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Original Article

The efficacy of biofeedback for the treatment of insomnia: a critical review

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ABSTRACT

Background: The popularity of biofeedback as a non-pharmacological treatment option for insomnia has increased in recent times despite inconsistent empirical evidence for its therapeutic efficacy.
Objective: The purpose of the current review was to systematically assess the efficacy of using biofeedback to treat insomnia.
Methods and results: A search of electronic databases (PubMED, MEDLINE, OvidSP, Ovid EMBASE, PsychInfo, The Cochrane Library including Cochrane Reviews), clinical trials databases and registries (Clinical Trials Database [US], Australian New Zealand Clinical Trials Registry [ANZCTR]) and online journal (eg, SLEEP, Sleep Medicine) identified 92 studies. Of these, 50 publications were descriptive or review papers about use of biofeedback for the treatment of insomnia, while an additional 37 did not meet the detailed inclusion criteria (ie not original research, participants do not meet the diagnostic criteria for insomnia). Six full-text articles met inclusion criteria and were included in this review. Methodological flaws including poor study design (small sample size, lack of control group) limit the validity of the body of work in this field to date and fail adequately to account for other unspecified factors likely to drive the observed changes, such as care and attention of those administering the treatment, as well as the expectations and motivations of the patient.
Conclusion: There is an urgent need for future studies to clarify the role of unspecific placebo effects when reporting biofeedback effects for the treatment of insomnia.

1. Introduction

Insomnia is the most prevalent sleep disorder, affecting 10–30% of the population [1]. Insomnia is characterized by a subjective complaint of difficulty falling asleep, maintaining sleep or non-restorative sleep coupled with daytime impairment [2]. The impact of the disorder on the quality of the lives of sufferers is widespread across both psychological and physiological functioning. Impairments to daytime functioning can be diverse and include fatigue, problems with cognitive performance, complaints of depressed mood and feelings of anxiety, and difficulty maintaining social relationships [2,3].

The most common medical treatment for insomnia is pharmacotherapy. Of those patients presenting with insomnia, 90% are prescribed medication, predominantly benzodiazepines [4]. Hypnotic medications are simple and quick to prescribe but are not best practice for durable treatment of insomnia [5], and are associated with high rates of adverse side effects, as well as the development of dependence and withdrawal effects following long-term use. A further plethora of adverse effects include rebound insomnia, adverse physiological effects and heightened risks of hepatic, renal, respiratory and cardiac disorders, daytime sedation, cognitive impairments and increased risk of falls in the elderly [6]. Conversely, non-pharmacological interventions for insomnia produce robust

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and durable therapeutic improvements, extending well beyond the period of therapy [7–10]. In addition, cognitive behavioral therapy for insomnia (CBTi) is typically superior to pharmacotherapy at reducing insomnia severity without the known side effects and issues of tolerance and dependence with medications [11]. Biofeedback is one of the many non-pharmacological treatment options for insomnia that has recently increased popularity.

Biofeedback aims to reduce conditioned arousal, particularly in the form of somatic, cognitive and/or cortical hyper-arousal, which is a key characteristic of insomnia [12–14]. Cognitive and somatic hyper-arousal are reflected physiologically at sleep onset and during lighter NREM sleep stages with beta and gamma electroencephalographic (EEG) activity [15–17], greater metabolic rate [18], elevated heart rate and impaired heart rate variability, and elevated muscle tension [19–22]. In regards to insomnia, biofeedback assumes the elevated frequencies of brain activity and associated cognitive hyper-arousal can be diminished through the use of instrumental EEG conditioning of slower frequencies [23]. Similarly, it is assumed that instrumental conditioning can be used to reduce somatic arousal across a range of somatic domains.

The purpose of this review was to assess the efficacy of biofeedback for the treatment of insomnia. To date, two types of biofeedback have been considered for the treatment of insomnia and their efficacy assessed in insomnia populations. These types of biofeedback include (1) neurofeedback that comprises theta EEG and/or sensorimotor rhythm (SMR) EEG, and (2) electromyography (EMG) feedback. Theta feedback is targeted at increasing the power of the theta rhythm (4–7 Hz). A predominance of theta EEG defines stage 1 sleep, and has been suggested characteristic of a very deep stage of relaxation [24]. It has been proposed that if increasing the power of theta can be learned, a quicker sleep onset will be facilitated [24]. SMR is designed to enhance EEG amplitude within the 12–15 Hz range, which are prominent over the sensorimotor cortex, and inhibit higher beta (20–30 Hz) power. Oscillations within the 12–15 Hz range are abundant during light NREM sleep and overlap with the sleep spindle frequency band. Early research has demonstrated neurofeedback training of the SMR (12–15 Hz) rhythm in the wake EEG in cats increased sleep spindle bursts and quiet sleep in addition to a suppression of motor activity [25]. The present review is the first to assess the efficacy of neurofeedback training of the SMR rhythm for improving the sleep of humans suffering insomnia. EMG feedback is aimed at decreasing muscle tension (or EMG power), and thereby reducing heightened physiological arousal, which is characteristic of some types of insomnia, to facilitate the onset of sleep.

2. Method

2.1. Literature search and inclusion criteria

Electronic databases (PubMED, MEDLINE, OvidSP, Ovid EMBASE, PsychInfo, The Cochrane Library), clinical trials databases and registries (Clinical Trials Databases: US clinicaltrials.gov, Australian New Zealand Clinical Trials Registry) and online journals (eg, SLEEP, Sleep Medicine) were used to identify and retrieve research articles assessing the clinical efficacy of biofeedback for the treatment of insomnia. Relevant articles were also manually identified from the reference lists of retrieved articles. Biofeedback included neurofeedback, comprising theta feedback and/or sensorimotor rhythm feedback, with or without electromyographic (EMG) feedback. The search terms used included “Insomnia AND Neurofeedback” and “Insomnia AND Biofeedback”. Relevant articles were also manually identified from the reference lists of retrieved articles. Peer-reviewed articles describing original research and containing at least one measure of sleep quality, duration, or insomnia severity were included. All literature was required to assess the clinical efficacy of biofeedback for the treatment of insomnia, therefore participants of included studies were required to meet the diagnostic criteria for insomnia according to the Diagnostic and Statistical Manual of Mental Disorders [3] or for earlier studies the diagnostic criteria relevant at the time of publication. All articles were published in English and no case studies were included. Study design was not used as a basis on which to select studies for the current review. The literature search was conducted on April 27th 2018 and included articles published between January 1970 and March 2018. The search was developed and conducted by the primary author (NL). The review was conducted according to the PRISMA guidelines (ie, the Preferred Reporting Items for Systematic Reviews and Meta-Analyses; See Table 1) [26].

