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Patterns of polysomnography parameters in 27 neuropsychiatric diseases: an umbrella review

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Abstract

Background. We provide an umbrella review of the reported polysomnographic changes in patients with neuropsychiatric diseases compared with healthy controls.

Methods. An electronic literature search was conducted in EMBASE, MEDLINE, All EBM databases, CINAHL, and PsycINFO. Meta-analyses of case–control studies investigating the polysomnographic changes in patients with neuropsychiatric diseases were included. For each meta-analysis, we estimated the summary effect size using random effects models, the 95% confidence interval, and the 95% prediction interval. We also estimated between-study heterogeneity, evidence of excess significance bias, and evidence of small-study effects. The levels of evidence of polysomnographic changes in neuropsychiatric diseases were ranked as follows: not significant, weak, suggestive, highly suggestive, or convincing.

Results. We identified 27 articles, including 465 case–control studies in 27 neuropsychiatric diseases. The levels of evidence of polysomnographic changes in neuropsychiatric diseases were highly suggestive for increased sleep latency and decreased sleep efficiency (SE) in major depressive disorder (MDD), increased N1 percentage, and decreased N2 percentage, SL and REML in narcolepsy, and decreased rapid eye movement (REM) sleep percentage in Parkinson's disease (PD). The suggestive evidence decreased REM latency in MDD, decreased total sleep time and SE in PD, and decreased SE in posttraumatic stress disorder and in narcolepsy.

Conclusions. The credibility of evidence for sleep characteristics in 27 neuropsychiatric diseases varied across polysomnographic variables and diseases. When considering the patterns of altered PSG variables, no two diseases had the same pattern of alterations, suggesting that specific sleep profiles might be important dimensions for defining distinct neuropsychiatric disorders.

Introduction

Neuropsychiatric diseases are significant causes of disability and death throughout the world (GBD 2016 Neurology Collaborators, 2019; GBD 2015 Neurological Disorders Collaborator Group, 2017; Vigo, Thornicroft, & Atun, 2016) and they take a large toll on individuals, families and health-care systems (GBD 2019 Diseasesand Injuries Collaborators, 2020; The Lancet, 2017). Sleep disturbances are frequent complaints in patients with neuropsychiatric diseases. Historically, sleep disturbances were viewed as clinical symptoms which result from the pathology of neuropsychiatric diseases. However, increasing evidence suggests a complex interrelationship and potential bidirectional causality between sleep disturbances and these diseases (Krystal, 2020). Sleep disturbances longitudinally predict the development of psychiatric diseases and neurological disorders (i.e. in depression, anxiety, and neurodegeneration) (Galbiati, Verga, Giora, Zucconi, & Ferini-Strambi, 2019; Hertenstein et al., 2019; Shi et al., 2018). Some treatments for sleep disturbances improve the symptoms of neuropsychiatric conditions, and vice versa, treating neuropsychiatric diseases may also affect sleep (Krystal, 2020). These findings suggest that clarifying the relationships between sleep and neuropsychiatric diseases may be helpful for understanding the pathology of the diseases and for improving their clinical management (Krystal, 2020).

Polysomnography (PSG) is the gold standard method for objectively assessing sleep features in clinical and non-clinical settings. PSG measured sleep reflects neurophysiological functioning in humans. For instance, evidence supports slow wave sleep's (SWS) role in energy restoration, clearing metabolites, hormone release, immunity, and memory consolidation (Leger et al., 2018). Rapid eye movement (REM) sleep helps maintain neuronal homeostasis in the brain as disturbances of REM sleep can affect brain excitability, synaptic pruning, and neurogenesis, and loss of REM sleep can lead to neurodegeneration (Chauhan & Mallick, 2019). Thus, investigating and comparing PSG sleep variables across neuropsychiatric diseases has

the potential to reveal neurobiological mechanisms of specific disorders and to reveal neural commonalities and differences that may help refine diagnostic categories and may have implications for more effective clinical management (Baglioni et al., 2016a).

Many case–control studies have reported various PSG changes for different neuropsychiatric diseases, and meta-analyses of PSG changes in some neuropsychiatric diseases have been published. Meta-analytic approaches are typically considered as the highest rank of evidence and can provide a more accurate 'big picture' for disease characteristics. However, they can also introduce confusion into the literature due to the low methodological standards of some published meta-analyses and, perhaps more importantly, of their included studies (Solmi, Correll, Carvalho, & Ioannidis, 2018). Thus, poorly conducted meta-analytic studies with their potentially flawed findings may obscure rather than clarify the state of science for a particular question (Ioannidis, 2016; Ioannidis, 2017). Specifically, meta-analyses are susceptible to reporting bias, publication bias, and residual confounding bias, and other types of problems which can result in inflated estimates (Ioannidis, 2008) or false positives (Ioannidis, 2005) for examined data parameters. These types of flaws have resulted in an excess of significant associations ($p < 0.05$) in psychological science and other medical fields (Boffetta et al., 2008; Ioannidis, Munafo, Fusar-Poli, Nosek, & David, 2014) that may have obscured the most important or distinguishing characteristics for a given disorder. Thus, it is important to comprehensively evaluate evidence from meta-analyses to minimize such quality concerns (Ioannidis, 2009, 2016).

An umbrella review, which summarizes, assesses, and grades the findings of multiple meta-analyses, is a standardized and systematic collection of data from studies on a specific topic (Fusar-Poli, Hijazi, Stahl, & Steyerberg, 2018; Ioannidis, 2009). This approach to data review allows a higher-level synthesis of the evidence and a better recognition of the uncertainties, weaknesses, various kinds of bias, and strengths of the available evidence (Bougioukas et al., 2019). Compared with the meta-analytic approach, which is usually restricted to one single topic, umbrella reviews have advantages because they can examine evidence across a broad and high-quality database and provide a comprehensive overview of a specific topic (Aromataris et al., 2015; Ioannidis, 2009). This capability has led to an increasing emphasis being placed to umbrella reviews to best address the extensive literature of complex neuropsychiatric science and other medical fields (Barbui et al., 2020; Hailes, Yu, Danese, & Fazel, 2019; Ioannidis, 2017).

To our knowledge, to date, no umbrella review has been conducted on the topic of PSG changes in neuropsychiatric diseases. Given the role that sleep plays in essentially all these diseases, such a review may provide unique insight into sleep changes across diseases. Therefore, we performed this first umbrella review of relevant meta-analyses of case–control studies and attempted to provide a comprehensive overview and examination of the strength of evidence, precision of the estimates, presence of biases, and robustness of the published PSG changes in patients with neuropsychiatric diseases compared with healthy controls (HCs).

Methods

This umbrella review was done following the PRISMA reporting guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009) and its protocol was registered (PROSPERO ID: CRD42020202318).

Search strategy, study selection, and eligibility criteria

The following terms were searched for in abstract or title: ('meta-analy*' or 'metaanaly*' or 'meta-analysis' or 'meta analy*') AND ('polysomnogra*' OR 'PSG' OR 'sleep architect*' OR 'sleep monit*' OR 'sleep stage*' OR 'electroencephalogra*' OR 'EEG'). The detailed search strategies used for each literature database are provided in online Supplementary Tables S1–S5. We initially searched MEDLINE, EMBASE, PsycINFO, and CINAHL, and All EBM databases from inception to 26 Nov 2020, to identify systematic reviews and meta-analyses of case–control studies exploring PSG changes in patients with neuropsychiatric diseases compared with non-neuropsychiatric HCs. We updated the literature search using the same search strategies on 28 Mar 2022, to find any newly published meta-analyses. Two investigators (YZ and RR), with a good inter-rater agreement for potentially eligible studies (Kappa = 0.837), independently selected the potential eligible articles. The references of relevant studies were manually screened to identify eligible articles. Any disagreements were discussed by three authors (YZ, RR, and XDT) to reach a final decision.

The included studies meet the following eligibility criteria: (1) the participants were patients with mental illnesses (including but not limited to depression, generalized anxiety disorder, schizophrenia, bipolar disorder, etc.) or neurological diseases [including but not limited to stroke, epilepsy, Parkinson's disease (PD), Huntington's disease (HD), etc.]. The diagnosis of mental illnesses was according to any edition of the Diagnostic and Statistical Manual of Mental Disorders or International Classification of Diseases criteria or a structured psychiatric diagnostic interview. The diagnosis of neurological disease was also according to established criteria (e.g. diagnosing PD according to Brain Bank criteria); (2) differences in PSG parameters (i.e. total sleep time (TST), wake time after sleep onset, sleep efficiency (SE), sleep latency (SL), and percentage of N1, N2, SWS and REM sleep, REM latency, periodic limb movement index, apnea hypopnea index, arousal index, cyclic alternating pattern (CAP) parameters, or power spectral data) between patients with neuropsychiatric diseases and non-neuropsychiatric HCs were explored by meta-analysis. The eligible articles were published in peer-reviewed journals with no language restrictions. The exclusion criteria are provided on online Supplementary Appendix pp3.

Data extraction

Data extraction was done independently by two investigators (YZ and RR) with a high inter-rater percentage agreement (99.5%). In the case of discrepancies, three investigators (YZ, RR and XDT) discussed the concerns and made the final decision. From each eligible article, we recorded the first author, year of publication, disease names, and number of comparisons included. If a quantitative synthesis was done, we extracted the study-specific estimated effect size of differences in PSG parameters between cases and HCs together with their corresponding 95% confidence intervals (CIs) and the number of cases and HCs in each study. If the eligible article only reported the pooled effect sizes and did not report the study-specific effect size, we extracted the study-specific effect size from the included individual component studies of each eligible article and then re-estimated their effect sizes. In one eligible article (Cox & Olatunji, 2020) which integrated various PSG parameters into three variables (sleep continuity,

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sleep depth, and REM pressure) but did not report detailed data on sleep continuity and sleep architecture (i.e. TST, SL, SE, N1, N2, SWS, and REM sleep), we also extracted the study-specific effect size from the individual component studies. Metrics followed those of the original meta-analyses [i.e. mean difference, standardized mean difference (SMD), or Hedge's g].

