulation group

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
									compared to sham ($p < 0.05$).
Prehn-Kristensen et al. [68]	so-tDCS	nse	nse	nse	nse	nse	nse	nse	Enhancement of SWA in stimulation group compared to sham ($p = 0.02$).
Eggert et al. [95]	so-tDCS	nse	n/a	n/a	nse	nse	Decrease in NREM3 in stimulation condition during the 5×1 min stimulation free interval (38.7±7 s vs. 64.3 ± 12.8 s, $p = 0.03$)	nse	Reduction of delta power at F7 ($p = 0.008$) and T3 ($p = 0.033$) in stimulation condition compared to sham.
Göder et al. [96]	so-tDCS	nse	nse	nse	nse	nse	nse	nse	n/a
Jakobson et al. [97]	tDCS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	nse
Marshall et al. [98]	so-tDCS	nse	nse	n/a	nse	Increase in NREM2 during stimulation periods compared to sham (202.8 ± 10.2 s vs. 132.0 ± 14.3 s, $p < 0.01$) Decrease in NREM2 during 30min interval after stimulation compared to sham ($29.3 \pm 2.0\%$ vs. $37.93 \pm 1.47\%$, $p < 0.001$).	Decrease in NREM3 during stimulation periods (80.4 ± 8.6 s vs. 146 ± 12.6 s, $p < 0.01$) Increase in NREM3 during 30min interval after stimulation compared to sham ($51.86 \pm 1.81\%$ vs. $46.8 \pm 1.45\%$, $p < 0.001$).	nse	NREM Theta -tDCS decreased delta frequency band within 5x1-min stimulation free intervals and during first 30 min interval following tDCS ($p < 0.001$). NREM theta -tDCS decreased slow spindle EEG frequency band within 5x1-min stimulation free intervals at Fz ($p = 0.002$). REM theta-tDCS increased beta (25-45 Hz) EEG power ($p = 0.053$). REM theta-tDCS increased gamma (25-45 Hz) EEG power across all electrodes ($p < 0.001$).
Roizenblatt et al. [69]	tDCS	nse	Increase in SL after tDCS over DLPFC by 133.4% compared to pre-tDCS ($p = 0.02$). No significant increase in sham stimulation or tDCS over M1.	Improved SE after M1 stimulation by 11.8% ($p = 0.004$). Decreased SE after DLPFC stimulation by 7.5% ($p = 0.02$).	nse	nse	nse	nse	Decrease in delta frequency band by 41.7% after DLPFC ($p = 0.009$) stimulation. Increase in delta frequency after M1 stimulation by 26.9% ($p = 0.01$). Increase in alpha frequency after DLPFC stimulation by 13% ($p = 0.005$). Alpha/

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
									delta index increased after M1 stimulation ($p = 0.001$) and decreased after DLPFC stimulation ($p = 0.04$).
Marshall et al. [42]	tDCS	nse	nse	n/a	n/a	n/a	n/a	n/a	Increase in power in frequency range <1 Hz in stimulation condition during the 15 s simulation-free intervals over parietal cortex ($p < 0.01$). TDCS suppressed theta and lower alpha activity (4-10 Hz) during periods of SWS ($p < 0.01$) and lower beta activity (15-20 Hz) during periods of NREM2 sleep ($p = <0.01$) in central and parietal leads.
Wang et al. [41]	tDCS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Higher delta power (0-4 Hz) in tES group, mainly in frontal and central regions ($p < 0.05$).
Nitsche et al. [99]	tDCS	nse	nse	n/a	nse	nse	nse	Increase in REM density (REM/30 s) in anodal tDCS during second and third REM period, decreased REM density in the cathodal tDCS during second REM period compared to sham, $p < 0.05$.	n/a
Hubbard et al. [82]	STAMPS	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Increase in EEG coherence in theta, alpha and fast spindle band in the stimulation group compared to the sham group ($p < 0.05$) Significant difference in brain connectivity in theta band spindle band ($p < 0.05$).
Pilly et al. [85]	STAMPS	n/a	n/a	nse	nse	nse	nse	nse	Lower spectral power in the slow spindle band in the second

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
									sham night compared to active night ($p = 0.025$).
Marshall et al. [100]	so-tDCS	nse	n/a	n/a	nse	nse	Increase in SWS in so-tDCS (170.8 ± 17.8 s) compared to sham (124.6 ± 19.2 s) during the 1-min-stimulation-free-intervals ($p < 0.05$)	nse	Increase in slow-wave power (0.5-1 Hz) with a maximum at the frontocentral recording site and in the slow spindle power at the frontal and parietal recording site during the five 1-min-stimulation-free intervals ($p < 0.01$).
Passmann et al. [56]	so-tDCS	n/a	n/a	n/a	nse	nse	Decrease in percent of NREM4 over the entire night in so-tDCS condition ($1.0 \pm 1.7\%$) compared to sham ($1.6 \pm 2.3\%$, $p = 0.046$, uncorrected)	nse	Higher SWA after so-tDCS compared to sham for prefrontal and frontal sites during 1-min-stimulation-free-epochs ($p \leq 0.039$) Delayed stimulation-induced changes (1-min interval 60 min poststimulation) in SWA at prefrontal site. Enhanced slow spindle power during the five 1-min-stimulation-free intervals but not during the 1 min interval 60 min poststimulation ($p \leq 0.039$).
Munz et al. [57]	so-tDCS	nse	n/a	nse	nse	nse	nse	nse	Increase in SO power over all electrodes in the 1-min-stimulation-free-intervals in the stimulation condition compared to sham during NREM4 ($p = 0.008$).
Antonenko et al. [58]	so-tDCS	nse	nse	nse	nse	nse	nse	nse	Increase in SWA (0.5-4 Hz) in stimulation condition in the first three stimulation-free intervals ($p < 0.05$), effect disappeared during subsequent post-stimulation intervals. Phase-coupling to the so-tDCS signal covering both the low

(*continued on next page*)

Table 2 (*continued*)

Study	Type of tES	Total Sleep Time	Sleep Latency	Sleep Efficiency	NREM 1	NREM 2	NREM 3	REM	Power Spectrum
									and high spindle frequency bands during the stimulation ($p < 0.05$).
Cellini et al. [59]	so-tDCS	nse	nse	nse	Decrease in NREM1 in stimulation condition ($9.10 \pm 6.35\%$) compared to sham ($20.27 \pm 10.17\%$, $p < 0.01$).	nse	Increase NREM3 in stimulation condition ($19.30 \pm 13.88\%$) compared to sham ($37.94 \pm 18.37\%$, $p < 0.001$).	nse	Enhancement of SO power around 0.75 Hz in stimulation condition especially over frontal channels ($p < 0.05$).
Johnson et al. [61]	so-tDCS	nse	n/a	Decrease in SE in 0.75 Hz stimulation condition compared ($84.19 \pm 2.55\%$) to sham ($90.91 \pm 1.15\%$) and 5 Hz stimulation condition ($90.04 \pm 1.64\%$, $p = 0.03$).	nse	nse	nse	nse	nse
Voss et al. [60]	tACS	nse	nse	nse	nse	nse	nse	nse	Increase in lower gamma frequency band during stimulation with 40 Hz compared to sham or control conditions ($p < 0.001$)
Reato et al. [62]	so-tDCS	n/a	n/a	n/a	nse	nse	nse	nse	Reduced decay of SWS (0.5-1 Hz) and wider band of SWA (0.5-4 Hz) during the night (4.5h analysis window) after stimulation compared to sham ($p = 0.001$ resp. $p < 0.05$).