3. Results and discussion

3.1. Selection of studies

Fig. 1 shows the flow of the literature search process. The search identified 93 publications. Of these, 50 publications provided only general background information about the use of biofeedback for the treatment of insomnia, while an additional 37 did not meet the inclusion criteria. The main reasons for exclusion were the absence of original research (n = 15) and participants who failed to meet diagnostic criteria for insomnia (n = 15). All reasons for exclusion are detailed in Fig. 1. A total of only six studies met the inclusion criteria and were subsequently included in this review. Retrieved studies were reviewed for eligibility independently by two of the authors (NL and LL) in an un-blinded and standardised manner in accordance with the selection criteria. Instances of disagreement between the reviewers were resolved by consensus.

The quality of the included studies was assessed across six domains of potential bias, including study participation (ie recruited sample is representative of the population of interest), study attrition, adequate measurement of the prognostic factor, satisfactory measurement of the outcome variable as well as confounding variables, and adequate statistical analyses [27]. A list of items, adapted from Hayden and colleagues [27], was used by the primary author (NL) to assess each potential opportunity for bias. The six domains of potential bias and the items used to assess each potential opportunity for bias are described in Table 2 below. Domains were rated as high risk for potential bias if they study accounted for no or very few of the items listed, neural risk if the items were partly accounted for, or low risk if most or all of the items were accounted for. The quality ratings of the included studies are presented in Table 3. The majority of studies (n = 5, 83%) accounted adequately for study participation and measurement of the prognostic factor, with many (n = 4, 67%) also having measured the outcome variable satisfactorily. Fewer studies accounted adequately for study attrition (n = 2), measurement of confounding variables (n = 3, 50%), and statistical analyses (n = 2, 33%). This information was not used in any subsequent data synthesis but rather to inform the overall quality of the published literature in this field at the present time.

3.2. Data extraction

For each of the included studies, the characteristics of the participants (including age, gender, diagnosis), type of biofeedback assessed and comparator, study design (including sample size), follow up period and limitations, and outcome measures for each diagnostic domain of insomnia, including subjective and objective sleep quality, perceived severity of insomnia, quality of life and daytime functioning were extracted. The primary outcome variables included sleep onset latency (SOL), wake after sleep onset (WASO), number of awakenings (NWAK), total sleep time (TST),

Table 1
PRISMA checklist adapted from Moher et al., [26].

Section/topic	#	Checklist item	Reported on page #
TITLE			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	3–5
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4–6
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (eg, Web address), and, if available, provide registration information including registration number.	N/A
Eligibility criteria	6	Specify study characteristics (eg, PICOS, length of follow-up) and report characteristics (eg, years considered, language, publication status) used as criteria for eligibility, giving rationale.	5–6
Information sources	7	Describe all information sources (eg, databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	5–6
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	5–6
Study selection	9	State the process for selecting studies (ie, screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	6–7
Data collection process	10	Describe method of data extraction from reports (eg, piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	6–8
Data items	11	List and define all variables for which data were sought (eg, PICOS, funding sources) and any assumptions and simplifications made.	7–8
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	7
Summary measures	13	State the principal summary measures (eg, risk ratio, difference in means).	9
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (eg, I^2) for each meta-analysis.	N/A
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (eg, publication bias, selective reporting within studies).	11
Additional analyses	16	Describe methods of additional analyses (eg, sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	N/A
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	6, Fig. 1
Study characteristics	18	For each study, present characteristics for which data were extracted (eg, study size, PICOS, follow-up period) and provide the citations.	9, Table 3
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	7, Table 3
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	N/A
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	N/A
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	N/A
Additional analysis	23	Give results of additional analyses, if done (eg, sensitivity or subgroup analyses, meta-regression [see Item 16]).	N/A
DISCUSSION			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (eg, healthcare providers, users, and policy makers).	11–19, 21–22
Limitations	25	Discuss limitations at study and outcome level (eg, risk of bias), and at review-level (eg, incomplete retrieval of identified research, reporting bias).	19–21
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	21–22
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (eg, supply of data); role of funders for the systematic review.	N/A

time in bed (TIB), sleep efficiency (SE), amount of time spent in each sleep stage, perceived insomnia severity (as assessed by the Insomnia Severity Index (ISI) [32]), self-reported quality of life, and daytime functioning. The primary author (NL) extracted the relevant data relevant, which was confirmed by senior author (LL). Any disagreements between the authors were resolved via consensus.

It is noteworthy to discuss the distinction between subjective sleep quality and perceived insomnia severity in assessing the efficacy of biofeedback. Perceived insomnia severity is considered separately from self-reported sleep quality to provide a comprehensive summary of the efficacy of biofeedback for the treatment of insomnia rather than poor sleep quality per se. Measurement tools, such as the Pittsburgh Sleep Quality Index (PSQI), used to assess self-reported sleep quality tap into sleep disturbance related to a number of different sleep disorders and are not specific to insomnia per se.