Quality assessments

AMSTAR 2 (A Measurement Tool to Assess Systematic Reviews), which has good inter-rater agreement, content validity, and test-retest reliability was used to assess the methodological quality of the meta-analyses (Shea et al., 2017). The domains which AMSTAR 2 evaluates and the detailed methods for use of AMSTAR 2 are provided on online Supplementary Appendix pp6. Two reviewers (YZ and RR) independently used AMSTAR 2 to assess the meta-analyses and the inter-rater agreement was good (Kappa = 0.82). Any disagreements were discussed by three authors (YZ, RR, and XDT) to reach a final decision.

Data analysis

Summary SMDs with 95% CI were re-estimated using common metric random effects methods (DerSimonian & Laird, 1986). The heterogeneity between studies was evaluated using Cochran's Q test (Cochran, 1954) and the I^2 statistic ($I^2 > 50\%$ indicates high heterogeneity) (Higgins, Thompson, Deeks, & Altman, 2003). We estimated the 95% prediction interval, the range in which we expect the PSG differences between groups will lie for 95% of future studies (Higgins, Thompson, & Spiegelhalter, 2009).

We noted when prediction intervals excluding the null value (0 in the case of SMDs) suggest that the statistically significant PSG changes in patients with neuropsychiatric diseases are likely to persist in future studies. We assessed whether there was evidence for small-study effects (i.e. whether smaller studies tend to give substantially larger estimates of effect size compared with larger studies) with the regression asymmetry test proposed by Egger et al. (Egger, Davey Smith, Schneider, & Minder, 1997). A p value less than 0.1 occurring in conjunction with more conservative effect sizes in larger studies compared with that found in the in random effects meta-analysis was judged to be evidence for small-study effects.

We evaluated the existence of excess significance bias to examine whether the observed number of studies with statistically significant results (positive studies, $p < 0.05$) in each meta-analysis was larger than their expected number (Ioannidis & Trikalinos, 2007). For each meta-analysis, the expected number was calculated as the sum of the statistical power estimates for each study in the meta-analysis. The power of each original case–control study was calculated by an algorithm using a non-central t distribution (Lubin & Gail, 1990), which is necessary for evaluating excess significance bias. The estimated power depends on the plausible SMD. Because the true SMD for any meta-analysis is unknown, we assumed that the most plausible effect is given by the largest study (smallest standard error) (Ioannidis, 2013). Excess significance bias for each meta-analysis was determined at a p value less than 0.10 (Ioannidis & Trikalinos, 2007).

Statistical analyses were conducted using Comprehensive Meta-Analysis software version 2.0 and STATA version 14.0. Power calculations were done in R version 3.5.1 and the pwr package. All p values were two tailed.

Credibility of evidence

As with earlier umbrella reviews (Barbui et al., 2020; Belbasis, Bellou, Evangelou, Ioannidis, & Tzoulaki, 2015; Kim et al., 2019, 2020), we classified the strength of PSG changes in each neuropsychiatric disease as convincing (class I), highly suggestive (class II), suggestive (class III), weak (class IV) or not significant (NS). Convincing evidence required p values in random effects models below 10^{-6}, number of cases > 1000, the largest study nominally significant ($p < 0.05$), no evidence of small-study effects, no large heterogeneity (i.e. $I^2 < 50\%$), no evidence of excess of significance bias, and 95% prediction intervals not including the null value. Highly suggestive evidence required p values $< 10^{-6}$, number of cases > 1000, and the largest study nominally significant ($p < 0.05$). Suggestive evidence required p values $< 10^{-3}$ and number of cases > 1000. Weak evidence required no specific number of cases and $p < 0.05$. For PSG comparisons classified as convincing, highly suggestive, or suggestive, we attempted further assessment for the robustness of the evidence by subset analyses limited to individual component studies that excluded patients taking medications impacting sleep, studies excluding patients with other psychiatric comorbidities, and studies using different PSG scoring methods [Rechtschaffen and Kales (R&K) *v.* American Academy Sleep Medicine (AASM)].

Results

Study selection

Our search identified 3537 publications. After removing duplicates and screening titles and abstracts, 64 full-text articles were assessed for eligibility. Twenty-seven systematic reviews (Baglioni et al., 2014, 2016a, 2016b; Bertrand et al., 2021; Biancardi, Sesso, Masi, Faraguna, & Sicca, 2021; Chan, Chung, Yung, & Yeung, 2017; Chen et al., 2021; Cox & Olatunji, 2020; D'Rozario et al., 2020; Díaz-Román, Hita-Yanez, & Buela-Casal, 2016; Keenan, Sherlock, Bramham, & Downes, 2021; Lugo et al., 2020; Mantua et al., 2018; Ng et al., 2015; Plante, 2018; Stanyer, Creeney, Nesbitt, Holland, & Hoffmann, 2021; Winsor et al., 2021; Winsper et al., 2017; Xu et al., 2020; Yeh et al., 2022a, 2022b; Zhang et al., 2019a, 2019b, 2020a, 2021, 2022; Zhang, Ren, Yang, Sanford, & Tang, 2020b), including 465 case–control studies, met inclusion criteria (Fig. 1). Details of the reviews excluded, and the reasons for exclusion, are provided in online Supplementary Table S6.

Description of the included systematic reviews and meta-analyses

From these 27 included systematic reviews, we extracted information on 321 pooled analyses exploring sleep macrostructure changes in 27 neuropsychiatric diseases compared with HCs (Table 1). Of the 321 pooled analyses of sleep macrostructure, there were 10 on schizophrenia, 9 on bipolar disorder, 12 on major depressive disorder (MDD), 10 on generalized anxiety disorder, 9 on obsessive compulsive disorder, 10 on panic disorder, 6 on social anxiety disorder (SAD), 10 on borderline personality disorder, 9 on insomnia, 10 on adult attention deficit hyperactivity disorder (ADHD), 12 on childhood ADHD, 9 on adult autism spectrum disorder (ASD), 10 on childhood ASD, 8 on anorexia nervosa, 10 on posttraumatic stress disorder (PTSD), 10 on stroke, 12 on mild cognitive impairment, 11 on traumatic brain injury, 12 on idiopathic REM sleep behavior disorder, 11 on idiopathic

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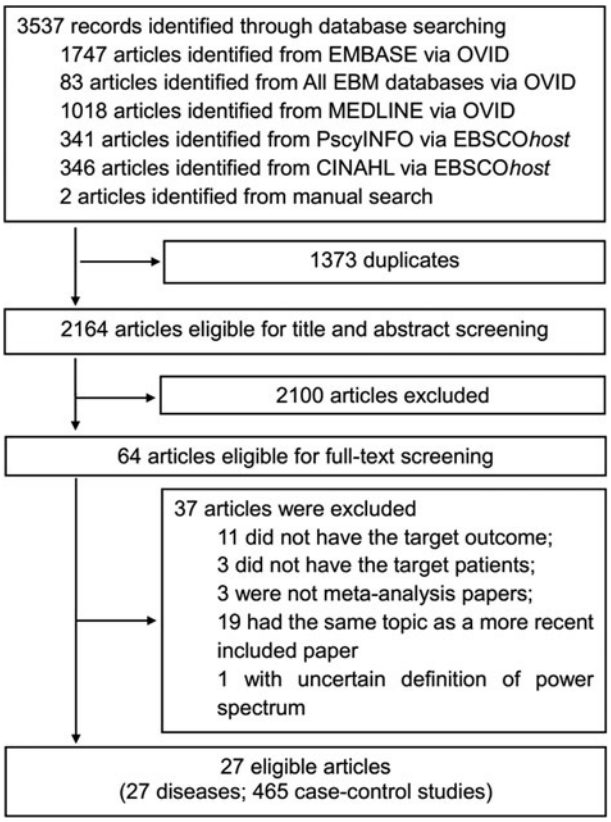

Fig. 1. Flow chart of literature search.

hypersomnia, 12 on HD, 13 on PD, 12 on Wilson's disease (WD), 12 on narcolepsy, 12 on Alzheimer's disease, 7 on seasonal affective disorder, 11 on adult migraine, 11 on child migraine, 10 on child and adolescent epilepsy, 12 on adult epilepsy, and 9 on persistent tic disorder. The 321 pooled analyses of sleep macrostructure were based on 27 neuropsychiatric diseases, 191 061 total participants, a median 135 neuropsychiatric cases per pooled analysis (interquartile range (IQR) 77–355, range 27–1663), and a median 285 total participants per pooled analysis (IQR 152–862, range 50–2975). As shown in Fig. 2, the overall patterns of sleep changes varied widely across different diseases. Furthermore, there were a total of 35 pooled analyses exploring sleep microstructure changes (CAP parameters) within three neuropsychiatric diseases (7 on narcolepsy, 23 on ADHD, and 5 on epilepsy; see descriptions in online Supplementary Table S7). The means for polysomnographic parameters in patients with neuropsychiatric diseases and HCs are provided in online Supplementary Table S8. The quality assessments of included systematic reviews and meta-analyses are provided on online Supplementary Appendix pp6.

Main analyses

For the main analyses of sleep macrostructural data, one hundred and forty-seven (45.8%) of 321 pooled analyses were statistically significant with $p < 0.05$, 73 (22.7%) with $p < 0.001$, and 30 (9.3%) with $p < 0.000001$. 21 (14.3%) of 147 statistically significant pooled analyses included more than 1000 neuropsychiatric cases per disease. 146 (45.5%) of 321 comparisons showed large heterogeneity ($I^2 > 50\%$). In 95 of the 321 pooled analyses (29.6%), the effect sizes of the largest study were nominally statistically significant at $p < 0.05$. The 95% prediction interval excluded the null in only 22 (6.9%) of 321 pooled analyses. Small-study effects were found for 37 pooled analyses (11.5%), and excess significance bias was identified for 62 pooled analyses (19.3%) (Table 1). For the main analyses of sleep microstructural data (CAP parameters), please see online Supplementary Table S7.