Abbreviations: n/a = not applicable, nse = no significant effects, TST = total sleep time, SE = sleep efficiency, SL = sleep latency, NREM = non-rapid eye movement sleep, tES = transcranial electrical stimulation, tACS = transcranial alternating current stimulation, (so-)tDCS = (slow oscillatory) transcranial direct current stimulation, TMS = transcranial magnetic stimulation, STAMPS = spatiotemporal amplitude-modulated patterns, SWS = slow-wave sleep, SO = slow oscillations, SWA = slow-wave activity, EEG = electroencephalogram, DLPFC = dorsolateral prefrontal cortex, M1 = primary motor cortex.

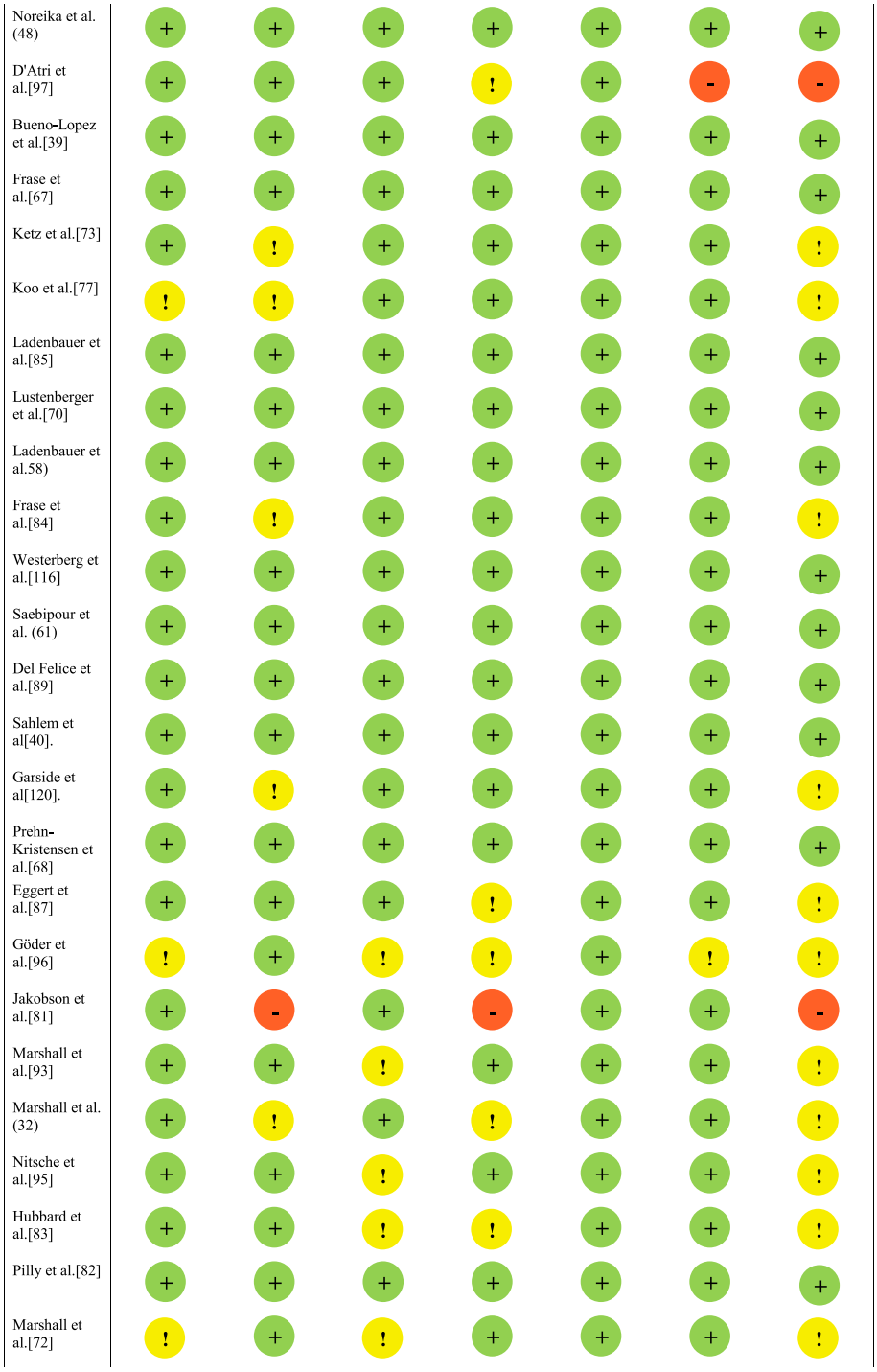
[a] Test for statistical significance not indicated or not performed.

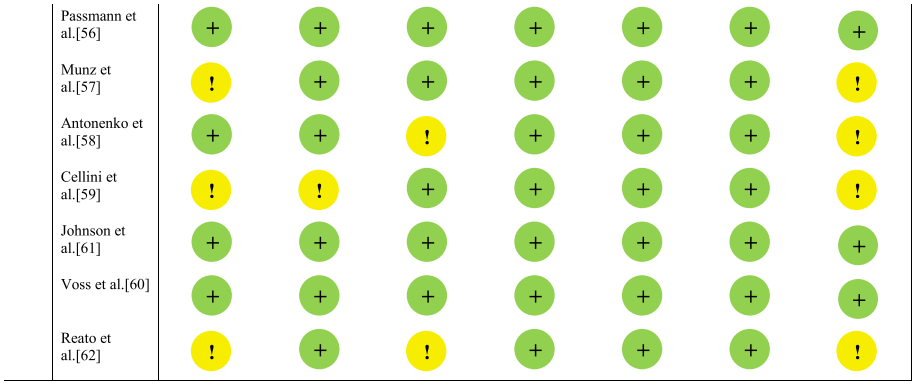
observed only in six of 24 studies [63,65,69,71,90,101]. For example, Roizenblatt et al. [69] reported an 18% increase in SE following tDCS over the primary motor cortex, but a 7.5% decrease after DLPFC stimulation in fibromyalgia patients. In the study with the largest sample size (N = 90) [71], SE and TST increased in both sham and active tDCS groups, with greater improvements in the active stimulation group. Authors attributed the improvements in the sham group to the concurrent antidepressant medication, while emphasizing the superiority of tDCS in enhancing sleep outcomes. Interestingly, Wang et al. [65], who applied tACS at individualized versus non-individualized but sleep stage-adapted frequencies, observed an increase in SE in both groups, but a decrease in SL only after individualized frequency tACS in insomnia patients compared to baseline.

Across the 42 studies investigating the impact of tES on sleep architecture in clinical (n = 14) and healthy (n = 28) populations, findings on sleep stage distribution revealed certain trends despite considerable heterogeneity. All studies [59,65,66] reporting a significant effect of tES on NREM1 sleep observed a decrease. Two studies [59,66] applied (so-) tDCS over frontal/prefrontal regions, while one [65] used tACS with sleep stage-dependent frequencies and stimulation sites. Regarding NREM2 sleep, except from one study [74] reporting a decrease after 60 Hz tACS over bifrontal cortex, all other studies [66,78,83,98] found increased NREM2 duration in the stimulation condition. In contrast, findings on NREM3 were inconsistent: Seven studies [40,59,65,66,81,83,100] reported an increase, while three studies [56,95,98] found a decrease. Despite targeting similar frontal regions, stimulation protocols varied widely in frequency (0.5-5 Hz), intensity (0.26-2 mA), session timing (daytime vs. nocturnal) and duration (few seconds to 20 min). For instance: Xie et al. [83] applied 5 Hz tACS (1.4 mA) during a nap, resulting in increased amount of NREM2 and NREM3. Hathaway et al. [81] used 0.5 Hz tACS (0.52 mA, 5 × 5 minutes) at night, finding an isolated increase in NREM3. Repeated session of tDCS with 2 mA led to an increase in NREM2 in patients in a vegetative state [78]. Cellini et al. [59] reported increased NREM3 and decreased NREM1 during a 90-min nap using 0.75 Hz so-tDCS (2 mA). The same effects were observed in the study by Wang et al. [65], which applied nocturnal tACS at sleep stage-dependent frequencies. Marshall et al. [100] found that nocturnal so-tDCS (0.75 Hz, bifrontal montage) increased slow-wave sleep in

Table 3
Risk of Bias Assessment Green (+) circles represent low risk of bias, red (−) circles high risk of bias and yellow (!) circles raising some concerns in risk of bias.