3.3. Characteristics of included studies

The characteristics of the included studies are summarised in Table 3. The efficacy of biofeedback was assessed in a total of 143 individuals diagnosed with insomnia across the six studies. Sample

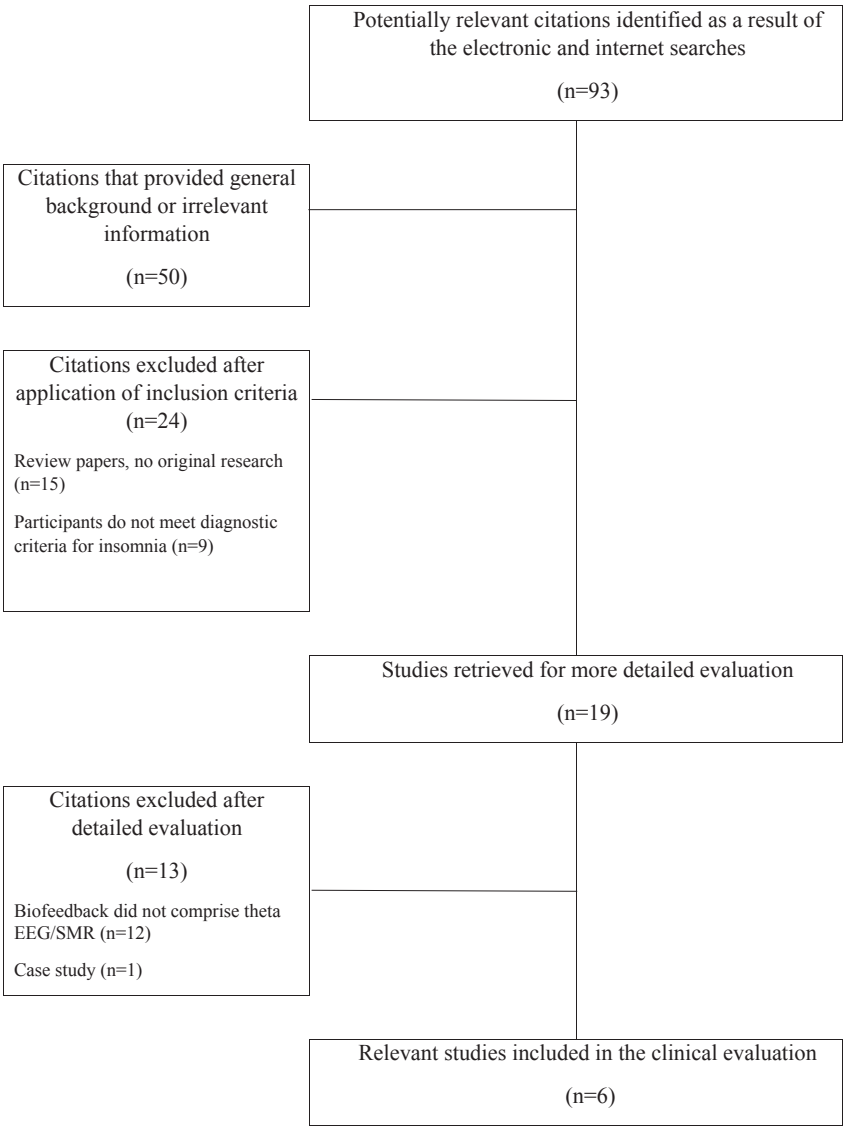

Fig. 1. Flow of literature through the search process.

sizes were typically small, ranging from eight to 48 subjects. Follow up periods varied from two weeks to nine months with a mean follow up of 5.0 months (SD = 4.01). All studies used an experimental approach with randomisation either between two or more treatment arms with some studies (n = 3, 50%) also including a no treatment or sham-conditioning group. Six protocols of biofeedback were evaluated in these studies including (1) SMR feedback (increase sensorimotor rhythm [12–15 Hz] and inhibit higher beta power [20–30 Hz] at Cz); (2) SMR in combination with EMG; (3) theta feedback (increase theta power [4–7 Hz]); and (4) an individually designed program (normalise amplitudes of delta, theta, alpha and beta) with or without (5) EMG feedback (decrease EMG power at Fpz).

3.4. Subjective sleep quality

Each of the six included studies monitored subjective sleep quality, using either sleep diaries or the PSQI, prior to treatment and again at follow up. Individually, these studies reported improved subjective estimates of sleep latency, WASO, NWAK, TST, and SE following biofeedback [28–30], the lack of a control (no treatment) group limits the validity of the findings reported in these studies and does not consider placebo effects as a plausible explanation for these improvements in sleep quality. Without the inclusion of a control (no treatment) group, subjective improvements cannot be confidently attributed to the effects of biofeedback. Biofeedback may have very high credibility as a treatment for its apparent physiological intervention, thus having an enhanced placebo effect size. It is crucial to distinguish the effects of genuine biofeedback from other non-specific factors that may promote a positive response, for example participant motivation, expectation or social support, when assessing the efficacy of biofeedback for the treatment of insomnia.

This methodological shortcoming has however been addressed by several of the included studies [23,24,30,31]. In his pioneering work, Hauri [24] randomly assigned 48 individuals suffering from insomnia to receive either sensorimotor rhythm (SMR) feedback, electromyographic (EMG) feedback, combined EMG and theta feedback, or no treatment (control). Participants received 26 training sessions for SMR and theta feedback and six sessions for EMG feedback on 2–3 occasions per week. Sleep was monitored using sleep logs kept by participants in the home environment

Table 2
Criteria list used for the assessment of the methodologic quality of the included studies, adapted from Hayden et al., [27].