Credibility of evidence

Of the 321 pooled analyses none had convincing strength of PSG differences according to quantitative umbrella review criteria (see Fig. 3). Only seven (2.2%) were supported by highly suggestive evidence; increased SL and decreased SE in MDD, increased N1 percentage, and decreased N2 percentage, SL and REML in narcolepsy, and decreased REM sleep percentage in PD. Five (1.6%) were supported by suggestive evidence; decreased REML in MDD, decreased SE in PTSD and in narcolepsy, and decreased TST and SE in PD. There were 136 (42.4%) pooled analyses supported by weak evidence and 174 (54.2%) showing no significant changes in sleep parameters in neuropsychiatric diseases compared with HCs. The findings of subset analyses are listed in online Supplementary Table S9 and Appendix pp16.

Discussion

To our knowledge, this is the first umbrella review of alterations in PSG parameters in neuropsychiatric diseases. Our umbrella review has the particular strength of including a robust hierarchical classification of the published evidence. We reviewed 27 systematic reviews of 321 pooled analyses of studies of PSG alterations in neuropsychiatric diseases compared with HCs. Overall, available experimental evidence shows that patients with neuropsychiatric diseases show altered PSG characteristics compared with HCs, but strength of these findings varied considerably. Seven of the 147 statistically significant pooled analyses were supported by highly suggestive evidence: increased SL and decreased SE in MDD, increased N1 percentage, and decreased N2 percentage, SL and REML in narcolepsy, and decreased REM sleep percentage in PD, while five pooled analyses were supported by suggestive evidence: decreased REML in MDD, decreased SE in PTSD and in narcolepsy, and decreased TST and SE in PD.

Overall, our umbrella review shows that, although alterations in multiple PSG characteristics in various neuropsychiatric diseases have been evaluated in multiple studies, reviews and meta-analyses, the number of changes of PSG characteristics that have suggestive or stronger support is limited. In addition, no significant pooled analyses concerning PSG changes are supported by convincing evidence. Consistent with umbrella review criteria, high between-study heterogeneity, random effects p value $> 10^{-6}$, sample size of cases < 1000, prediction intervals including the null value, and small-study effects bias are common contributors that downgrade the overall confidence of published meta-analyses. Our umbrella review finds that small sample sizes in the individual studies and meta-analyses are the main factor downgrading PSG findings in neuropsychiatric diseases. This may be attributable to the relatively low incidence of some diseases (i.e. HD, WD, and SAD) in the general population, and the methodological challenges of putting patients who exhibit complex combinations of neurological symptoms (i.e. motor and cognitive impairments) and psychiatric features through the

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Table 1. Characteristics, quantitative synthesis, and bias assessment of the eligible articles

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
Chan et al., 2017, Schizophrenia									Low/high
TST min	31	487/513	−0.82 (−1.13 to −0.51)	2.73×10^{-7}	80.5	−2.46 to 0.82	0.047	No/yes	
SL min	31	486/519	1.19 (0.89 to 1.49)	1.55×10^{-14}	77.9	−0.37 to 2.75	0.146	Yes/no	
SE %	23	306/336	−1.06 (−1.35 to −0.77)	3.57×10^{-13}	62.9	−2.23 to 0.11	0.221	Yes/no	
WASO min	7	109/136	1.17 (0.68 to 1.66)	2.56×10^{-6}	64.7	−0.31 to 2.65	0.821	Yes/no	
N1%	25	387/411	0.50 (0.23 to 0.77)	0.0003	69.6	−0.70 to 1.69	0.050	Yes/no	
N2%	25	387/411	−0.02 (−0.31 to 0.27)	0.889	73.1	−1.32 to 1.28	0.550	Yes/no	
SWS%	30	488/511	−0.43 (−0.64 to −0.22)	0.00007	60.7	−1.38 to 0.52	0.828	No/no	
REM%	30	475/485	−0.18 (−0.38 to 0.02)	0.077	55.0	−1.04 to 0.68	0.846	Yes/no	
REML min	31	449/485	−0.43 (−0.66 to −0.20)	0.0002	64.0	−1.51 to 0.64	0.201	Yes/no	
REMD	17	259/265	0.32 (0.07 to 0.58)	0.012	47.7	−0.49 to 1.14	0.250	No/ no	
Ng et al., 2015, Bipolar disorder									Critically low/critically low
TST min	3	51/52	0.27 (−0.11 to 0.65)	0.164	1.1	−2.23 to 2.77	0.440	No/no	
SL min	3	51/52	0.18 (−0.19 to 0.56)	0.333	0	−2.24 to 2.61	0.835	No/no	
SE %	3	51/52	−0.12 (−0.49 to 0.26)	0.544	0	−2.54 to 2.31	0.059	No/no	
WASO min	3	51/52	0.09 (−0.29 to 0.46)	0.652	0	−2.33 to 2.50	0.345	No/no	
N1%	2	32/32	0.56 (0.08 to 1.04)	0.023	0	NA	NA	No/no	
N2%	2	32/32	−0.31 (−1.32 to 0.71)	0.551	74.0	NA	NA	No/no	
SWS%	3	46/47	−0.23 (−0.63 to 0.17)	0.257	0	−2.80 to 2.35	0.866	No/no	
REM%	3	46/47	0.58 (−0.20 to 1.36)	0.144	71.3	−8.35 to 9.51	0.432	No/no	
REML min	3	46/47	−0.12 (−0.51 to 0.27)	0.545	0	−2.66 to 2.42	0.422	No/no	
Cox & Olatunji, 2020, MDD									Critically low/low
TST min	50	1518/1197	−0.23 (−0.39 to −0.06)	0.006	73.6	−1.21 to 0.76	0.222	Yes/ no	
SL min	55	1663/1312	0.48 (0.38 to 0.58)	1.87×10^{-20}	37.8	0.01 to 0.94	0.749	Yes/yes	
SE %	45	1310/1007	−0.52 (−0.69 to −0.35)	1.47×10^{-9}	69.7	−1.46 to 0.42	0.299	Yes/no	
WASO min	9	239/222	0.26 (−0.02 to 0.54)	0.068	51.9	−0.53 to 1.06	0.00003	Yes/no	
N1%	41	1260/939	0.19 (0.07 to 0.30)	0.002	37.6	−0.28 to 0.65	0.960	No/no	
N2%	41	1260/939	−0.21 (−0.33 to −0.10)	0.003	36.3	−0.67 to 0.24	0.020	No/no	
SWS%	42	1284/963	−0.12 (−0.25 to 0.01)	0.061	49.8	−0.72 to 0.47	0.124	Yes/no	

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
REM%	42	1284/963	0.18 (0.02 to 0.34)	0.025	66.0	−0.65 to 1.01	0.068	No/yes	
REML min	50	1426/1165	−0.30 (−0.43 to −0.16)	0.00001	59.3	−1.02 to 0.43	0.160	Yes/no	
AHI (events/h)	3	77/60	0.08 (−0.26 to 0.42)	0.630	0	−2.13 to 2.29	0.798	No/no	
AI (events/h)	2	45/45	0.63 (0.21 to 1.05)	0.004	0	NA	NA	Yes/no	
REMD	36	990/787	0.36 (0.17 to 0.54)	0.0002	68.1	−0.58 to 1.29	0.627	No/yes	
Baglioni et al., 2016a, Anorexia nervosa									Low/high
TST min	3	42/34	−0.88 (−1.36 to −0.40)	0.0003	0	−3.98 to 2.22	0.398	No/no	
SL min	3	32/32	0.17 (−0.74 to 1.08)	0.711	69.2	−10.15 to 10.49	0.334	No/no	
SE %	5	44/44	−1.28 (−1.79 to −0.78)	6.93×10^{-7}	14.4	−2.37 to −0.20	0.027	Yes/no	
N1%	5	53/45	0.52 (−0.19 to 1.22)	0.149	62.7	−1.79 to 2.83	0.257	Yes/no	
N2%	5	53/45	0.10 (−0.56 to 0.75)	0.772	58.3	−1.99 to 2.18	0.982	No/no	
SWS%	5	53/45	−0.25 (−1.27 to 0.78)	0.633	82.1	−4.00 to 3.50	0.240	Yes/no	
REM%	5	53/45	−0.77 (−1.55 to 0.01)	0.054	68.0	−3.39 to 1.85	0.0004	No/yes	
REML min	6	64/56	0.41 (−0.32 to 1.14)	0.270	72.0	−1.95 to 2.77	0.042	Yes/no	
Cox & Olatunji, 2020, GAD									Critically low/low
TST min	4	72/80	−0.05 (−0.65 to 0.55)	0.870	68.2	−2.58 to 2.48	0.319	No/no	
SL min	3	43/43	0.48 (−0.68 to 1.63)	0.420	85.0	−13.61 to 14.56	0.417	No/no	
SE %	3	43/43	−0.28 (−1.41 to 0.84)	0.622	84.5	−13.98 to 13.42	0.517	Yes/no	
WASO min	2	31/31	−0.20 (−0.89 to 0.50)	0.557	47.3	NA	NA	No/no	
N1%	3	43/43	−0.05 (−0.68 to 0.59)	0.885	54.0	−6.69 to 6.60	0.642	No/no	
N2%	4	72/80	−0.15 (−0.60 to 0.31)	0.524	45.7	−1.82 to 1.53	0.671	No/no	
SWS%	4	72/80	−0.15 (−0.47 to 0.17)	0.364	0	−0.85 to 0.55	0.142	No/no	
REM%	4	72/80	0.18 (−0.31 to 0.68)	0.470	54.1	−1.75 to 2.11	0.818	No/no	
REML min	4	72/80	−0.25 (−0.70 to 0.20)	0.278	44.7	−1.90 to 1.40	0.935	No/no	
REMD	2	27/27	0.62 (−0.37 to 1.60)	0.217	67.6	NA	NA	No/no	
Cox & Olatunji, 2020, OCD									Critically low/low
TST min	4	98/104	−0.81 (−1.25 to −0.37)	0.0003	42.1	−2.39 to 0.78	0.803	Yes/no	
SL min	5	108/114	0.04 (−0.41 to 0.49)	0.861	52.4	−1.32 to 1.40	0.720	No/no	
SE %	6	130/136	−0.52 (−0.86 to −0.18)	0.003	36.7	−1.37 to 0.33	0.519	Yes/no	
N1%	5	120/126	0.22 (−0.29 to 0.73)	0.403	68.7	−1.50 to 1.93	0.476	No/no	