	Study	Randomisation process	Period and carryover effects	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall
Parallel design	Li et al [63]	+		+	+	+	+	+
	Yu et al.[90]	+		+	+	+	+	+
	Robert et al.[76]	!		!	-	+	+	-
	Li et al. [118]	+		!	+	+	+	!
	Zhou et al.[71]	+		+	+	+	+	+
	Charest et al.[119]	+		+	+	+	+	+
	Lafon et al.[52]	-		+	+	+	!	-
	Roizenblatt et al.[69]	+		+	+	+	+	+
	Wang et al.[41]	+		!	!	+	+	!
Crossover design	Wang et al. [65]	+	+	+	+	+	+	+
	Thom et al. [79]	+	+	!	+	+	+	!
	Shi et al. [88]	+	+	+	+	+	+	+
	Tabarak et al. [91]	+	+	+	!	+	+	!
	Ladenbauer et al. (39)	+	!	+	!	+	!	!
	Motamedi et al.[64]	+	+	+	+	+	+	+
	Xie et al.[92]	+	+	+	+	+	!	!
	Dehnavi et al.[74]	+	+	+	+	+	+	+
	Hathaway et al.[94]	+	+	+	!	+	+	!


Noreika et al. (48)
D'Atri et al.[97]
Bueno-Lopez et al.[39]
Frase et al.[67]
Ketz et al.[73]
Koo et al.[77]
Ladenbauer et al.[85]
Lustenberger et al.[70]
Ladenbauer et al.58)
Frase et al.[84]
Westerberg et al.[116]
Saebipour et al. (61)
Del Felice et al.[89]
Sahlem et al[40].
Garside et al[120].
Prehn-Kristensen et al.[68]
Eggert et al.[87]
Göder et al.[96]
Jakobson et al.[81]
Marshall et al.[93]
Marshall et al. (32)
Nitsche et al.[95]
Hubbard et al.[83]
Pilly et al.[82]
Marshall et al.[72]



young adults. Using the same protocol, Saebipour et al. [66] found similar effects in insomnia patients – reduced NREM1 and increased NREM3 – along with fewer transitions from NREM2 to wake. Conversely, two studies [56,95] applying the same protocol in elderly participants reported opposite effects showing both a decrease in deep sleep after anodal so-tDCS.

Regarding REM sleep, only two of 38 studies reported significant findings; Nitsche et al. [99] observed increased REM density after tDCS over the DLPFC during REM sleep. Zhou et al. [53] noted a trend toward increased REM percentage and SE after combined TMS and tDCS in a fibromyalgia patient.

3.3.2. *Effect of tES on power spectrum*

Power spectral analysis of sleep EEG was performed in 40 of 51 studies (78.4%). Almost all studies (n = 15) showing significant effects and applying 0.75 Hz so-tDCS or tACS at frequencies $\leq$ 5Hz reported increased slow wave and delta activity. In contrast, a few studies using tACS or so-tDCS at higher frequencies [70,74,98] observed reduced slow-wave and/or delta power, but sometimes increased power in higher frequency bands (beta, gamma) [78,90,98]. Application of tDCS at different intensities led to a decrease in slow-wave power in one study [67] and an increase in two studies [41,42]. Among studies applying closed-loop individual frequency slow-wave tACS or so-tDCS, results differed considerably: Ketz et al. [87] reported short-lasting increase in 0.5-4.7 Hz power after individual-frequency tACS (0.5-1.2 Hz). Dehnavi et al. [80] observed increased fast spindle power and density, while Koo et al. [88] found elevated fast spindle power only in centroparietal regions. In contrast, Robert et al. [77] found no significant spectral changes. Ladenbauer et al. [75] observed only increased coupling between sleep spindles and slow oscillation after 0.75 Hz so-tDCS, but not after stimulation at individual frequencies.

Using the Marshall protocol (so-tDCS, 0.75 Hz, 0-260 μA, 5 × 5 minutes), several studies [58,75,89,91] found enhancements in slow-wave power and spindle activity during stimulation-free intervals - particularly in frontal areas and in older adults or in patients with mild cognitive impairment. Additionally, Roizenblatt et al. [69] reported delta entrainment after tDCS over the motor cortex, but not over the DLPFC, in fibromyalgia patients. Two studies applying STAMPs (40 Hz, 150 μA – 1.5 mA) [82,85] found increased EEG power and coherence in the slow-spindle band.

3.4. *Risk of bias assessment*

Risk of bias was assessed for 50 of the 51 included studies. One article [53] was a case report and was excluded from the assessment. 24 studies (48%) were rated as having an overall low risk of bias, while 22 studies (44%) raised some concerns, and four studies (8%) were rated as having a high risk of bias.

Notably, randomization-related bias was observed in eight studies [52,57,61,62,77,88,96,100]. Among crossover designs (n = 41), most studies (n = 33, 80.5%) demonstrated low risk of bias due to period and carryover effects. Except for one study [97], in which all conditions were tested in a single night, all other crossover trials maintained appropriated washout periods – typically 1-2 weeks – between conditions. A lack of blinding among participants, caregivers, or outcome assessors was frequently observed in studies categorized as having "some concerns" due to deviations from intended interventions. Bias due to missing outcome data was present in 11 studies, often exacerbated by small sample sizes. In two of these studies [77,97], participant dropouts occurred for reasons likely to have affected the results, leading to a high risk of attrition bias. All studies used objective sleep measures – either PSG, standard/high-density EEG, or intracranial EEG – as required by the inclusion criteria. As such, none of the studies were found to have a risk of bias in the "measurement of the outcome" domain. However, reporting bias was a concern in five studies [52,75,77,86,96], where significant deviations from pre-specified analysis plans were identified. Although not formally assessed by the Cochrane RoB 2 framework, limited sex representativeness was identified: five studies [41,42,57,68,70] included only male participants, where one study [69] examined only female participants. These gender imbalances may limit the generalizability of findings. A detailed risk of bias assessment by domain and study is presented in Table 3.

Reviewed articles, using a substantial variability of stimulation protocols, reported heterogeneous effects of tES on electrophysiological sleep parameters. Nevertheless, tES was associated with a reduction in NREM1 and increases in total sleep time, NREM2 and NREM3. Moreover, slow-oscillatory tES showed the capacity to entrain endogenous slow oscillations during sleep. However, a minority of studies yielded statistically significant results, and more than 50% of the studies showed considerable risk of bias in the RoB2 assessment.

4. Discussion

Interest in non-invasive transcranial electrical stimulation (tES) has grown considerably in recent years, with researchers exploring its effects across various populations and conditions [5,10]. Given the restorative role of sleep in brain processes such as memory consolidation, numerous studies have investigated the impact of tES on neurophysiological sleep - particularly on slow-wave sleep and slow oscillatory activity, which are critically involved in memory processing [81,83,89,90,102]. The

present review demonstrates that tES can modulate brain oscillations during sleep; however, significant changes in sleep architecture were observed in only a minority of the studies (25.6%). Below, we discuss these findings in detail and outline future directions.