Potential bias	Items considered for assessment of potential bias
Study participation	
The study sample represents the population of interest on key characteristics, sufficient to limit potential bias to the results	• The population of interest is adequately described for key characteristics. • The sampling frame and recruitment are adequately described including methods used to identify the sample, and the period and place of recruitment. • Inclusion and exclusion criteria are adequately described. • There is adequate participation in the study by eligible individuals. • The baseline study sample is adequately described for key characteristics.
Study attrition	
Loss to follow-up (from sample to study population) that is not associated with key characteristics (ie, the study data adequately represents the sample), sufficient to limit potential bias.	• Response rate (ie, proportion of study sample completing the study and providing outcome data) is adequate. • Attempts to collect information on participants who dropped out of the study are described. • Reasons for loss to follow-up are provided. • Participants lost to follow-up are adequately described for key characteristics. • There are no important differences between key characteristics and outcomes in participants who completed the study and those who did not.
Measurement of the prognostic factor	
The prognostic factor of interest is adequately measured in study participants to sufficiently limit potential bias.	• A clear definition or description of the prognostic factor measures is provided (eg, duration of exposure, clear description of the method of measurement). • Continuous variables are reported or appropriate (ie, not data-dependent) cut-points are used. • The prognostic factor measure and method are adequately valid and reliable to limit misclassification bias. • Adequate proportion of the study sample has complete data for prognostic factors. • The method and setting of measurement are the same for all study participants. • Appropriate methods are used if imputation is used for missing prognostic factor data.
Outcome measurement	
The outcome of interest is adequately measured in study participants to sufficiently limit potential bias	• A clear definition of the outcome of interest is provided, including duration of follow-up and extent of the outcome construct. • The outcome measure and method used are adequately valid and reliable to limit misclassification bias. • The method and setting of measurement are the same for all study participants.
Confounding measurement and account	
Important potential confounders are appropriately accounted for, limiting potential bias with respect to the prognostic factor of interest	• All important confounders (including key variables in conceptual model) are measured. • Clear definitions of the important confounders measured are provided (eg, including dose, level, and duration of exposures) • Measurement of all important confounders is adequately valid and reliable. • The method and setting of confounding measurement are the same for all study participants. • Appropriate methods are used if imputation is used for missing confounder data. • Important potential confounders are accounted for in the study design (eg, matching for key variables, stratification, or initial assembly of comparable groups). • Important potential confounders are accounted for in the analysis.
Statistical analyses	
The statistical analysis is appropriate for the design of the study, limiting potential for presentation of invalid results	• There is sufficient presentation of the data to assess the adequacy of the analysis • The strategy for model building (ie, inclusion of variables) is appropriate and is based on a conceptual model or framework. • The selected model is adequate for the design of the study • There is no selective reporting of results.

prior to treatment and again at nine-month follow up. Participants who received SMR, EMG feedback, and the combination, all reported improvements in sleep at follow-up. However, these improvements did not differ significantly from the control (no treatment) group. The considerable amount of work and commitment on behalf of the participants in this study protocol may also enhance the expectation and placebo effects.

Despite this non-significant finding, Hauri [24] reported an interaction between treatment type and the type of insomnia. To further investigate this observation, participants in each of the three treatment groups were ranked according to self-reported tension at pre-treatment. Participants who were more tense prior to treatment, received the most benefits to self-reported sleep from EMG biofeedback. A similar pattern of results was reported for the EMG-theta group. In the SMR biofeedback group, those participants who were least tense at pre-treatment received the greatest therapeutic effect. It is worth noting, this *post hoc* finding was limited as it could not be evaluated statistically and may simply reflect the chance that one of many pre-treatment measures differentiates treatment efficacy. This finding would be best evaluated with a new study in which self-reported tension is a selection criteria prior to randomisation to either the appropriate treatment, inappropriate treatment or no treatment control.

The following year, Hauri et al's [30] group attempted to replicate the earlier findings. Sixteen individuals (mean age = 48.8 years) diagnosed with psychophysiological insomnia were randomly assigned to either SMR or theta feedback. Sleep was monitored immediately before treatment, after treatment and nine-months later. Sleep logs indicated increased total sleep time and decreased sleep onset latency for both SMR and theta feedback groups, improvements that were maintained at follow up ($p = 0.005$). Based on their earlier findings, the sample was split into 'relaxed' and 'tense' individuals, based on ratings of psychological anxiety [33] and physiological arousal (average EMG level over 1-minute assessed 5-minutes following lights out time during the initial three-night laboratory stay) assessed at pre-treatment.

Table 3
Characteristics of included studies.

Study	Study Design	Intervention	Number of Patients	Patient details	Comparator	Follow-Up	Limitations	Study quality ranking[a]
Cortoos et al. [28]	Randomised	SMR feedback (increase sensorimotor rhythm [12−15 Hz] and inhibit theta power [4−8 Hz] and higher beta power [20−30 Hz] at Cz. EMG feedback (decrease EMG power at Fpz). Training sessions 4 × 5mins every 2 −3 weeks for eight weeks.	N = 17 SMR feedback N = 9 EMG feedback N = 8	Insomnia (mean age 42.7 years, 65% male)	Nil	Two weeks	Small sample size, short term follow up, no control group	Participation + Attrition -Prognostic factor + Outcome measure + Confounders + Statistical analysis ±
Hammer et al. [29]	Randomised	SMR feedback (increase of sensorimotor rhythm (12−15 Hz) and inhibition of excessive theta (4 −8 Hz) and high beta (25−30 Hz). Individually designed program (normalise amplitudes of delta, theta, alpha and beta). Training session 15 × 20 mins	N = 8 SMR feedback N = 5 Individually designed protocol group N = 3	Insomnia (mean age 49.6 years)	Nil	6−9 months.	Small sample, no control group, ISI only at follow up.	Participation ± Attrition + Prognostic factor + Outcome measure + Confounders − Statistical analysis ±
Hauri [24]	Randomised	SMR feedback (increase of sensorimotor rhythm (12−15 Hz) and inhibition of excessive theta (4 −8 Hz) and high beta (25−30 Hz). EMG feedback (decrease EMG power). Combined EMG and theta feedback (increase theta power [4−7 Hz]).	N = 48	Insomnia	No treatment (control)	Nine months.	Subjective reports of sleep quality	Participation + Attrition + Prognostic factor + Outcome measure ± Confounders + Statistical analysis ±
Hauri et al. [30]	Randomised	SMR feedback (increase of sensorimotor rhythm (12−15 Hz) and inhibition of excessive theta (4 −8 Hz) and high beta (25−30 Hz). Theta feedback (increase theta power [4−7 Hz]). Training 26 sessions for SMR and Theta feedback, 6 sessions for EMG.	N = 16 SMR feedback N = 8 Theta feedback N = 8	Insomnia (mean age 48.8 years)	Nil	Nine months	Small sample size, no control group	Participation + Attrition ± Prognostic factor ± Outcome measure + Confounders ± Statistical analysis +
Schabus et al. [23]	Randomised, within-subjects counterbalanced	SMR feedback, (enhance EEG amplitude within the 12−15 Hz range) 10 training sessions (8 × 3 min each)	N = 24	Insomnia (mean age 34.5 years)	Sham-conditioning training	2−4 weeks	Follow up no EEG, no long term follow up. PSG assessment following first treatment also served as pre assessment for the subsequent treatment.	Participation + Attrition − Prognostic factor + Outcome measure ± Confounders ± Statistical analysis ±
Schabus et al. [31]	Double-blind cross over design, counterbalanced	SMR feedback (enhance EEG amplitude within the 12−15 Hz range) 12 training sessions (8 × 5 min each)	N = 30	Insomnia (mean age 38.6 years)	Sham-conditioning training	Three months	Two week retrospective assessment of subjective sleep quality. Control group included but did not undergo SMR or sham-conditioning training	Participation + Attrition − Prognostic factor + Outcome measure + Confounders ± Statistical analysis ±