N2%	5	120/126	−0.39 (−0.69 to −0.08)	0.013	19.9	−1.09 to 0.32	0.866	Yes/no
SWS%	5	120/126	−0.03 (−0.41 to 0.35)	0.890	45.8	−1.14 to 1.08	0.824	No/no
REM%	5	120/126	0.09 (−0.16 to 0.34)	0.484	0	−0.32 to 0.50	0.809	No/no
REML min	6	130/136	−0.44 (−0.75 to −0.13)	0.006	27.6	−1.16 to 0.28	0.099	No/yes
REMD	4	107/113	0.37 (0.10 to 0.64)	0.007	0	−0.22 to 0.96	0.965	Yes/no
Cox & Olatunji, 2020, Panic disorder								Critically low/low
TST min	6	108/87	−0.46 (−0.92 to 0.00)	0.049	58.5	−1.84 to 0.92	0.014	No/yes
SL min	5	77/76	0.60 (0.19 to 1.00)	0.004	32.8	−0.47 to 1.67	0.886	Yes/no
SE %	4	59/53	−0.70 (−1.09 to −0.31)	0.0004	0	−1.55 to 0.15	0.177	No/yes
WASO min	3	46/46	0.37 (−0.19 to 0.93)	0.193	44.0	−5.12 to 5.86	0.010	No/no
N1%	5	84/69	0.15 (−0.18 to 0.48)	0.378	0	−0.39 to 0.68	0.651	No/no
N2%	5	77/76	0.21 (−0.22 to 0.64)	0.341	41.9	−1.01 to 1.43	0.470	No/no
SWS%	5	77/76	−0.52 (−0.84 to −0.19)	0.002	0	−1.05 to 0.01	0.205	No/no
REM%	4	61/60	−0.14 (−0.50 to 0.22)	0.455	0	−0.93 to 0.66	0.609	No/no
REML min	5	77/76	0.19 (−0.13 to 0.52)	0.239	0	−0.33 to 0.72	0.937	No/no
REMD	2	31/30	0.14 (−0.37 to 0.65)	0.592	0	NA	NA	No/no
Cox & Olatunji, 2020, SAD								Critically low/low
TST min	2	31/30	0.45 (−0.06 to 0.96)	0.081	0	NA	NA	No/no
N1%	2	31/30	0.55 (−0.82 to 1.91)	0.433	84.9	NA	NA	No/no
N2%	2	31/30	0.01 (−0.49 to 0.52)	0.957	0	NA	NA	No/no
SWS%	2	31/30	−0.21 (−0.79 to 0.38)	0.492	25.7	NA	NA	No/no
REM%	2	31/30	0.09 (−0.41 to 0.59)	0.727	0	NA	NA	No/no
REML min	2	31/30	0.30 (−0.21 to 0.81)	0.245	0	NA	NA	No/no
Winsper et al., 2017, BPD								Critically low/high
TST min	7	88/101	−0.84 (−1.35 to −0.33)	0.0012	61.9	−2.36 to 0.68	0.687	No/no
SL min	11	155/161	0.79 (0.35 to 1.24)	0.0005	71.0	−0.72 to 2.30	0.101	No/yes
SE %	9	125/126	−1.01 (−1.34 to −0.69)	1.00×10^{-9}	29.7	−1.76 to −0.27	0.077	Yes/yes
WASO min	5	69/70	0.58 (−0.01 to 1.17)	0.053	62.6	−1.34 to 2.50	0.190	No/no
N1%	11	155/161	0.17 (−0.13 to 0.48)	0.269	42.9	−0.66 to 1.00	0.494	No/no
N2%	11	155/161	−0.12 (−0.44 to 0.21)	0.481	48.7	−1.04 to 0.81	0.271	No/no
SWS%	11	155/161	−0.45 (−0.76 to −0.13)	0.006	45.6	−1.33 to 0.44	0.986	No/no
REM%	10	145/151	0.19 (−0.18 to 0.56)	0.313	57.8	−0.93 to 1.30	0.542	No/yes
REML min	12	179/175	−0.72 (−1.08 to −0.36)	0.00008	60.1	−1.87 to 0.43	0.641	Yes/no
REMD	7	107/100	0.74 (0.34 to 1.14)	0.0003	46.7	−0.34 to 1.82	0.322	Yes/no

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
Baglioni et al., 2014, Insomnia									Critically low/low
TST min	22	548/464	−0.61 (−0.83 to −0.39)	4.50×10^{-8}	60.3	−1.46 to 0.23	0.365	Yes/no	
SL min	22	539/456	0.40 (0.13 to 0.68)	0.004	74.5	−0.77 to 1.57	0.172	No/yes	
SE %	24	562/480	−0.88 (−1.10 to −0.66)	6.69×10^{-15}	60.3	−1.76 to 0.00	0.071	Yes/yes	
WASO min	14	321/237	0.71 (0.47 to 0.95)	8.22×10^{-9}	41.6	0.02 to 1.39	0.942	Yes/no	
N1%	16	355/334	0.24 (−0.05 to 0.52)	0.100	65.7	−0.78 to 1.26	0.857	No/no	
N2%	17	411/366	0.09 (−0.17 to 0.35)	0.482	64.0	−0.84 to 1.03	0.986	No/yes	
SWS%	17	411/366	−0.31 (−0.51 to −0.11)	0.002	39.4	−0.89 to 0.27	0.434	No/no	
REM%	17	377/355	−0.46 (−0.68 to −0.25)	0.00002	41.9	−1.10 to 0.17	0.832	Yes/no	
REML min	13	327/306	0.11 (−0.05 to 0.27)	0.176	0	−0.07 to 0.29	0.592	No/no	
Díaz-Román et al., 2016, Child ADHD									Critically low/moderate
TST min	7	127/163	0.16 (−0.08 to 0.40)	0.185	0	−0.15 to 0.48	0.028	No/no	
SL min	8	154/190	0.23 (0.01 to 0.45)	0.036	0	−0.04 to 0.51	0.711	No/no	
SE %	8	154/190	−0.12 (−0.47 to 0.22)	0.473	55.4	−1.10 to 0.85	0.493	Yes/no	
WASO min	3	55/44	0.26 (−0.14 to 0.66)	0.206	0	−2.35 to 2.87	0.740	No/no	
N1%	4	80/67	0.08 (−0.37 to 0.52)	0.737	44.1	−1.52 to 1.67	0.243	No/no	
N2%	4	80/67	0.03 (−0.29 to 0.36)	0.845	0	−0.68 to 0.75	0.572	No/no	
SWS%	4	80/67	−0.22 (−0.55 to 0.11)	0.194	0	−0.94 to 0.50	0.832	No/no	
REM%	4	80/67	0.17 (−0.15 to 0.50)	0.298	0	−0.55 to 0.89	0.319	No/no	
REML min	5	92/79	0.01 (−0.30 to 0.31)	0.962	0	−0.49 to 0.50	0.235	No/no	
AHI (events/h)	2	40/61	−0.35 (−0.76 to 0.06)	0.095	0	NA	NA	No/no	
PLMI (events/h)	2	40/61	−0.53 (−0.94 to −0.12)	0.011	0	NA	NA	Yes/no	
AI (events/h)	2	41/39	0.13 (−0.68 to 0.94)	0.750	66.4	NA	NA	No/no	
Lugo et al., 2020, Adult ADHD									High/high
TST min	3	74/56	0.23 (−0.13 to 0.58)	0.215	0	−2.09 to 2.54	0.801	No/no	
SL min	4	98/80	0.17 (−0.51 to 0.85)	0.629	79.3	−2.89 to 3.22	0.667	No/yes	
SE %	4	98/80	−0.05 (−0.53 to 0.43)	0.852	59.5	−1.98 to 1.89	0.609	No/no	
N1%	4	98/80	0.12 (−0.18 to 0.42)	0.429	0	−0.54 to 0.78	0.559	No/no	
N2%	4	98/80	−0.24 (−0.54 to 0.06)	0.122	0	−0.90 to 0.43	0.226	No/no	
SWS%	4	98/80	0.15 (−0.15 to 0.46)	0.316	0	−0.51 to 0.82	0.070	No/no	