4.1. Safety and stimulation methods

tES is generally considered a safe method for modulating neuronal network excitability and brain oscillations. Reported adverse effects were mostly mild (e.g. tingling, itching, or headaches) and occurred equally in sham and active stimulation conditions [56,66,89,91,95], supporting a favorable safety profile. However, as a relevant number of studies (n = 25) did not explicitly mention safety monitoring, the absence of adverse events can only be assumed.

To date, no standardized stimulation protocol of tES has been established. With the application of the RATES checklist we tended to enhance comparability of the included study. Although several studies did not comprehensively report all participant and stimulation parameters recommended by the RATES checklist, thereby limiting transparency, comparability and reproducibility of the studies, parameters related to electrodes and current characteristics were generally indicated. Stimulation protocols of the included references varied widely across studies and were strongly influenced by factors such as current intensity, session duration, polarity, frequency (especially in tACS protocols), waveform, and repetition rate [46,103,104]. Moreover, important elements, including electrode shape and size, stimulator brand, and ramping procedures, were not consistently reported, despite their potential effectiveness of tES. Overall, this heterogeneity in stimulation protocols likely contributed to the divergent findings.

While short tDCS sessions can modulate cortical excitability, longer applications appear necessary to produce lasting effects [12,75,105,106]. Most tDCS studies employed intensities of 1-2 mA; higher intensities (>4 mA) increase the risk of adverse effects, while lower doses may be ineffective [103,107]. Non-linear dose-response relationships have been suggested, potentially due to variations in neurotransmitter receptor and ion channel responsiveness [77,107,108]. For instance, Jones et al. [103] and Robert et al. [77] observed an inverted U-shaped effect of tACS on memory performance, suggesting that overstimulation may reduce efficacy. Batsikadze et al. [104] showed that polarity-specific effects of tDCS may reverse at higher intensities: while 1 mA tDCS induces classical anodal-cathodal effects, 2 mA may enhance excitability under both electrodes.

Most studies targeted the prefrontal or frontal cortex, aiming to influence the top-down pathway in sleep regulation by modulating cortical excitability or oscillatory dynamics [33,35–38]. Although tES primarily affects superficial cortical areas, some evidence suggests that stimulation of prefrontal or temporal regions can also modulate deeper structures, such as the ventral striatum, hippocampus, or reticular formation – key regions in sleep-wake regulation [109–111]. Emerging approaches even explore stimulation of non-cortical sites, such as the cerebellum or spinal cord, broadening the scope of NIBS applications [112–114].

4.2. Effect of tES on sleep architecture and endogenous brain oscillation during sleep

While tES did not consistently alter sleep architecture, several studies reported trends toward increased TST, NREM2, and NREM3 following stimulation. TST prolongation was primarily associated with tDCS [71,78,92]. Increases in NREM3 were exclusively observed in studies using time-varying stimulation frontal/prefrontal areas at ≤ 5 Hz, suggesting that low-frequency stimulation promotes slow-wave sleep. However, three studies [56,95,98] using similar protocols reported reduced NREM3, and one of these [98] reported an increase in NREM2 instead. These contradictory findings could not be explained by differences in the stimulation protocol or study population.

Findings for NREM1 and NREM2 were more consistent: NREM1 tended to decrease and NREM2 increased following tES, regardless of stimulation type [53,66,78,83,98,115]. These changes likely reflect the common goal of most protocols – to promote deep sleep by enhancing slow oscillatory activity. However, due to the combination of repetitive TMS and tDCS in the case report from Zhou et al. [53] effects on sleep architecture cannot be attributed to tDCS alone.

Time-varying tES techniques (tACS or so-tDCS) appear to enhance cortical oscillations at or near the stimulation frequency, a phenomenon known as resonance [46,60,70]. Stimulation efficacy may depend on the alignment between external stimulation and intrinsic brain rhythms. For example, Marshall et al. [100] observed increased slow oscillations when so-tDCS was applied early in the night, while Voss et al. [60] reported enhanced gamma activity during REM sleep following 40 Hz stimulation, but not with lower frequencies. Studies by Ladenbauer et al. [89] and Antonenko et al. [58] indicate that so-tDCS can enhance both slow oscillation and spindle activity, as well as their functional coupling – an interaction essential for memory consolidation. Findings of Tabarak et al. [74] support the assumption that the increase in slow oscillations and SO-spindle coupling depends on the stimulation frequency of so-tDCS or tACS, as 60 Hz stimulation resulted in a reduction of SO-spindle coupling. However, studies using closed-loop algorithms tailored to individual slow-wave frequencies did not outperform standard 0.75 Hz so-tDCS [65,75,77,80,87,88].

Results of tDCS on EEG spectral power were more variable. Cathodal tDCS has been associated with reduced beta/gamma activity and increased theta/delta activity, while anodal tDCS typically enhances beta power and reduces delta, theta, and alpha power [12,116–118]. Some studies, however, reported increased slow-wave activity during sleep following anodal stimulation [41,42,78,84,86], with regional effects differing by target area (e.g. DLPFC vs. motor cortex) and timing (daytime vs. nocturnal stimulation) [69].

The responsiveness to tES depends not only on the stimulation parameters and session duration but also on the number of sessions, the timing of stimulation, other device-related factors - such as the montage, placement accuracy and properties of the electrodes -, and anatomical factors of individuals. Even small differences in these parameters can substantially influence the effective current distribution and neurophysiological outcomes [1,10,103,106]. Consequently, standardization of stimulation procedures and control for participant-specific variables is crucial to enhance reproducibility and interpretability of tES effects on sleep.

Sleep architecture and continuity undergo significant alterations with aging, characterized by reduced total sleep time and efficiency, a decline in slow-wave and REM sleep, and a shift toward lighter sleep stages [16,119]. Although age-related differences in responsiveness to tES seem plausible, reviewed studies did not reveal consistent distinctions in younger and older participants, with most studies focusing on a population < 35 years. Further participant-related factors - such as sex, baseline sleep quality, medication use and circadian preference - were heterogeneous among reviewed study and contribute to the limited analysis and comparability of the results [1,2,10,106]. In several studies where sleep was not the primary outcome, authors often examined effects of tES on memory consolidation. While Marshall et al. [100] demonstrated improved SO activity and memory retention in young adults after 0.75 Hz so-tDCS, Eggert et al. [95] did not replicate these

effects in older adults using a sinusoidal version of the same stimulation protocol. In contrast, Ladenbauer et al. [91] and Westerberg et al. [94] reported beneficial effects of so-tDCS on SO activity, spindle power, and memory in older adults. These findings underline the need for well-powered trials, focusing on aging and clinical population to assess the cognitive and potentially therapeutic effects of tES.

Our findings algin with previous reviews showing limited effects on sleep architecture but more consistent modulation of brain oscillations and subjective sleep ratings [14,45]. These effects have been attributed to the cortical origin of slow waves, especially in prefrontal and motor regions [45,120], as well as potential involvement of deeper structures like the reticular formation [110]. The mismatch between subjective improvement and absence of objective EEG changes may reflect stimulation effects in regions not easily captured by scalp EEG – highlighting the value of intracranial EEG in future studies.

4.2.1. *Methodological quality and bias*

In many cases, a risk of bias was identified due to study dropouts, either because the reasons for these dropouts were not stated, or because they may have influenced the results. Given the low sample size in most of the studies, missing outcome data is more likely to influence the results. In addition, substantial differences in or missing baseline information of the allocation groups contribute to potential bias. Authors should take appropriate measures and tests to avoid deviations from attended interventions and the influence of possible confounders.