[a] Quality ratings are based on the scale developed by Hayden et al., [27] and scored across the following domains study participation, study attrition, prognostic factor measurement, outcome measurement, confounding measurement, and analysis. + indicates low probability of bias, ± indicates neutral probability of bias, and − indicates high probabilty quality.

Significant improvements in sleep latency and efficiency from pre-treatment to follow up, as assessed by sleep logs, were reported by both 'tense' and 'relaxed' individuals irrespective of whether they received SMR or theta feedback. However, the lack of a control (no treatment) group, and participant selection based on self-reported tension, leaves the efficacy of SMR and theta feedback as well as any differential treatment efficacy based on self-reported tension, unresolved.

More recently, Schabus et al., [23] compared the sleep of 24 individuals with insomnia (mean age = 34.5 years) following SMR and sham-conditioning training (enhance EEG amplitude for randomized 3 Hz frequency bins between 7 Hz and 20 Hz [except 12–15 Hz]) using a randomised, counterbalanced, within-subjects design. Participants received a total of 15 training sessions, 10 for SMR and five for the sham-conditioning. Each training session was conducted in a standardized manner with participants completing 8 × 3 minute trials in the laboratory under the supervision of research staff. Improvements in subjective sleep quality were observed ($t_{23} = 7.75$, $p < 0.001$), however, these improvements did not differ following SMR when compared to sham-conditioning training. Notably, Schabus et al., [34] reports participants felt more socially supported when they received biofeedback, compared to sham-conditioning, in his earlier work [23] which suggests non-treatment specific factors may account for the observed improvements in subjective sleep quality.

Schabus et al., [31] later published a replication of their earlier work using a double-blind, counterbalanced, cross-over study design. SMR and sham-conditioning training (enhancement of random frequency ranges between 7 Hz and 20 Hz [except 12–15 Hz]) was administered to 30 individuals diagnosed insomnia (mean age = 38.6 years). Patients received a total of 24 training sessions, 12 sessions each for SMR and sham-condition training. The training sessions comprised 8 × 5 minute trials conducted under the supervision of research staff in a standardized manner. Although subjective sleep complaints decreased significantly following SMR, this decline was not specific to SMR and did not differ significantly when compared to sham-conditioning training ($F_{1,21} = 0.17$, $p = 0.69$, $np^2 = 0.008$). Subjective total sleep time and sleep onset latency remained unchanged following both SMR and sham-conditioning training.

3.5. Objective sleep quality

Hauri and colleagues [24] were the first to assess objective sleep quality when evaluating the efficacy of biofeedback. No significant improvements were observed in any objective measure of sleep as measured by polysomnography (PSG) following either SMR or theta feedback for the group. Based on their earlier findings [35], the sample was split into 'relaxed' and 'tense' individuals (as described previously) [30]. Significant improvements in objective sleep latency, TST and SE were observed but only when individuals received the biofeedback that was appropriate for their level of reported arousal. Individuals who rated themselves as 'relaxed' showed objective therapeutic benefits in response to SMR but not theta feedback, while the contrary was demonstrated for the 'tense' individuals. These improvements were maintained at nine-month follow up. Although the improvements were typically modest (10-min reduction in SOL, 30-min improvement in TST, 5% increase in SE), they are promising given objective measures of sleep quality and duration usually show little change with treatment [10].

Since the pioneering work of Hauri [24], biofeedback has been reported to improve objective sleep onset latency, number of awakenings, duration of slow wave sleep and total sleep time [23,28]. Cortoos and colleagues [28] randomised 17 participants to receive either SMR or EMG feedback. Both groups received 20-minutes (4 × 5 min) of training every 2–3 weeks for a total duration of eight weeks in their home environment. Laboratory-based PSG was used to assess sleep at pre-treatment and two weeks following treatment. Significant improvements were shown for sleep latency, which decreased from pre to post treatment in both groups ($F_{1,15} = 10.56$; $p = 0.01$; $r = 0.41$), but a significant interaction effect demonstrated an increase in TST was only found for the SMR group ($F_{1,15} = 5.03$; $p = 0.05$; $r = 0.25$). The magnitude of these improvements is unclear due to the omission of mean values included in the published paper. The inclusion of a sham-conditioning control group is particularly important within the biofeedback arena where the sophisticated appearance of the hardware and scientific plausibility can contribute to high placebo effect. Additionally, the lack of a no treatment control group limits any firm conclusions about the efficacy of SMR without accounting for the potential role of non-treatment specific effects.