REM%	4	98/80	−0.17 (−0.72 to 0.38)	0.541	68.9	−2.51 to 2.17	0.836	No/no	
REML min	3	59/62	0.11 (−0.47 to 0.68)	0.720	60.1	−6.14 to 6.35	0.061	No/no	
REMD	2	44/44	−0.59 (−1.01 to −0.16)	0.007	0	NA	NA	Yes/no	
AI (events/h)	2	35/38	0.37 (−0.33 to 1.06)	0.302	54.4	NA	NA	Yes/ no	
Chen et al., 2021, Child ASD									Critically low/low
TST min	10	202/147	−0.34 (−0.63 to −0.06)	0.018	35.3	−1.05 to 0.36	0.858	Yes/no	
SL min	10	152/143	0.47 (0.21 to 0.72)	0.0003	12.4	0.02 to 0.91	0.775	No/no	
SE %	11	220/161	−0.50 (−0.74 to −0.25)	0.00006	19.8	−1.00 to 0	0.894	No/no	
WASO min	7	168/101	0.02 (−0.43 to 0.48)	0.914	65.2	−1.36 to 1.40	0.265	No/yes	
N1%	8	110/111	0.25 (−0.02 to 0.52)	0.065	0	−0.08 to 0.58	0.697	No/no	
N2%	8	110/111	−0.09 (−0.36 to 0.17)	0.494	0	−0.43 to 0.24	0.685	No/no	
SWS%	8	110/111	−0.02 (−0.41 to 0.36)	0.905	49.9	−1.09 to 1.05	0.735	No/no	
REM%	8	110/111	−0.32 (−0.59 to −0.05)	0.020	0	−0.65 to 0.02	0.288	No/no	
REML min	7	91/94	−0.10 (−0.41 to 0.21)	0.527	9.4	−0.62 to 0.42	0.407	No/no	
AI (events/h)	2	42/32	0.11 (−0.36 to 0.57)	0.647	0	NA	NA	No/no	
Lugo et al., 2020, Adult ASD									High/high
TST min	3	51/38	−0.17 (−0.60 to 0.25)	0.427	0	−2.93 to 2.59	0.432	No/no	
SL min	4	68/49	0.52 (0.14 to 0.90)	0.007	0	−0.31 to 1.35	0.872	No/no	
SE %	3	51/38	−0.43 (−0.86 to 0.00)	0.049	0	−3.21 to 2.35	0.882	No/no	
WASO min	2	36/26	0.48 (−0.04 to 1.00)	0.068	0	NA	NA	No/no	
N1%	3	51/38	0.63 (0.19 to 1.06)	0.005	0	−2.19 to 3.45	0.498	Yes/no	
N2%	3	51/38	−0.13 (−0.65 to 0.40)	0.635	32.6	−4.89 to 4.64	0.524	No/no	
SWS%	3	51/38	−0.27 (−1.06 to 0.52)	0.504	69.8	−9.30 to 8.76	0.668	No/no	
REM%	3	51/38	0.06 (−0.36 to 0.49)	0.766	0	−2.70 to 2.83	0.462	No/no	
REML min	4	68/49	0.06 (−0.31 to 0.43)	0.750	0	−0.75 to 0.87	0.605	No/no	
Zhang et al., 2019a, PTSD									High/high
TST min	40	1111/948	−0.21 (−0.35 to −0.06)	0.006	45.8	−0.83 to 0.42	0.0003	Yes/no	
SL min	36	960/865	0.09 (−0.04 to 0.22)	0.193	22.2	−0.30 to 0.47	0.094	No/no	
SE %	38	1036/887	−0.32 (−0.50 to −0.13)	0.0009	63.1	−1.23 to 0.59	0.00004	Yes/yes	
WASO min	24	664/826	0.25 (0.10 to 0.40)	0.0011	36.9	−0.22 to 0.73	7.70×10^{-8}	Yes/no	
N1%	38	1073/924	0.15 (−0.02 to 0.32)	0.087	57.5	−0.64 to 0.93	0.003	No/yes	
N2%	39	1085/936	0.04 (−0.11 to 0.19)	0.587	46.6	−0.59 to 0.68	0.289	No/no	
SWS%	40	1099/948	−0.21 (−0.39 to −0.04)	0.016	61.4	−1.07 to 0.64	0.0001	Yes/yes	
REM%	40	1137/973	0.01 (−0.16 to 0.19)	0.894	63.7	−0.87 to 0.89	0.214	No/yes	

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
REML min	33	834/446	−0.06 (−0.21 to 0.09)	0.421	25.7	−0.53 to 0.41	0.655	No/no	
REMD	14	288/224	0.19 (−0.02 to 0.40)	0.075	26.0	−0.31 to 0.69	0.00006	No/no	
Baglioni et al., 2016b, Stroke									Low/high
TST min	11	276/1194	−0.48 (−0.66 to −0.29)	3.52×10^{-7}	13.2	−0.81 to −0.15	0.762	Yes/no	
SL min	9	251/1176	0.26 (0.02 to 0.50)	0.036	42.2	−0.36 to 0.87	0.199	Yes/no	
SE %	7	228/1148	−0.70 (−0.88 to −0.52)	1.56×10^{-14}	0	−0.93 to −0.47	0.244	Yes/no	
WASO min	3	63/40	0.80 (0.38 to 1.23)	0.0002	0	−1.96 to 3.56	0.581	Yes/no	
N1%	4	105/61	0.28 (−0.05 to 0.61)	0.098	0	−0.45 to 1.01	0.180	No/no	
N2%	4	105/61	−0.44 (−0.78 to −0.11)	0.010	0	−1.18 to 0.29	0.182	No/no	
SWS%	7	201/1154	−0.29 (−0.54 to −0.05)	0.019	38.3	−0.90 to 0.31	0.527	No/no	
REM%	7	201/1154	−0.24 (−0.48 to 0.01)	0.059	38.8	−0.85 to 0.37	0.695	Yes/no	
REML min	4	115/61	−0.08 (−0.41 to 0.24)	0.616	0	−0.80 to 0.63	0.907	No/no	
AHI (events/h)	2	32/25	0.61 (−0.20 to 1.43)	0.141	46.9	NA	NA	No/no	
D'Rozario et al., 2020, MCI									Critically low/low
TST min	9	177/189	−0.41 (−0.66 to −0.16)	0.001	23.6	−0.94 to 0.11	0.005	No/no	
SL min	6	138/147	0.44 (0.20 to 0.68)	0.0003	0	0.10 to 0.77	0.948	Yes/no	
SE %	7	168/177	−0.48 (−0.69 to −0.26)	0.00001	0	−0.76 to 0.19	0.009	No/no	
WASO min	8	172/183	0.35 (0.13 to 0.56)	0.001	0	0.08 to 0.61	0.011	No/no	
N1%	8	179/188	0.32 (0.12 to 0.53)	0.002	0	0.06 to 0.58	0.346	No/no	
N2%	8	179/188	−0.09 (−0.45 to 0.26)	0.607	62.9	−1.17 to 0.98	0.805	No/no	
SWS%	9	200/209	−0.19 (−0.52 to 0.14)	0.264	60.8	−1.18 to 0.80	0.263	No/yes	
REM%	9	204/213	−0.50 (−0.83 to −0.17)	0.003	61.5	−1.50 to 0.50	0.104	No/yes	
REML min	4	76/80	0.32 (−0.07 to 0.72)	0.107	23.9	−0.90 to 1.55	0.807	No/no	
AHI (events/h)	5	125/123	−0.06 (−0.53 to 0.40)	0.794	65.2	−1.57 to 1.45	0.751	No/yes	
PLMI (events/h)	2	44/45	0.28 (−0.14 to 0.69)	0.196	0	NA	NA	No/no	
AI (events/h)	3	88/83	0.01 (−0.30 to 0.31)	0.960	2.5	−2.06 to 2.07	0.113	No/no	
Mantua et al., 2018, TBI									Critically low/high
TST min	12	229/234	−0.24 (−0.62 to 0.14)	0.213	74.4	−1.58 to 1.10	0.274	No/no	
SL min	10	189/200	0.03 (−0.30 to 0.37)	0.852	60.5	−0.99 to 1.05	0.880	No/no	
SE %	10	160/160	−0.23 (−0.61 to 0.14)	0.223	62.6	−1.41 to 0.94	0.616	No/no	
WASO min	6	94/93	0.32 (−0.12 to 0.77)	0.155	55.1	−0.98 to 1.63	0.099	No/no	

N1%	11	202/213	0 (−0.36 to 0.35)	0.983	67.4	−1.16 to 1.15	0.879	Yes/no	
N2%	13	236/241	−0.12 (−0.37 to 0.14)	0.370	46.5	−0.87 to 0.63	0.908	No/no	
SWS%	13	236/241	0.32 (0.02 to 0.63)	0.035	61.2	−0.67 to 1.32	0.853	No/yes	
REM%	13	236/241	−0.17 (−0.42 to 0.08)	0.190	45.1	−0.90 to 0.56	0.127	No/no	
REML min	7	120/119	−0.06 (−0.43 to 0.30)	0.733	46.8	−1.04 to 0.92	0.448	No/no	
AHI (events/h)	3	61/72	0.25 (−0.53 to 1.04)	0.527	75.6	−8.81 to 9.32	0.349	No/no	
AI (events/h)	2	53/64	0.08 (−0.39 to 0.56)	0.730	38.4	NA	NA	No/no	
Zhang et al., 2020b, iRBD									High/high
TST min	29	900/584	−0.17 (−0.32 to −0.03)	0.020	36.4	−0.67 to 0.32	0.454	No/no	
SL min	31	710/589	0.20 (0.09 to 0.31)	0.0005	0	0.08 to 0.31	0.735	No/no	
SE %	37	1036/741	−0.18 (−0.33 to −0.02)	0.024	54.3	−0.89 to 0.53	0.804	No/yes	
WASO min	6	111/84	0.11 (−0.39 to 0.62)	0.657	59.5	−1.38 to 1.60	0.096	Yes/no	
N1%	35	806/676	0.07 (−0.04 to 0.18)	0.238	8.4	−0.16 to 0.30	0.680	No/no	
N2%	35	806/676	−0.27 (−0.40 to −0.13)	0.0002	35.4	−0.77 to 0.24	0.945	Yes/no	
SWS%	34	801/671	0.17 (0.02 to 0.32)	0.023	42.8	−0.41 to 0.75	0.491	No/no	
REM%	35	800/670	0.15 (0.02 to 0.28)	0.022	28.1	−0.28 to 0.58	0.546	No/no	
REML min	23	367/366	0.03 (−0.17 to 0.24)	0.735	44.2	−0.67 to 0.74	0.436	Yes/no	
AHI (events/h)	16	465/281	−0.15 (−0.38 to 0.09)	0.215	48.0	−0.87 to 0.58	0.031	Yes/no	
PLMI (events/h)	22	667/489	0.40 (0.17 to 0.63)	0.0006	66.8	−0.53 to 1.33	0.884	No/yes	
AI (events/h)	17	471/283	0.28 (0.12 to 0.44)	0.0005	0	0.11 to 0.45	0.020	Yes/no	
Plante, 2018, IH									Critically low/low
TST min	10	242/220	0.94 (0.48 to 1.41)	0.00007	80.3	−0.67 to 2.55	0.128	No/yes	
SL min	8	220/194	−0.47 (−0.82 to −0.12)	0.009	62.9	−1.53 to 0.59	0.904	Yes/no	
SE %	9	230/208	0.03 (−0.30 to 0.37)	0.850	62.1	−0.98 to 1.04	0.199	No/no	
WASO min	3	83/79	0.53 (−0.09 to 1.15)	0.091	63.0	−6.27 to 7.33	0.427	No/no	
N1%	7	145/164	0.33 (−0.05 to 0.72)	0.089	58.2	−0.78 to 1.45	0.842	No/no	
N2%	7	145/164	−0.02 (−0.26 to 0.21)	0.863	3.2	−0.36 to 0.32	0.969	No/no	
SWS%	9	232/206	−0.29 (−0.54 to −0.04)	0.021	33.0	−0.88 to 0.30	0.756	No/no	
REM%	9	232/206	0.38 (0.09 to 0.67)	0.009	48.3	−0.40 to 1.16	0.048	No/yes	
REML min	6	187/150	0.14 (−0.22 to 0.50)	0.437	55.5	−0.89 to 1.17	0.362	Yes/no	
AHI (events/h)	4	114/67	0.53 (−0.39 to 1.46)	0.255	85.2	−3.68 to 4.75	0.064	Yes/no	
PLMI (events/h)	3	104/53	−0.11 (−0.56 to 0.34)	0.630	32.1	−4.22 to 4.00	0.028	No/no	