4.3. *Strengths, limitations and future perspectives*

A major strength of this systematic review is its focus on objectively measured electrophysiological sleep outcomes, distinguishing it from previous reviews that relied also on subjective sleep assessments [14,45]. The inclusion of both healthy and clinical populations further enhances the generalizability of findings. However, the main limitation of the reviewed studies and this review lies in the substantial heterogeneity in stimulation protocols, study populations, and assessed sleep parameters. The lack of standardized stimulation protocols and variations in device- and participant-related factors, as assessed using the RATES checklist, likely contributed to inconsistent findings and limit the interpretability and comparability of results, making a quantitative analysis unfeasible. The inclusion of an uncontrolled study and a case report represents an additional limitation, but provides hypothesis-generating insights. In addition, most studies lacked long-term follow-up, preventing conclusions about sustained effects. Only a minority of the reviewed studies reported statistically significant outcomes, and many studies were underpowered due to small sample sizes. Moreover, the risk of bias was considerable in over half of the included study, particularly related to randomization procedures and missing outcome data. Consequently, due to the lack of quantitative analysis and the heterogeneity of the included studies, some of the results are indicative, but do not allow conclusive statements.

Despite these limitations, non-invasive electrical brain stimulation - particularly slow-oscillatory protocols - remains a promising and safe approach to modulate cortical activity, with potential benefits for sleep, sleep-related disturbances in neurological and psychiatric disorders and memory-related processes. However, the current lack of clear evidence for the effects of tES on electrophysiological sleep highlights the need for larger-scaled studies with robust methodological designs, standardized stimulation procedures, and control for participant-specific variables to enhance reproducibility and interpretability. Authors should consequently employ the RATES checklist for planning and reporting tES studies and quantitative synthesis of electrophysiological outcomes should be integrated into future research to improve comparability across studies. Determining the optimal stimulation parameters – frequency, intensity, timing, duration, and anatomical target – as well as identifying biological and demographic predictors of stimulation responsiveness will be essential to refine and individualize stimulation

Practice Points

- Transcranial electrical stimulation (tES) is a safe method for modulating cortical brain activity in both healthy individuals and clinical populations.
- tES shows heterogeneous effects on electrophysiological sleep architecture, with a trend toward increased total sleep time and enhancement of deeper sleep stages.
- Slow-oscillatory stimulation protocols can entrain endogenous brain oscillations during sleep.

Research Agenda

To better understand the effects of transcranial electrical stimulation (tES) and its therapeutic potential, future studies should:

- Focus on well-defined, sleep-specific outcome measures.
- Ensure adequate sample sizes to allow for statistically robust conclusions.
- Identify biological and sociodemographic predictors of individual response.
- Systematically compare stimulation protocols in terms of their effects on electrophysiological sleep parameters.
- Develop and implement methodological strategies to reduce sources of bias.

protocols. The inclusion of advanced neurophysiological methods, such as intracranial EEG, may provide further insights into the mechanisms underlying the effects of tES. Given the central role of sleep in cognitive health and the increasing burden of neurodegenerative diseases, further investigation in larger and more diverse populations is warranted to evaluate the therapeutic potential of tES in sleep medicine.

Conflict of interest

FF holds equity in and serves as a consultant for Electromedical Products International (EPI). FF is the lead inventor of non-invasive brain stimulation technology and receives royalty payments from the University of North Carolina at Chapel Hill. FF is the founder and sole owner of Flavio Labs LLC, a coaching and consulting firm, and co-founder and equity holder of Qortical Clinical Partners Inc. FF holds an adjunct professorship at Inselspital, Bern University Hospital (University of Bern, Switzerland) and serves asa consultant to that institution. FF receives royalty payments from Academic Press for the textbook *Network Neuroscience*.

Acknowledgments

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2026.102321.