Schabus et al., [23] assessed the objective sleep of 24 individuals following SMR and sham-conditioning training using a randomized, within-subjects design with the order of conditions counterbalanced. They reported a trend towards improvement in objective sleep onset latency. Improvements in the number of awakenings ($F_{2,40} = 4.03$, $p = 0.025$) and duration of slow wave sleep ($F_{2,40} = 4.24$, $p = 0.021$) following SMR when compared to sham-conditioning were also demonstrated. However, both the number of awakenings and duration of slow wave sleep showed a reversal when the other condition was administered. The remainder of objective sleep outcome variables assessed remained unchanged following SMR and did not differ from sham-conditioning. The failure of the application of any multiple comparisons corrections (at least 14 separate statistical tests were used) and the use of 1-tailed probability limit the interpretation of the improvements reported in number of awakenings and duration of slow wave sleep following SMR.

Schabus et al., [31] aimed to replicate their earlier work adopting a rigorous double-blind, counterbalanced, cross-over study design to overcome the limitations of earlier work. Objective sleep parameters, including SOL, WASO, NWAK, TST, TIB, SE, and percentage of each sleep stage (N1–N3 and REM), were assessed prior to and following SMR and sham-conditioning training in a group of 30 individuals with insomnia. No significant changes were observed in any measure of objective sleep following SMR or sham-conditioning training. These results suggest the positive findings that have been reported for SMR in the earlier single-blind study of Schabus et al., [23] are likely not specific to SMR but may be attributed to other non-treatment specific effects such as care, attention and support from research staff. In fact, Schabus reports participants felt more socially supported when they received biofeedback, compared to sham-conditioning, in their earlier protocol. This lends support to the role of other non-treatment specific effects in the observed positive findings in their initial work.

3.6. Quality of life and daytime functioning

The assessment of quality of life and daytime functioning following the use of biofeedback is limited. Of the included studies, only three included outcome measures of quality of life and daytime functioning [23,29,31]. The work of Hammer and colleagues [29] and Schabus et al., [23] demonstrated significant improvements in overall quality of life following both SMR and an individualised treatment program (aimed at normalizing amplitudes of delta, theta, alpha and beta). Despite the limited number of studies assessing changes in quality of life, support for the use of biofeedback for the improvement of self-reported quality of life is provided. In two separate studies, Schabus and colleagues [23,31] investigated the use of SMR for memory consolidation using a declarative word-pair association task [36,37].

3.7. Perceived insomnia severity

To date, Hammer and colleagues [29] have been the only group to assess the efficacy of biofeedback for improving perceived insomnia severity. Eight individuals diagnosed with insomnia were randomized to receive SMR (n = 5) or an individually designed treatment program (n = 3). The individually designed treatment program aimed at normalising abnormal amplitudes of delta, theta, alpha and beta, in addition to asymmetry, coherence and phase lag between the EEG sites, as determined by z-scores derived from pre-treatment qualitative EEG. Each group received 20-minute training sessions on 15 occasions under the supervision of the investigators. Perceived insomnia severity was assessed using the Insomnia Severity Index (ISI) [32] at pre- and post-treatment as well as 6–9 months later. Significant reductions in the ISI were demonstrated from pre- ($M = 17.1$, 95%$CI = 15.7–18.5$) to post-treatment ($M = 6.6$, 95%CI = 5.9–7.2). At post-treatment, all eight completers scored below the clinical cut-off (ISI < 10) for insomnia. The six participants who completed the study sustained these improvements at follow up with a mean ISI score of 7. However, there were no significant differences between SMR and the individually designed treatment program groups. It is worth noting the frequency and intensity of individual contact employed in this study was likely to maximise the placebo effect. Without a control group, placebo effects are unable to be differentiated from the potential therapeutic effects of SMR or the individually designed treatment program.

3.8. Limitations and future research directions

The limitations of the current review should be considered when interpreting these outcomes. A total of 93 publications were identified on the topic of biofeedback for the treatment of insomnia, yet only six fulfilled criteria for inclusion in this review. The large majority of published literature provides general background information about the proposed mechanisms involved in biofeedback, historical accounts of biofeedback, or reviews other datasets without the inclusions of any original research. Furthermore, of the peer reviewed original research publications retrieved, many did not assess the efficacy of biofeedback using an appropriate sample (ie, participants meeting the diagnostic criteria for insomnia according to the Diagnostic and Statistical Manual of Mental Disorders [3] or for earlier studies the diagnostic criteria relevant at the time of publication). This is an essential requirement when establishing the efficacy of biofeedback for the treatment of insomnia disorder. Study design was not used as a basis to select studies for inclusion into the current review. The authors acknowledge that limiting the review to the results of randomised controlled trials (RCTs) on biofeedback would be ideal, but at present published RCTs are rare. It is recognised that publication bias may play a role in supporting the notion that biofeedback is an efficacious treatment for insomnia simply because negative findings are likely under-represented in the published literature [34,38,39]. The authors acknowledge a meta-analysis of these studies would be ideal, however the limited number of published papers resulted in fundamental difficulties in doing so including extremely small sample sizes in each group, and a general lack of control groups accounting for unspecific effects.

There is a fundamental need to improve study designs to advance this field, particularly to understand the neural mechanisms involved and develop optimal administration protocols. The use of rigorously controlled studies, ideally double-blind designs, including plausible placebo controls [34,38,40–43]. Sham-conditioning would be ideal. The inclusion of objective data is required to assess the efficacy of different types of biofeedback [40]. The use of double-blind designs incorporating functional electroencephalography (EEG) and Magnetic Resonance Imaging (MRI) to demonstrate systematic changes in the brain in response to different types and administration protocols of biofeedback, and the use of machine learning to promote a more accurate and descriptive understanding of neural signatures of key mental states is encouraged [34,39]. Another possible consideration for future research is a re-evaluation of the types of bio-feedback used for the treatment of insomnia. There may be other possible candidate protocols which may well offer greater possible benefits for sleep (eg, increased HR variability, increasing alpha amplitude followed by increased theta, increasing alpha followed by hypnagogic imagery, etc.).