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
Zhang et al., 2019b, HD									Moderate/moderate
TST min	7	152/144	−0.32 (−0.75 to 0.11)	0.149	69.4	−1.68 to 1.04	0.775	No/no	
SL min	7	152/144	0.32 (−0.01 to 0.66)	0.059	49.6	−0.60 to 1.25	0.104	No/no	
SE %	7	152/144	−0.88 (−1.36 to −0.41)	0.0003	72.5	−2.40 to 0.64	0.337	No/yes	
WASO min	5	127/118	0.69 (0.29 to 1.09)	0.0008	56.0	−0.57 to 1.95	0.559	No/no	
N1%	7	152/144	0.45 (0.21 to 0.68)	0.0002	0	0.14 to 0.75	0.370	No/no	
N2%	7	152/144	−0.02 (−0.31 to 0.26)	0.876	31.2	−0.68 to 0.64	0.199	No/no	
SWS%	7	152/144	−0.53 (−1.02 to −0.04)	0.035	75.4	−2.12 to 1.07	0.622	No/no	
REM%	7	152/144	−0.58 (−1.01 to −0.15)	0.008	68.0	−1.92 to 0.76	0.896	No/no	
REML min	6	122/114	0.42 (0.05 to 0.80)	0.027	48.3	−0.61 to 1.46	0.390	No/no	
AHI (events/h)	5	105/103	−0.20 (−0.48 to 0.07)	0.143	0	−0.65 to 0.24	0.112	No/no	
PLMI (events/h)	4	96/93	0.52 (−0.16 to 1.20)	0.136	80.0	−2.53 to 3.56	0.897	No/yes	
AI (events/h)	5	105/103	0.18 (−0.48 to 0.84)	0.593	80.5	−2.19 to 2.55	0.557	No/no	
Zhang et al., 2020a, PD									High/high
TST min	52	1049/1085	−0.46 (−0.59 to −0.33)	3.05×10^{-12}	48.9	−1.12 to 0.20	0.290	No/yes	
SL min	36	918/796	0.16 (−0.03 to 0.34)	0.100	68.1	−0.78 to 1.09	0.957	No/yes	
SE %	52	1308/1204	−0.58 (−0.74 to −0.41)	7.71×10^{-12}	72.0	−1.58 to 0.43	0.002	No/yes	
WASO min	20	421/354	0.51 (0.29 to 0.74)	7.81×10^{-6}	54.6	−0.31 to 1.33	0.879	Yes/no	
N1%	37	900/840	0.30 (0.18 to 0.42)	1.59×10^{-6}	31.4	−0.14 to 0.74	0.469	No/yes	
N2%	38	933/877	−0.19 (−0.35 to −0.03)	0.018	61.3	−0.98 to 0.60	0.686	No/yes	
SWS%	50	1118/1083	−0.21 (−0.37 to −0.04)	0.013	69.9	−1.19 to 0.78	0.227	No/yes	
REM%	55	1330/1252	−0.43 (−0.60 to −0.27)	3.63×10^{-7}	74.0	−1.50 to 0.63	0.416	Yes/yes	
REML min	29	714/661	0.37 (0.26 to 0.48)	7.80×10^{-11}	0.9	0.24 to 0.50	0.614	Yes/no	
REMD	4	80/93	−0.80 (−1.51 to −0.08)	0.029	77.0	−3.90 to 2.31	0.191	No/no	
AHI (events/h)	27	564/555	0.24 (0.09 to 0.39)	0.002	29.5	−0.22 to 0.70	0.823	No/yes	
PLMI (events/h)	25	674/590	0.15 (0.01 to 0.29)	0.031	23.0	−0.21 to 0.51	0.951	No/no	
AI (events/h)	22	429/444	−0.20 (−0.43 to 0.04)	0.102	62.2	−1.12 to 0.73	0.812	No/yes	
Xu et al., 2020, WD									Critically low/critically low
TST min	4	117/132	−0.77 (−1.03 to −0.52)	4.36×10^{-9}	0	−1.34 to −0.21	0.156	Yes/no	
SL min	3	95/107	0.58 (0.30 to 0.87)	0.00005	0	−1.25 to 2.41	0.891	Yes/no	
SE %	4	117/132	−1.08 (−1.35 to −0.82)	1.67×10^{-15}	0	−1.67 to −0.50	0.140	Yes/no	

WASO min	2	70/83	1.21 (0.86 to 1.56)	6.84×10^{-12}	0	NA	NA	Yes/no
N1%	4	117/132	0.41 (0.14 to 0.68)	0.003	12.9	−0.33 to 1.15	0.303	Yes/no
N2%	4	117/132	−0.63 (−0.90 to −0.37)	2.80×10^{-6}	6.0	−1.28 to 0.01	0.926	Yes/no
SWS%	4	117/132	0.21 (−0.10 to 0.52)	0.178	32.8	−0.82 to 1.24	0.404	No/no
REM%	4	117/132	0.08 (−0.33 to 0.48)	0.718	61.1	−1.58 to 1.73	0.118	No/no
REML min	3	95/107	0.53 (0.25 to 0.81)	0.0002	0	−1.30 to 2.35	0.709	Yes/no
AHI (events/h)	3	95/107	−0.18 (−0.46 to 0.10)	0.208	0	−1.98 to 1.62	0.052	No/no
PLMI (events/h)	3	95/107	0.35 (0.07 to 0.62)	0.015	0	−1.46 to 2.15	0.048	No/no
AI (events/h)	3	95/107	0.87 (0.54 to 1.21)	2.47×10^{-7}	22.9	−1.92 to 3.67	0.165	Yes/no
Zhang et al., 2021, Narcolepsy								High/high
TST min	60	1321/1166	0.09 (−0.05 to 0.23)	0.288	62.5	−0.77 to 0.95	0.001	Yes/no
SL min	49	1254/1056	−0.95 (−1.11 to −0.80)	1.73×10^{-32}	64.0	−1.83 to −0.08	0.016	Yes/no
SE %	62	1439/1236	−0.25 (−0.37 to −0.13)	0.00005	51.2	−0.92 to 0.42	0.092	No/yes
WASO min	23	461/431	0.67 (0.44 to 0.90)	8.67×10^{-9}	58.4	−0.21 to 1.55	0.377	Yes/no
N1%	55	1231/1075	1.14 (0.94 to 1.33)	2.97×10^{-29}	76.4	−0.13 to 2.40	0.0003	Yes/no
N2%	55	1201/1065	−0.81 (−0.98 to −0.64)	5.04×10^{-20}	70.5	−1.88 to 0.26	0.591	Yes/no
SWS%	61	1315/1167	−0.23 (−0.42 to −0.04)	0.018	78.8	−1.53 to 1.07	0.006	No/yes
REM%	63	1386/1208	0.13 (−0.01 to 0.25)	0.059	57.2	−0.64 to 0.89	0.596	No/yes
REML min	48	1008/902	−1.32 (−1.55 to −1.09)	3.37×10^{-30}	78.0	−2.70 to 0.06	1.62×10^{-6}	Yes/yes
AHI (events/h)	16	293/274	0.25 (0.02 to 0.49)	0.033	36.9	−0.0.40 to 0.90	0.135	No/no
PLMI (events/h)	21	641/461	1.03 (0.78 to 1.28)	4.44×10^{-16}	67.5	0.05 to 2.02	0.008	Yes/yes
AI (events/h)	11	281/226	0.17 (−0.33 to 0.66)	0.505	85.2	−1.64 to 1.97	0.583	No/yes
Zhang et al., 2021, AD								High/high
TST min	23	644/660	−0.60 (−0.86 to −0.34)	7.49×10^{-6}	78.5	−1.75 to 0.56	0.403	Yes/no
SL min	20	588/610	0.45 (0.29 to 0.61)	2.45×10^{-8}	37.0	−0.02 to 0.92	0.415	Yes/no
SE %	20	579/594	−0.96 (−1.36 to −0.57)	1.88×10^{-6}	89.1	−2.76 to 0.84	0.108	No/yes
WASO min	16	540/541	0.74 (0.38 to 1.10)	0.00007	86.6	−0.76 to 2.23	0.164	No/yes
N1%	19	504/495	0.82 (0.37 to 1.27)	0.0004	90.3	−1.22 to 2.86	0.088	No/yes
N2%	20	551/539	0.09 (−0.23 to 0.42)	0.580	84.0	−1.33 to 1.51	1.000	No/yes
SWS%	25	644/659	−0.86 (−1.14 to −0.58)	1.93×10^{-9}	80.9	−2.18 to 0.46	0.682	Yes/no
REM%	25	668/711	−0.77 (−1.14 to −0.39)	0.00006	90.1	−2.64 to 1.11	0.053	No/yes
REMD	7	129/165	−0.29 (−0.54 to −0.03)	0.03	9.7	−0.73 to 0.16	0.269	No/no
REML min	23	609/646	0.35 (0.13 to 0.58)	0.002	70.5	−0.59 to 1.30	0.490	No/yes