References

[1] Polanía R, Nitsche MA, Ruff CC. Studying and modifying brain function with non-invasive brain stimulation. Nat Neurosci 2018;21(2):174–87.
[2] Fregni F, El-Hagrassy MM, Pacheco-Barrios K, Carvalho S, Leite J, Simis M, et al. Evidence-based guidelines and secondary meta-analysis for the use of transcranial direct current stimulation in neurological and psychiatric disorders. Int J Neuropsychopharmacol 2021;24(4):256–313.
[3] Herrmann CS, Rach S, Neuling T, Strüber D. Transcranial alternating current stimulation: a review of the underlying mechanisms and modulation of cognitive processes. Front Hum Neurosci 2013;7:279.
[4] Liu A, Vöröslakos M, Kronberg G, Henin S, Krause MR, Huang Y, et al. Immediate neurophysiological effects of transcranial electrical stimulation. Nat Commun 2018;9(1):5092.
[5] Mattioli F, Maglianella V, D'Antonio S, Trimarco E, Caligiore D. Non-invasive brain stimulation for patients and healthy subjects: current challenges and future perspectives. J Neurol Sci 2024;456:122825.
[6] Loo CK, Alonzo A, Martin D, Mitchell PB, Galvez V, Sachdev P. Transcranial direct current stimulation for depression: 3-week, randomised, sham-controlled trial. Br J Psychiatry 2012;200(1):52–9.
[7] Fagerlund AJ, Hansen OA, Aslaksen PM. Transcranial direct current stimulation as a treatment for patients with fibromyalgia: a randomized controlled trial. Pain 2015;156(1):62–71.
[8] Benninger DH, Lomarev M, Lopez G, Wassermann EM, Li X, Considine E, et al. Transcranial direct current stimulation for the treatment of Parkinson's disease. J Neurol Neurosurg Psychiatry 2010;81(10):1105–11.
[9] Benster LL, Weissman CR, Stolz LA, Daskalakis ZJ, Appelbaum LG. Pre-clinical indications of brain stimulation treatments for non- affective psychiatric disorders, a status update. Transl Psychiatry 2023;13(1):390.
[10] Lefaucheur JP, Antal A, Ayache SS, Benninger DH, Brunelin J, Cogiamanian F, et al. Evidence-based guidelines on the therapeutic use of transcranial direct current stimulation (tDCS). Clin Neurophysiol 2017;128(1):56–92.
[11] Bhattacharya A, Mrudula K, Sreepada SS, Sathyaprabha TN, Pal PK, Chen R, et al. An overview of noninvasive brain stimulation: basic principles and clinical applications. Can J Neurol Sci 2022;49(4):479–92.
[12] Nitsche MA, Paulus W. Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. J Physiol 2000;527(Pt 3):633–9.
[13] Klink K, Paßmann S, Kasten FH, Peter J. The modulation of cognitive performance with transcranial alternating current stimulation: a systematic review of frequency-specific effects. Brain Sci 2020;10(12).
[14] Herrero Babiloni A, Bellemare A, Beetz G, Vinet SA, Martel MO, Lavigne GJ, et al. The effects of non-invasive brain stimulation on sleep disturbances among different neurological and neuropsychiatric conditions: a systematic review. Sleep Med Rev 2021;55:101381.
[15] Nejati V, Vaziri Z, Antal A, Antonenko D, Behroozmand R, Bestmann S, et al. Report approval for transcranial electrical stimulation (RATES): expert recommendation based on a Delphi consensus study. Nat Protoc 2025.
[16] Chokroverty S. 22An overview of normal sleep. In: Chokroverty S, Allen RP, Walters AS, Montagna P, editors. Sleep and movement disorders. Oxford University Press; 2013.
[17] Walker MP. A refined model of sleep and the time course of memory formation. Behav Brain Sci 2005;28(1):51–64. discussion –104.
[18] Gehrman E. A child's need for sleep. Sleep Issue [Internet] 2022 (Sleep Issue).
[19] Rasch B, Born J. About sleep's role in memory. Physiol Rev 2013;93(2):681–766.
[20] Paruthi S, Brooks LJ, D'Ambrosio C, Hall WA, Kotagal S, Lloyd RM, et al. Consensus statement of the American academy of sleep medicine on the recommended amount of sleep for healthy children: methodology and discussion. J Clin Sleep Med 2016;12(11):1549–61.
[21] Dzierzewski JM, Perez E, Ravyts SG, Dautovich N. Sleep and cognition: a narrative review focused on older adults. Sleep Med Clin 2022;17(2):205–22.
[22] Reddy OC, van der Werf YD. The sleeping brain: harnessing the power of the glymphatic system through lifestyle choices. Brain Sci 2020;10(11).
[23] Jessen NA, Munk AS, Lundgaard I, Nedergaard M. The glymphatic system: a beginner's guide. Neurochem Res 2015;40(12):2583–99.
[24] Xie L, Kang H, Xu Q, Chen MJ, Liao Y, Thiyagarajan M, et al. Sleep drives metabolite clearance from the adult brain. Science 2013;342(6156):373–7.
[25] Miao A, Luo T, Hsieh B, Edge CJ, Gridley M, Wong RTC, et al. Brain clearance is reduced during sleep and anesthesia. Nat Neurosci 2024;27(6):1046–50.
[26] Ferris CF. Rethinking the conditions and mechanism for glymphatic clearance. Front Neurosci 2021;15:624690.
[27] Jaspan VN, Greenberg GS, Parihar S, Park CM, Somers VK, Shapiro MD, et al. The role of sleep in cardiovascular disease. Curr Atheroscler Rep 2024;26(7):249–62.
[28] Trenell MI, Marshall NS, Rogers NL. Sleep and metabolic control: waking to a problem? Clin Exp Pharmacol Physiol 2007;34(1–2):1–9.
[29] Walker MP. Cognitive consequences of sleep and sleep loss. Sleep Med 2008;9 (Suppl 1):S29–34.
[30] Wang S, Zheng X, Huang J, Liu J, Li C, Shang H. Sleep characteristics and risk of alzheimer's disease: a systematic review and meta-analysis of longitudinal studies. J Neurol 2024;271(7):3782–93.
[31] Hanke JM, Schindler KA, Seiler A. On the relationships between epilepsy, sleep, and Alzheimer's disease: a narrative review. Epilepsy Behav 2022;129:108609.
[32] Europe A. Dementia in Europe yearbook 2019: estimating the prevalence of dementia in Europe. Luxembourg: Alzheimer Europe; 2019.
[33] Jones BE. Arousal systems. Front Biosci 2003;8:s438–51.
[34] Magoun HW. The ascending reticular activating system. Res Publ Assoc Res Nerv Ment Dis 1952;30:480–92.
[35] Krone L, Frase L, Piosczyk H, Selhausen P, Zittel S, Jahn F, et al. Top-down control of arousal and sleep: fundamentals and clinical implications. Sleep Med Rev 2017;31:17–24.
[36] Steriade M. Grouping of brain rhythms in corticothalamic systems. Neuroscience 2006;137(4):1087–106.
[37] Steriade M. The corticothalamic system in sleep. Front Biosci 2003;8:d878–99.
[38] Destexhe A, Contreras D, Steriade M. Cortically-induced coherence of a thalamic-generated oscillation. Neuroscience 1999;92(2):427–43.
[39] Bueno-Lopez A, Eggert T, Dorn H, Danker-Hopfe H. Slow oscillatory transcranial direct current stimulation (so-tDCS) during slow wave sleep has no effects on declarative memory in healthy young subjects. Brain Stimul 2019;12(4):948–58.
[40] Sahlem GL, Badran BW, Halford JJ, Williams NR, Korte JE, Leslie K, et al. Oscillating square wave transcranial direct current stimulation (tDCS) delivered during slow wave sleep does not improve declarative memory more than Sham: a randomized Sham controlled crossover study. Brain Stimul 2015;8(3):528–34.
[41] Wang Y, Cao Q, Wei C, Xu F, Zhang P, Zeng H, et al. The effect of transcranial electrical stimulation on the recovery of sleep quality after sleep deprivation based on an EEG analysis. Brain Sci 2023;13(6).
[42] Marshall L, Mölle M, Hallschmid M, Born J. Transcranial direct current stimulation during sleep improves declarative memory. J Neurosci 2004;24(44): 9985–92.
[43] Fehér KD, Wunderlin M, Maier JG, Hertenstein E, Schneider CL, Mikutta C, et al. Shaping the slow waves of sleep: a systematic and integrative review of sleep slow wave modulation in humans using non-invasive brain stimulation. Sleep Med Rev 2021;58:101438.
[44] Krone LB, Fehér KD, Rivero T, Omlin X. Brain stimulation techniques as novel treatment options for insomnia: a systematic review. J Sleep Res 2023;32(6): e13927.
[45] Dondé C, Brunelin J, Micoulaud-Franchi JA, Maruani J, Lejoyeux M, Polosan M, et al. The effects of transcranial electrical stimulation of the brain on sleep: a systematic review. Front Psychiatr 2021;12:646569.
[46] Annarumma L, D'Atri A, Alfonsi V, De Gennaro L. The efficacy of transcranial current stimulation techniques to modulate resting-state EEG, to affect vigilance and to promote sleepiness. Brain Sci 2018;8(7).
[47] Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. Br Med J 2021;372:n71.
[48] Berry RB, Brooks R, Gamaldo C, Harding SM, Lloyd RM, Quan SF, et al. AASM scoring manual updates for 2017 (Version 2.4). J Clin Sleep Med 2017;13:665–6. United States.
[49] Mölle M, Born J. Slow oscillations orchestrating fast oscillations and memory consolidation. Prog Brain Res 2011;193:93–110.