Future research is also urged to carefully consider the type of control group used [41]. Of the few studies that have included a control group, most involve 'replaying' the biofeedback received by a participant to a participant in the control condition [34]. Schabus [34] highlights this type of control group is highly problematic as it promotes learned helplessness. Over extended periods, participants in the control group learn they have no control over the feedback they receive likely inducing negative training effects in the control participants. Ideally the feedback given to the control group and active treatment group should be closely matched as demonstrated in Schabus and colleagues [31] most recent study. Ideally, protocols should be designed to include not only the active biofeedback group, but also a credible placebo (active control) and an inactive control (no feedback/scientific apparatus [44], contact with research staff etc.). The best example of this ideal scenario is demonstrated in the recent work of Schabus and colleagues [31] who used a rigorous double-blind, counterbalanced, cross-over study design including an active biofeedback group and an active control. The inclusion of an inactive control in future protocols would further strengthen conclusions about the efficacy of biofeedback and undoubtedly rule out the possibility of any placebo or non-specific treatment effects [39]. Of course the costs of including this control arm would need to be weighed against the potential addition of scientific value in terms of the outcomes. As suggested by Thibault and colleagues [41], other factors likely to influence behavioural changes (eg, the amount of positive feedback) should also be matched between the active and control group. The assessment of non-specific treatment factors, including motivation, sense of control over the signal, confidence in the therapeutic effects of the protocol and believed group assignments, are also required to confirm the effectiveness of the control groups [38,40,41,45]. The field is also encouraged to work to develop standardization for data acquisition and analysis, and pre-register studies and main hypotheses [34,38,39].

4. Conclusions

Despite the 36-year history of biofeedback for the treatment of insomnia, there are very few investigations and well-designed experimental studies. Although improvements in sleep, quality of life and daytime functioning, and perceived insomnia severity are demonstrated in some studies, poor study design (small sample size, lack of control group) ultimately limits confidence in the strength of evidence for a therapeutic effect of biofeedback for insomnia and leaves open the possibility of a potential role of placebo effects [38–40,42,43,46]. Of the six studies reviewed, the recent work of Schabus and colleagues [32] utilized a sound study design and in doing so provides promising support for the use of SMR for the treatment of insomnia but cautions the therapeutic effects may not be specific to SMR but other unspecified factors likely to drive the observed changes, such as care and attention of those administering the treatment, as well as the expectations and motivations of the patient [39,45,46]. Hauri and colleagues [24,30]

have suggested that biofeedback may be beneficial for certain subtypes of insomnia. The confirmation of the ideas of Hauri and colleagues is an avenue ripe for future research ideally explored with multisite studies using experimental designs whereby patients are randomised on the basis of self-reported tension to either the appropriate treatment, inappropriate treatment or no treatment control.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.12.011.