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
AHI (events/h)	3	42/44	0.59 (−0.20 to 1.37)	0.145	64.8	−8.13 to 9.30	0.350	No/no	
PLMI (events/h)	5	80/88	−0.16 (−0.50 to 0.18)	0.366	18.9	−0.93 to 0.62	0.001	No/no	
Bertrand et al., 2021, Seasonal affective disorder									Critically low/critically low
TST min	5	88/74	0.66 (−0.36 to 1.68)	0.207	88.0	−3.13 to 4.44	0.170	No/no	
SE %	4	81/67	0.76 (−0.21 to 1.73)	0.125	85.8	−3.65 to 5.17	0.007	No/no	
N1%	2	44/33	−0.27 (−0.89 to 0.35)	0.397	41.0	NA	NA	No/no	
N2%	2	44/33	0.26 (−0.20 to 0.72)	0.261	0	NA	NA	No/no	
SWS%	3	51/40	−0.31 (−0.95 to 0.33)	0.347	50	−6.86 to 6.25	0.895	No/no	
REM%	5	88/74	0.77 (0.06 to 1.49)	0.034	76.2	−1.72 to 3.26	0.178	Yes/no	
REML min	5	88/74	−0.73 (−1.55 to 0.08)	0.078	81.6	−3.66 to 2.19	0.041	No/yes	
Stanyer et al., 2021, Adult migraine									Low/high
TST min	7	215/971	0.10 (−0.06 to 0.26)	0.210	0	−0.11 to 0.31	0.820	No/no	
SL min	4	147/902	0.26 (0.03 to 0.49)	0.024	22.1	−0.43 to 0.95	0.849	Yes/no	
SE %	7	215/971	−0.21 (−0.54 to 0.11)	0.202	67.7	−1.18 to 0.76	0.312	No/yes	
N1%	6	182/937	0.02 (−0.25 to 0.28)	0.908	43.8	−0.67 to 0.70	0.926	No/no	
N2%	6	182/937	0.12 (−0.13 to 0.38)	0.343	41.6	−0.53 to 0.78	0.577	Yes/no	
SWS%	6	182/937	−0.26 (−0.47 to −0.05)	0.018	37.5	−0.79 to 0.27	0.573	Yes/no	
REM%	6	182/937	−0.13 (−0.34 to 0.08)	0.213	19.5	−0.57 to 0.30	0.791	No/no	
REML min	4	149/904	0.12 (−0.07 to 0.30)	0.208	0	−0.28 to 0.52	0.802	No/no	
AHI (events/h)	4	164/919	−0.09 (−0.27 to 0.09)	0.322	0	−0.48 to 0.30	0.055	No/no	
PLMI (events/h)	2	55/57	0 (−0.37 to 0.37)	0.983	0	NA	NA	No/no	
AI (events/h)	7	215/971	−0.21 (−0.49 to 0.08)	0.151	57.4	−1.00 to 0.58	0.104	No/no	
Stanyer et al., 2021, Child migraine									Low/high
TST min	4	292/303	−1.50 (−2.77 to −0.23)	0.021	97.0	−7.63 to 4.63	0.002	No/yes	
SL min	5	302/313	0.01 (−0.57 to 0.58)	0.976	88.2	−2.11 to 2.13	0.341	Yes/no	
SE %	5	302/313	−0.44 (−1.13 to 0.25)	0.212	91.8	−3.05 to 2.17	0.190	Yes/no	
N1%	5	302/313	−0.21 (−0.64 to 0.22)	0.336	78.8	−1.70 to 1.28	0.004	Yes/no	
N2%	5	302/313	0.18 (−0.45 to 0.81)	0.582	90.4	−2.18 to 2.53	0.182	Yes/no	
SWS%	5	302/313	0.33 (−0.73 to 1.39)	0.541	96.5	−3.78 to 4.44	0.078	Yes/no	
REM%	5	302/313	−0.73 (−1.30 to −0.15)	0.013	87.7	−2.84 to 1.38	0.207	No/yes	

REML min	2	50/30	0.09 (−0.37 to 0.55)	0.702	0	NA	NA	No/no	
AHI (events/h)	4	117/133	−0.16 (−0.60 to 0.29)	0.500	63.6	−1.99 to 1.68	0.489	No/no	
PLMI (events/h)	2	74/71	1.89 (0.11 to 3.67)	0.037	94.0	NA	NA	Yes/no	
AI (events/h)	3	235/210	0.82 (−0.12 to 1.77)	0.088	88.8	−10.76 to 12.41	0.547	Yes/no	
Winsor et al., 2021, Child and adolescent epilepsy									Critically low/high
TST min	7	98/130	−1.04 (−1.97 to −0.11)	0.029	88.7	−4.30 to 2.22	0.220	No/yes	
SL min	8	95/123	0.32 (0.03 to 0.60)	0.031	0	−0.04 to 0.67	0.782	No/no	
SE %	6	72/99	−1.17 (−1.75 to −0.59)	0.00007	61.7	−2.95 to 0.61	0.932	Yes/no	
N1%	8	95/123	0.60 (0 to 1.19)	0.051	74.9	−1.37 to 2.56	0.209	No/yes	
N2%	8	95/123	0.51 (0.04 to 0.99)	0.034	61.2	−0.92 to 1.94	0.318	Yes/no	
SWS%	9	135/150	−0.96 (−1.70 to −0.23)	0.010	85.9	−3.56 to 1.63	0.062	No/yes	
REM%	9	135/150	−1.22 (−2.08 to −0.36)	0.005	89.3	−4.29 to 1.85	0.047	No/no	
REML min	4	60/44	0.19 (−0.21 to 0.59)	0.348	0	−0.69 to 1.07	0.029	No/no	
PLMI (events/h)	2	37/20	0.35 (−0.20 to 0.90)	0.217	0	NA	NA	No/no	
AI (events/h)	2	37/20	0.37 (−0.58 to 1.31)	0.445	63.1	NA	NA	Yes/no	
Yeh et al., 2022b, Adult epilepsy									High/high
TST min	19	381/391	−0.23 (−0.38 to −0.09)	0.001	0	−0.39 to −0.08	0.088	No/no	
SL min	18	356/396	0.20 (0.05 to 0.36)	0.011	9.2	−0.07 to 0.48	0.503	No/yes	
SE %	20	386/426	−0.53 (−0.67 to −0.38)	5.50×10^{-13}	0	−0.68 to −0.37	0.565	No/no	
WASO min	10	198/221	0.62 (0.30 to 0.94)	0.0001	57.4	−0.34 to 1.58	0.821	Yes/no	
N1%	21	411/451	−0.03 (−0.25 to 0.19)	0.800	58.6	−0.87 to 0.81	0.587	No/no	
N2%	21	411/451	0.05 (−0.29 to 0.40)	0.754	82.2	−1.48 to 1.59	0.012	No/yes	
SWS%	21	411/451	0.27 (0.07 to 0.47)	0.007	48.9	−0.42 to 0.97	0.804	No/yes	
REM%	21	411/451	−0.56 (−0.80 to −0.33)	1.78×10^{-6}	61.5	−1.47 to 0.34	0.771	No/no	
REML min	15	294/336	0.57 (0.30 to 0.84)	0.00003	59.7	−0.35 to 1.49	0.978	Yes/no	
AHI (events/h)	8	147/145	0.16 (−0.16 to 0.48)	0.321	45.2	−0.69 to 1.01	0.860	No/no	
PLMI (events/h)	10	188/210	0.47 (0.26 to 0.67)	6.51×10^{-6}	0	0.23 to 0.71	0.016	No/yes	
AI (events/h)	8	161/185	0.40 (0.12 to 0.68)	0.005	38.3	−0.30 to 1.11	0.280	No/no	
Keenan et al., 2021, PTD									Low/high
TST min	5	90/75	−0.07 (−0.50 to 0.36)	0.738	44.4	−1.31 to 1.17	0.138	No/no	
SL min	4	73/59	0.60 (0.24 to 0.95)	0.001	0	−0.18 to 1.38	0.691	Yes/no	
SE %	5	90/75	−0.77 (−1.09 to −0.45)	2.61×10^{-6}	0	−1.29 to −0.25	0.208	No/no	
N1%	5	90/75	0.12 (−0.32 to 0.56)	0.592	47.8	−1.19 to 1.43	0.606	No/no	
N2%	5	90/75	−0.52 (−0.95 to −0.08)	0.020	44.7	−1.78 to 0.75	0.288	No/no	

(*Continued*)

Table 1. (*Continued.*)

Outcomes	Number of comparisons	Number of cases/ controls	Random effects summary estimate (95% CI)	Random effects *p* value	I^2 (%)	95% prediction interval	Egger *p* value	LS/ESB	AMSTAR 2 quality/AMSTAR 2 quality when protocol assessment and a list of excluded studies were ruled out
SWS%	5	90/75	0.17 (−0.40 to 0.74)	0.559	67.8	−1.75 to 2.08	0.358	No/no	
REM%	5	90/75	−0.02 (−0.66 to 0.63)	0.954	74.9	−2.28 to 2.24	0.426	Yes/no	
REML min	5	90/75	0.06 (−0.43 to 0.56)	0.801	58.4	−1.52 to 1.65	0.527	Yes/no	
PLMI (events/h)	2	27/23	1.04 (−0.13 to 2.21)	0.083	65.4	NA	NA	No/no	

Abbreviations of disease names: AD, Alzheimer's disease; ADHD, attention deficit hyperactivity disorder; ASD, autism spectrum disorder; BPD, borderline personality disorder; GAD, generalized anxiety disorder; HD, Huntington's disease; IH, idiopathic hypersomnia; iRBD, idiopathic rapid eye movement sleep behavior disorder; MCI, mild cognitive impairment; MDD, major depressive disorder; OCD, obsessive compulsive disorder; PD, Parkinson's disease; PTD, persistent tic disorder; PTSD, posttraumatic stress disorder, SAD, social anxiety disorder; TBI, traumatic brain injury, WD, Wilson's disease. **Other non-disease related abbreviations:** AMSTAR 2, A Measurement Tool to Assess Systematic Reviews; ESB, excess significance bias (which was used to examine whether the observed number of studies with statistically significant results (positive studies, $p < 0.05$) in each pooled analysis was larger than their expected number); LS, largest study with significant effect (which was used to reflect whether the largest study (study with smallest standard error) of a pooled analysis attend a statistically significant level). NA, applicable. **Abbreviations for sleep parameters:** AHI, apnea hypopnea index; AI, arousal index; PLMI, Periodic limb movement index; REM, rapid eye movement sleep; REMD, rapid eye movement sleep density REML, rapid eye movement sleep latency; SE, sleep efficiency; SL, sleep latency; SWS, slow wave sleep; TST, total sleep time; WASO, wake time after sleep onset.

relatively intense protocols required for PSG research. Furthermore, sleep problems in some neuropsychiatric diseases tend to go undiagnosed by physicians and underreported by patients, possibly due to a lack of insight or perceived relative unimportance of sleep disturbances compared with the motor, cognitive and psychiatric features that are recognized as key features of the diseases (Videnovic, Lazar, Barker, & Overeem, 2014). This may result in PSG examinations not being prescribed for many patients with neuropsychiatric diseases.