[50] Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system of sleep stages in human subjects. Los Angeles: Brain Information Service/Brain Research Institute, University of California; 1968.
[51] Sterne JAC, Savovic J, Page MJ, Elbers RG, Blencowe NS, Boutron I, et al. RoB 2: a revised tool for assessing risk of bias in randomised trials. Br Med J 2019;366:l4898.
[52] Lafon B, Henin S, Huang Y, Friedman D, Melloni L, Thesen T, et al. Low frequency transcranial electrical stimulation does not entrain sleep rhythms measured by human intracranial recordings. Nat Commun 2017;8(1):1199.
[53] Zhou Q, Liu Z, Zhao S, Yu J, Zhou D, Xu W, et al. Transcranial magnetic stimulation combined with transcranial direct current stimulation in patients with chronic insomnia: a case report. J Clin Sleep Med 2022;18:2871–4. United States: © 2022 American Academy of Sleep Medicine.
[54] Tang NKY, Stella MT, Banks PDW, Sandhu HK, Berna C. The effect of opioid therapy on sleep quality in patients with chronic non-malignant pain: a systematic review and exploratory meta-analysis. Sleep Med Rev 2019;45:105–26.
[55] Herrero Babiloni A, Guay S, Nixdorf DR, de Beaumont L, Lavigne G. Non-invasive brain stimulation in chronic orofacial pain: a systematic review. J Pain Res 2018;11:1445–57.
[56] Paßmann S, Külzow N, Ladenbauer J, Antonenko D, Grittner U, Tamm S, et al. Boosting slow oscillatory activity using tDCS during early nocturnal slow wave sleep does not improve memory consolidation in healthy older adults. Brain Stimul 2016;9(5):730–9.
[57] Munz MT, Prehn-Kristensen A, Thielking F, Mölle M, Göder R, Baving L. Slow oscillating transcranial direct current stimulation during non-rapid eye movement sleep improves behavioral inhibition in attention-deficit/hyperactivity disorder. Front Cell Neurosci 2015;9:307.
[58] Antonenko D, Diekelmann S, Olsen C, Born J, Mölle M. Napping to renew learning capacity: enhanced encoding after stimulation of sleep slow oscillations. Eur J Neurosci 2013;37(7):1142–51.
[59] Cellini N, Shimizu RE, Connolly PM, Armstrong DM, Hernandez LT, Polakiewicz AG, et al. Short duration repetitive transcranial electrical stimulation during sleep enhances declarative memory of facts. Front Hum Neurosci 2019;13:123.
[60] Voss U, Holzmann R, Hobson A, Paulus W, Koppehele-Gossel J, Klimke A, et al. Induction of self awareness in dreams through frontal low current stimulation of gamma activity. Nat Neurosci 2014;17(6):810–2.
[61] Johnson JM, Durrant SJ. The effect of cathodal transcranial direct current stimulation during rapid eye-movement sleep on neutral and emotional memory. R Soc Open Sci 2018;5(7):172353.
[62] Reato D, Gasca F, Datta A, Bikson M, Marshall L, Parra LC. Transcranial electrical stimulation accelerates human sleep homeostasis. PLoS Comput Biol 2013;9(2):e1002898.
[63] Li J, Li A, Jiao J, Hu K, Jiang Y, Hu X, et al. Effects of high-definition transcranial direct current stimulation for the treatment of chronic insomnia: a randomized, double-blind, controlled trial. Sci Rep 2025;15(1):26574.
[64] Motamedi GK, Jeliazkov PG, Oyegbile-Chidi TO, Song SS, Sharma VD, Mete M, et al. Transcranial alternating current stimulation (tACS) as a treatment for insomnia. Can J Neurol Sci 2023;50(3):446–9.
[65] Wang L, Yu Y, Ying S, Wang M, Qin Y, Liu T, et al. A personalized closed-loop brain stimulation protocol for difficulty falling asleep. IEEE Trans Neural Syst Rehabil Eng 2025;33:2368–80.
[66] Saebipour MR, Joghataei MT, Yoonessi A, Sadeghniiat-Haghighi K, Khalighinejad N, Khademi S. Slow oscillating transcranial direct current stimulation during sleep has a sleep-stabilizing effect in chronic insomnia: a pilot study. J Sleep Res 2015;24(5):518–25.
[67] Frase L, Selhausen P, Krone L, Tsodor S, Jahn F, Feige B, et al. Differential effects of bifrontal tDCS on arousal and sleep duration in insomnia patients and healthy controls. Brain Stimul 2019;12(3):674–83.
[68] Prehn-Kristensen A, Munz M, Göder R, Wilhelm I, Korr K, Vahl W, et al. Transcranial oscillatory direct current stimulation during sleep improves declarative memory consolidation in children with attention-deficit/hyperactivity disorder to a level comparable to healthy controls. Brain Stimul 2014;7(6):793–9.
[69] Roizenblatt S, Fregni F, Gimenez R, Wetzel T, Rigonatti SP, Tufik S, et al. Site-specific effects of transcranial direct current stimulation on sleep and pain in fibromyalgia: a randomized, sham-controlled study. Pain Pract 2007;7(4):297–306.
[70] Lustenberger C, Boyle MR, Alagapan S, Mellin JM, Vaughn BV, Fröhlich F. Feedback-controlled transcranial alternating current stimulation reveals a functional role of sleep spindles in motor memory consolidation. Curr Biol 2016;26(16):2127–36.
[71] Zhou Q, Yu C, Yu H, Zhang Y, Liu Z, Hu Z, et al. The effects of repeated transcranial direct current stimulation on sleep quality and depression symptoms in patients with major depression and insomnia. Sleep Med 2020;70:17–26.
[72] Thom JL, Staresina BP. Human sleep spindles track experimentally excited brain circuits. Sleep 2025;48(7).
[73] Shi Y, Tian X, Li T, Hu Y, Xie Y, Li H, et al. The influence of transcranial alternating current stimulation on EEG spectral power during subsequent sleep: a randomized crossover study. Sleep Med 2025;126:185–93.
[74] Tabarak S, Zhu X, Li P, Weber FD, Shi L, Gong Y, et al. Temporal dynamics of negative emotional memory reprocessing during sleep. Transl Psychiatry 2024;14(1):434.
[75] Ladenbauer J, Khakimova L, Malinowski R, Obst D, Tönnies E, Antonenko D, et al. Towards optimization of oscillatory stimulation during sleep. Neuromodulation 2023;26(8):1592–601.
[76] Li Z, Zhao X, Feng L, Zhao Y, Pan W, Liu Y, et al. Can daytime transcranial direct current stimulation treatment change the sleep electroencephalogram complexity of REM sleep in depressed patients? A double-blinded, randomized, placebo-controlled trial. Front Psychiatr 2022;13:851908.
[77] Robert BM, Jones AP, Mullins TS, Trumbo MCS, Ketz NA, Howard MD, et al. Closed-loop transcranial alternating current stimulation of slow wave oscillations during sleep reduces declarative learning the next day. Brain Stimul 2022;15(6):1565–6.
[78] Yu J, Wu Y, Wu B, Xu C, Cai J, Wen X, et al. Sleep patterns correlates with the efficacy of tDCS on post-stroke patients with prolonged disorders of consciousness. J Transl Med 2022;20(1):601.
[79] Charest J, Marois A, Bastien CH. Can a tDCS treatment enhance subjective and objective sleep among student-athletes? J Am Coll Health 2021;69(4):378–89.
[80] Dehnavi F, Koo-Poeggel PC, Ghorbani M, Marshall L. Spontaneous slow oscillation-slow spindle features predict induced overnight memory retention. Sleep 2021;44(10).
[81] Hathaway E, Morgan K, Carson M, Shusterman R, Fernandez-Corazza M, Luu P, et al. Transcranial electrical Stimulation targeting limbic cortex increases the duration of human deep sleep. Sleep Med 2021;81:350–7.
[82] Hubbard RJ, Zadeh I, Jones AP, Robert B, Bryant NB, Clark VP, et al. Brain connectivity alterations during sleep by closed-loop transcranial neurostimulation predict metamemory sensitivity. Network Neurosci 2021;5(3):734–56.
[83] Xie J, Wang L, Xiao C, Ying S, Ren J, Chen Z, et al. Low frequency transcranial alternating current stimulation accelerates sleep onset process. IEEE Trans Neural Syst Rehabil Eng 2021;29:2540–9.
[84] Noreika V, Windt JM, Kern M, Valli K, Salonen T, Parkkola R, et al. Modulating dream experience: noninvasive brain stimulation over the sensorimotor cortex reduces dream movement. Sci Rep 2020;10(1):6735.
[85] Pilly PK, Skorheim SW, Hubbard RJ, Ketz NA, Roach SM, Lerner I, et al. One-shot tagging during wake and cueing during sleep with spatiotemporal patterns of transcranial electrical stimulation can boost long-term metamemory of individual episodes in humans. Front Neurosci 2019;13:1416.
[86] D'Atri A, De Simoni E, Gorgoni M, Ferrara M, Ferlazzo F, Rossini PM, et al. Electrical stimulation of the frontal cortex enhances slow-frequency EEG activity and sleepiness. Neuroscience 2016;324:119–30.
[87] Ketz N, Jones AP, Bryant NB, Clark VP, Pilly PK. Closed-loop slow-wave tACS improves sleep-dependent long-term memory generalization by modulating endogenous oscillations. J Neurosci 2018;38(33):7314–26.
[88] Koo PC, Mölle M, Marshall L. Efficacy of slow oscillatory-transcranial direct current stimulation on EEG and memory - contribution of an inter-individual factor. Eur J Neurosci 2018;47(7):812–23.
[89] Ladenbauer J, Külzow N, de Boor R, Avramova E, Grittner U, Flöel A. Promoting sleep oscillations and their functional coupling by transcranial stimulation enhances memory consolidation in mild cognitive impairment. J Neurosci 2017;37(30):7111–24.
[90] Frase L, Piosczyk H, Zittel S, Jahn F, Selhausen P, Krone L, et al. Modulation of total sleep time by transcranial direct current stimulation (tDCS). Neuropsychopharmacology 2016;41(10):2577–86.
[91] Ladenbauer J, Külzow N, Passmann S, Antonenko D, Grittner U, Tamm S, et al. Brain stimulation during an afternoon nap boosts slow oscillatory activity and memory consolidation in older adults. Neuroimage 2016;142:311–23.
[92] Del Felice A, Magalini A, Masiero S. Slow-oscillatory transcranial direct current stimulation modulates memory in temporal lobe Epilepsy by altering sleep spindle generators: a possible rehabilitation tool. Brain Stimul 2015;8(3):567–73.
[93] Garside P, Arizpe J, Lau CI, Goh C, Walsh V. Cross-hemispheric alternating current stimulation during a nap disrupts slow wave activity and associated memory consolidation. Brain Stimul 2015;8(3):520–7.
[94] Westerberg CE, Florczak SM, Weintraub S, Mesulam MM, Marshall L, Zee PC, et al. Memory improvement via slow-oscillatory stimulation during sleep in older adults. Neurobiol Aging 2015;36(9):2577–86.
[95] Eggert T, Dorn H, Sauter C, Nitsche MA, Bajbouj M, Danker-Hopfe H. No effects of slow oscillatory transcranial direct current stimulation (tDCS) on sleep-dependent memory consolidation in healthy elderly subjects. Brain Stimul 2013;6(6):938–45.
[96] Göder R, Baier PC, Beith B, Baecker C, Seeck-Hirschner M, Junghanns K, et al. Effects of transcranial direct current stimulation during sleep on memory performance in patients with schizophrenia. Schizophr Res 2013;144(1–3):153–4.
[97] Jakobson AJ, Fitzgerald PB, Conduit R. Induction of visual dream reports after transcranial direct current stimulation (tDCs) during stage 2 sleep. J Sleep Res 2012;21(4):369–79.
[98] Marshall L, Kirov R, Brade J, Mölle M, Born J. Transcranial electrical currents to probe EEG brain rhythms and memory consolidation during sleep in humans. PLoS One 2011;6(2):e16905.
[99] Nitsche MA, Jakoubkova M, Thirugnanasambandam N, Schmalfuss L, Hullemann S, Sonka K, et al. Contribution of the premotor cortex to consolidation of motor sequence learning in humans during sleep. J Neurophysiol 2010;104(5):2603–14.
[100] Marshall L, Helgadóttir H, Mölle M, Born J. Boosting slow oscillations during sleep potentiates memory. Nature 2006;444(7119):610–3.
[101] Johnson DR, Wefel JS. Relationship between cognitive function and prognosis in glioblastoma. CNS Oncol 2013;2(2):195–201.