References

[1] Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6(2):97–111.
[2] American Academy of Sleep Medicine. The international classification of sleep disorders: diagnostic and coding manual. 2nd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
[3] American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, D.C: American Psychiatric Association; 2013.
[4] Miller CB, Valenti L, Harrison CM, et al. Time trends in the family physician management of insomnia: the Australian experience (2000–2015). J Clin Sleep Med 2017;13(6):785–90. https://doi.org/10.5664/jcsm.6616.
[5] Riemann D, Perlis M. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioural therapies. Sleep Med Rev 2009;13:205–14.
[6] Benca RM. Diagnosis and treatment of chronic insomnia: a review. Psychiatr Serv 2005;56(3):332–43. doi:56/3/332 [pii]1176/appi.ps.56.3.332.
[7] Lovato N, Lack L, Wright H, et al. Evaluation of a brief treatment program of cognitive behavior therapy for insomnia in older adults. Sleep 2014;37(1):117–26. https://doi.org/10.5665/sleep.3320.
[8] Sweetman A, Lack L, Lambert S, et al. Does comorbid obstructive sleep apnea impair the effectiveness of cognitive and behavioral therapy for insomnia? Sleep Med 2017;39:38–46. https://doi.org/10.1016/j.sleep.2017.09.003.
[9] Trauer JM, Qian MY, Doyle JS, et al. Cognitive behavioral therapy for chronic insomnia: a systematic review and meta-analysis. Ann Intern Med 2015;163(3):191–204. https://doi.org/10.7326/M14-2841.
[10] van Straten A, van der Zweerde T, Kleiboer A, et al. Cognitive and behavioral therapies in the treatment of insomnia: a meta-analysis. Sleep Med Rev 2018;38:3–16. https://doi.org/10.1016/j.smrv.2017.02.001.
[11] Morin CM, Colecchi C, Stone J, et al. Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. JAMA 1999;281(11):991–9.
[12] Freedman RR. EEG power spectra in sleep-onset insomnia. Electroencephalogr Clin Neurophysiol 1986;63(5):408–13.
[13] Perlis ML, Giles DE, Mendelson WB, et al. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6(3):179–88.
[14] Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. Sleep Med Rev 2010;14(1):19–31. doi:S1087-0792(09)00041-0 [pii] 10.1016/j.smrv.2009.04.002.
[15] Buysse DJ, Germain A, Hall ML, et al. EEG spectral analysis in primary insomnia: NREM period effects and sex differences. Sleep 2008;31(12):1673–82.
[16] Perlis M, Smith M, Andrews P, et al. Beta/Gamma activity in patients with primary and secondary insomnia and good sleeper controls. Sleep 2001;24(1):110–7.
[17] Perlis ML, Kehr EL, Smith MT, et al. Temporal and stagewise distribution of high frequency EEG activity in patients with primary and secondary insomnia and in good sleeper controls. J Sleep Res 2001;10(2):93–104.
[18] Nofzinger EA, Nissen C, Germain A, et al. Regional cerebral metabolic correlates of WASO during NREM sleep in insomnia. J Clin Sleep Med 2006;2(3):316–22.
[19] Bonnet M, Arand D. Heart rate variability in insomniacs and matched normal sleepers. Psychosom Med 1998;60:610–5.
[20] Rodenbeck A, Cohrs S, Jordan W, et al. The sleep-improving effects of doxepin are paralleled by a normalized plasma cortisol secretion in primary insomnia. A placebo-controlled, double-blind, randomized, cross-over study followed by an open treatment over 3 weeks. Psychopharmacology 2003;170(4):423–8.
[21] Stepanski E, Glinn M, Zorick F, et al. Heart rate changes in chronic insomnia. Stress Med 1994;10:261–6.
[22] Vgontzas A, Bixler E, Lin H, et al. Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications. J Clin Endocrinol Metab 2001;86(8):3787–94.
[23] Schabus M, Heib DP, Lechinger J, et al. Enhancing sleep quality and memory in insomnia using instrumental sensorimotor rhythm conditioning. Biol Psychol 2014;95:126–34. https://doi.org/10.1016/j.biopsycho.2013.02.020. S0301-0511(13)00094-X [pii].
[24] Hauri P. Treating psychophysiologic insomnia with biofeedback. Arch Gen Psychiatry 1981;38(7):752–8.
[25] Sterman MB, Howe RC, MacDonald LR. Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. Science 1970;167:1146–8.
[26] Moher D, Liberati A, Tetzlaff J, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. PLoS Med 2009;6(7), e1000097. https://doi.org/10.1371/journal.pmed.1000097.
[27] Hayden JA, Cote P, Bombardier C. Evaluation of the quality of prognosis studies in systematic reviews. Ann Intern Med 2006;144:427–37.
[28] Cortoos A, De Valck E, Arns M, et al. An exploratory study on the effects of tele-neurofeedback and tele-biofeedback on objective and subjective sleep in patients with primary insomnia. Appl Psychophysiol Biofeedback 2010;35(2):125–34. https://doi.org/10.1007/s10484-009-9116-z.
[29] Hammer BU, Colbert AP, Brown KA, et al. Neurofeedback for insomnia: a pilot study of Z-score SMR and individualized protocols. Appl Psychophysiol Biofeedback 2011;36(4):251–64. https://doi.org/10.1007/s10484-011-9165-y.
[30] Hauri PJ, Percy L, Hellekson C, et al. The treatment of psychophysiologic insomnia with biofeedback: a replication study. Biofeedback Self Regul 1982;7(2):223–35.
[31] Schabus M, Griessenberger H, Gnjezda MT, et al. Better than sham? A double-blind placebo-controlled neurofeedback study in primary insomnia. Brain 2017;140(4):1041–52. https://doi.org/10.1093/brain/awx011.
[32] Bastien C, Vallieres A, Morin C. Validation of the insomnia severity index as an outcome measure for insomnia research. Sleep Med 2001;2:297–307.
[33] Cattell RB. The dimensional (unitary-component) measurement of anxiety, excitement, effort stress, and other mood reaction patterns. In: Ohr, Miller, editors. Drugs and behavior. New York: Wiley; 1960.
[34] Schabus M. Reply: on assessing neurofeedback effects: should double-blind replace neurophysiological mechanisms? Brain 2017;140. doi:ARTN e64 1093/brain/awx212.
[35] Hauri P, editor. Sleep hygiene, relaxation therapy, and cognitive interventions; 1991.
[36] Hoedlmoser K, Pecherstorfer T, Gruber G, et al. Instrumental conditioning of human sensorimotor rhythm (12–15 Hz) and its impact on sleep as well as declarative learning. Sleep 2008;31(10):1401–8.
[37] Schabus M, Hodlmoser K, Pecherstorfer T, et al. Influence of midday naps on declarative memory performance and motivation. Somnologie 2005;9:148–53.
[38] Thibault RT, Lifshitz M, Birbaumer N, et al. Neurofeedback, self-regulation, and brain imaging: clinical science and fad in the service of mental disorders. Psychother Psychosom 2015;84(4):193–207. https://doi.org/10.1159/000371714.
[39] Thibault RT, Lifshitz M, Raz A. The climate of neurofeedback: scientific rigour and the perils of ideology. Brain 2018;141(2):e11. https://doi.org/10.1093/brain/awx330.
[40] Schonenberg M, Wiedemann E, Schneidt A, et al. Neurofeedback, sham neurofeedback, and cognitive-behavioural group therapy in adults with attention-deficit hyperactivity disorder: a triple-blind, randomised, controlled trial. Lancet Psychiatry 2017;4(9):673–84. https://doi.org/10.1016/S2215-0366(17)30291-2.
[41] Thibault RT, Lifshitz M, Raz A. The self-regulating brain and neurofeedback: experimental science and clinical promise. Cortex 2016;74:247–61. https://doi.org/10.1016/j.cortex.2015.10.024.
[42] Thibault RT, Lifshitz M, Raz A. Neurofeedback or neuroplacebo? Brain 2017;140(4):862–4. https://doi.org/10.1093/brain/awx033.
[43] Thibault RT, Raz A. Neurofeedback: the power of psychosocial therapeutics. Lancet Psychiatry 2016a;3(11):e18. https://doi.org/10.1016/S2215-0366(16)30326-1.
[44] Thibault RT, Veissiere S, Olson JA, et al. Treating ADHD with suggestion: neurofeedback and placebo therapeutics. J Atten Disord 2018;22(8):707–11. https://doi.org/10.1177/1087054718770012.
[45] Thibault RT, Raz A. When can neurofeedback join the clinical armamentarium? Lancet Psychiatry 2016b;3(6):497–8. https://doi.org/10.1016/S2215-0366(16)30040-2.
[46] Thibault RT, Raz A. The psychology of neurofeedback: clinical intervention even if applied placebo. Am Psychol 2017;72(7):679–88. https://doi.org/10.1037/amp0000118.