[102] Born J, Wilhelm I. System consolidation of memory during sleep. Psychol Res 2012;76(2):192–203.
[103] Jones AP, Choe J, Bryant NB, Robinson CSH, Ketz NA, Skorheim SW, et al. Dose-dependent effects of closed-loop tACS delivered during slow-wave oscillations on memory consolidation. Front Neurosci 2018;12:867.
[104] Batsikadze G, Moliadze V, Paulus W, Kuo MF, Nitsche MA. Partially non-linear stimulation intensity-dependent effects of direct current stimulation on motor cortex excitability in humans. J Physiol 2013;591(7):1987–2000.
[105] Priori A. Brain polarization in humans: a reappraisal of an old tool for prolonged non-invasive modulation of brain excitability. Clin Neurophysiol 2003;114(4):589–95.
[106] Nitsche MA, Liebetanz D, Antal A, Lang N, Tergau F, Paulus W. Modulation of cortical excitability by weak direct current stimulation– technical, safety and functional aspects. Suppl Clin neurophysiol 2003;56:255–76.
[107] Bikson M, Grossman P, Thomas C, Zannou AL, Jiang J, Adnan T, et al. Safety of transcranial direct current stimulation: evidence based update 2016. Brain Stimul 2016;9(5):641–61.
[108] Nitsche MA, Muller-Dahlhaus F, Paulus W, Ziemann U. The pharmacology of neuroplasticity induced by non-invasive brain stimulation: building models for the clinical use of CNS active drugs. J Physiol 2012;590(19):4641–62.
[109] Fonteneau C, Redoute J, Haesebaert F, Le Bars D, Costes N, Suaud-Chagny MF, et al. Frontal transcranial direct current stimulation induces dopamine release in the ventral striatum in human. Cerebr Cortex 2018;28(7):2636–46.
[110] Acler M, Bocci T, Valenti D, Turri M, Priori A, Bertolasi L. Transcranial direct current stimulation (tDCS) for sleep disturbances and fatigue in patients with post-polio syndrome. Restor Neurol Neurosci 2013;31(5):661–8.
[111] Grossman N, Bono D, Dedic N, Kodandaramaiah SB, Rudenko A, Suk HJ, et al. Noninvasive deep brain stimulation via temporally interfering electric fields. Cell 2017;169(6):1029. 41.e16.
[112] Singh G, Sharma P, Forrest G, Harkema S, Behrman A, Gerasimenko Y. Spinal cord transcutaneous stimulation in cervical spinal cord injury: a review examining upper extremity neuromotor control, recovery mechanisms, and future directions. J Neurotrauma 2024.
[113] Grimaldi G, Argyropoulos GP, Bastian A, Cortes M, Davis NJ, Edwards DJ, et al. Cerebellar transcranial direct current stimulation (ctDCS): a novel approach to understanding cerebellar function in health and disease. Neuroscientist 2016;22(1):83–97.
[114] Nierat MC, Similowski T, Lamy JC. Does trans-spinal direct current stimulation alter phrenic motoneurons and respiratory neuromechanical outputs in humans? A double-blind, sham- controlled, randomized, crossover study. J Neurosci 2014;34(43):14420–9.
[115] Cellini N, Mednick SC. Stimulating the sleeping brain: current approaches to modulating memory-related sleep physiology. J Neurosci Methods 2019;316:125–36.
[116] Antal A, Varga ET, Kincses TZ, Nitsche MA, Paulus W. Oscillatory brain activity and transcranial direct current stimulation in humans. Neuroreport 2004;15(8):1307–10.
[117] Maeoka H, Matsuo A, Hiyamizu M, Morioka S, Ando H. Influence of transcranial direct current stimulation of the dorsolateral prefrontal cortex on pain related emotions: a study using electroencephalographic power spectrum analysis. Neurosci Lett 2012;512(1):12–6.
[118] Wirth M, Rahman RA, Kuenecke J, Koenig T, Horn H, Sommer W, et al. Effects of transcranial direct current stimulation (tDCS) on behaviour and electrophysiology of language production. Neuropsychologia 2011;49(14):3989–98.
[119] Edwards BA, O'Driscoll DM, Ali A, Jordan AS, Trinder J, Malhotra A. Aging and sleep: physiology and pathophysiology. Semin Respir Crit Care Med 2010;31(5):618–33.
[120] Murphy M, Huber R, Esser S, Riedner BA, Massimini M, Ferrarelli F, et al. The cortical topography of local sleep. Curr Top Med Chem 2011;11(19):2438–46.