Nevertheless, from a clinical perspective, exploring PSG characteristics in neuropsychiatric diseases can provide valuable information and insight. Sleep comprises approximately one third of human life and is a critical state for basic brain function and neuropsychiatric health (Baglioni et al., 2016a; Harvey, Murray, Chandler, & Soehner, 2011; Regier, Kuhl, Narrow, & Kupfer, 2012). Our umbrella review revealed that increased SL and decreased SE in MDD, increased N1 percentage in narcolepsy, and decreased REM sleep percentage in PD ranked as highly suggestive evidence. These findings could be seen in other neuropsychiatric diseases (i.e. PTSD, schizophrenia, and HD), although the level of credibility of evidence varied; suggesting that single PSG parameter changes should be considered as transdiagnostic sleep characteristics across various neuropsychiatric diseases rather than disease-specific sleep features. Still lacking is robust evidence supporting that any single sleep variable alteration is specific for a single disease, as suggested by Benca, Obermeyer, Thisted, & Gillin (1992) (Benca et al., 1992) and Baglioni *et al.* (Baglioni et al., 2016a). By comparison, looking across PSG variables reveals that no two diseases have the same sleep profile (Fig. 2). This suggests that specific profiles of sleep alterations may best define distinct disorders rather than alterations in a single sleep variable.

A great amount of research has been conducted on genes, proteins, and neural circuits to try to find biomarkers which could identify or predict neuropsychiatric diseases; however, to date, no specific marker has been found which confidently identifies or distinguishes different neuropsychiatric diseases. In addition, psychomotor activity, mood, cognition, suicidal ideation, psychotic symptoms, and neurological symptoms, have been traditionally considered basic dimensions in neuropsychiatric diseases (Cuthbert & Kozak, 2013; Morris, Rumsey, & Cuthbert, 2014; Sanislow et al., 2010). Our results suggest that the overall change patterns of PSG parameters should be comprehensively evaluated as an important basic dimension and potential disease-specific biomarker for neuropsychiatric diseases (Lim et al., 2020). However, the umbrella review method we employed did not allow a statistical analysis that would test the ability of specific sleep profiles to identify or distinguish different neuropsychiatric conditions. This hypothesis could be potentially tested using machine learning methodology in a large sample study consisting of various neuropsychiatric diseases.

Existing evidence shows that successful treatment of sleep disturbances has a positive impact on the course of neuropsychiatric diseases (Gee et al., 2018; Krystal, 2020). Traditionally, pharmacologic (i.e. hypnotics) and psychosocial interventions (i.e. cognitive behavioral therapy for insomnia) are the main options for treating sleep disturbances in various neuropsychiatric diseases (Qaseem, Kansagara, Forciea, Cooke, & Denberg, 2016; van der Zweerde, Bisdounis, Kyle, Lancee, & van Straten, 2019). It has been suggested that these therapies may improve some altered PSG determined variables, such as TST and SE, which are seen in different neuropsychiatric diseases (Monti, Torterolo, & Pandi Perumal,

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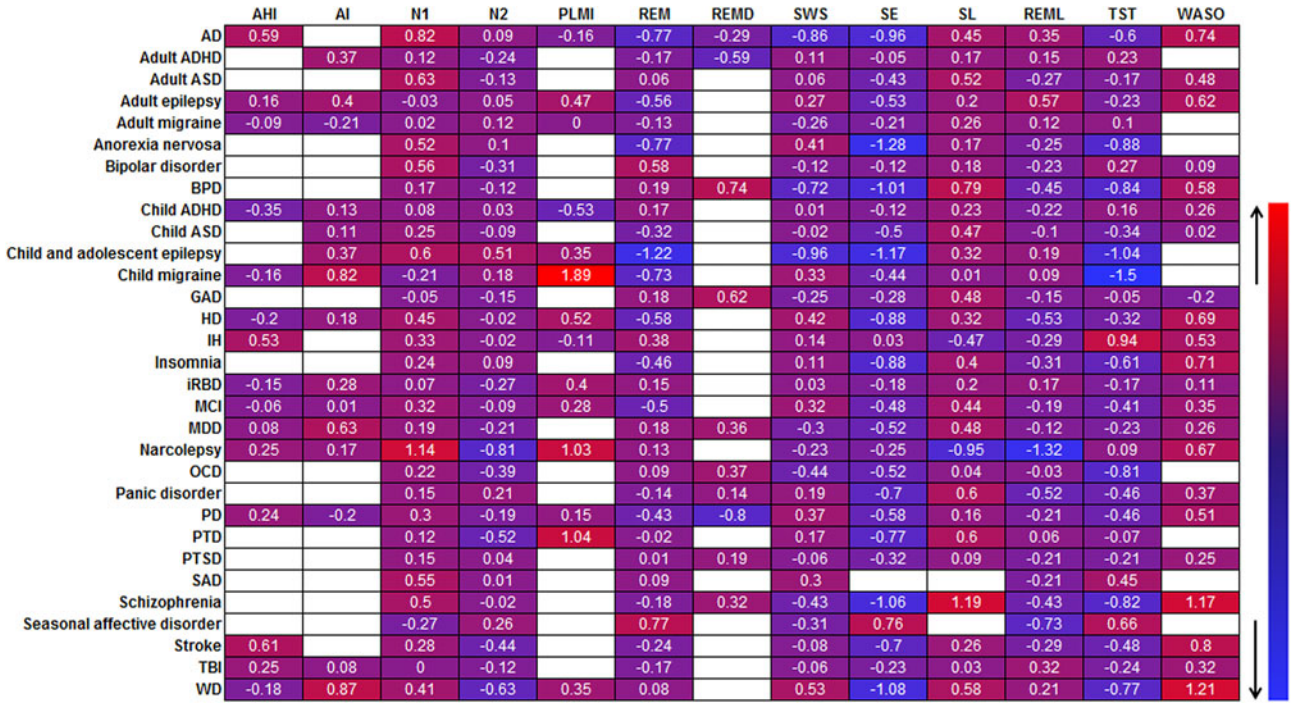

Fig. 2. Change patterns (standardized mean differences) of sleep parameters in 27 neuropsychiatric diseases. **Abbreviations of disease names:** AD, Alzheimer's disease; ADHD, attention deficit hyperactivity disorder; ASD, autism spectrum disorder; BPD, borderline personality disorder; GAD, generalized anxiety disorder; HD, Huntington's disease; IH, idiopathic hypersomnia; iRBD, idiopathic rapid eye movement sleep behavior disorder; MCI, mild cognitive impairment; MDD, major depressive disorder; OCD, obsessive compulsive disorder; PD, Parkinson's disease; PTD, persistent tic disorder; PTSD, posttraumatic stress disorder, SAD, social anxiety disorder; TBI, traumatic brain injury, WD, Wilson's disease. **Abbreviations for sleep parameters:** AHI, apnea hypopnea index; AI, arousal index; PLMI, Periodic limb movement index; REM, rapid eye movement sleep; REMD, rapid eye movement sleep density REML, rapid eye movement sleep latency; SE, sleep efficiency; SL, sleep latency; SWS, slow wave sleep; TST, total sleep time; WASO, wake time after sleep onset.

2017; Talbot et al., 2014). Thus, from the prospective of improvements of PSG variables, traditional pharmacologic and psychosocial interventions have the properties of transdiagnostic treatments (one treatment that could improve sleep disturbance across patients with different diagnosis). Harvey and colleagues have proposed that the use of transdiagnostic treatment protocols could decrease the burden on clinicians, who currently must learn multiple specific treatment protocols that often share many common theoretical underpinnings and components (Harvey et al., 2011). On the other hand, given the different PSG patterns across different neuropsychiatric diseases seen in our umbrella review, it would appear that 'one size fits all approach' treatment protocols may be insufficient to improve sleep in all neuropsychiatric diseases. Rather more targeted treatment approaches should emphasize disease-specific altered sleep patterns in developing new sleep intervention protocols across different neuropsychiatric diseases. This idea was also proposed by Harvey and colleges (Harvey, 2009; Harvey et al., 2011) who suggested that new sleep intervention protocols should include core treatment modules that would be delivered regardless of diagnosis, in addition to optional modules to cover treatment of disorder-specific symptoms.

Clinically, serious psychiatric and neurological symptoms and sleep disturbances in some neuropsychiatric patients do not allow the withdrawal of treatment. When performing PSG examinations, some medications (i.e. anti-depressants and hypnotics) may affect sleep measures. Additionally, one neuropsychiatric disease may co-occur with other neuropsychiatric diseases (i.e. depression in PTSD, schizophrenia, and PD) which may interact and produce either over- or under-estimations of PSG changes in patients with neuropsychiatric diseases. Thus, we limited analyses to studies excluding patients with comorbidities, and studies excluding patients taking antidepressant and hypnotics, which revealed that only decreased SE and increased SL in MDD remained as highly suggestive evidence. Majority of other comparisons in the subset analyses were downgraded to weak evidence or changed to no significant PSG differences between cases and HCs. In fact, stratifying the analysis by the aforementioned factors inevitably decreased the power of the analysis. As shown in Table 1 and online Supplementary Table S9 except for MDD, the sample size in other subset analyses were largely decreased compared with the whole sample analysis, which may be the main factor that decreased the power of the analysis. Nevertheless, it should be noted that majority of the patients in the whole sample analysis were drug-naïve or had a washout period before PSG examination and that most of the component studies had excluded patients with other comorbid neuropsychiatric diseases, minimizing or alleviating these potential confounds to accurate PSG measurement.

This umbrella review has some limitations. First, some meta-analyses were excluded from predictive intervals and excess significance tests because they did not provide adequate data necessary to conduct the respective analyses. Second, we did not assess the quality of component studies of each of the meta-analyses as it was beyond the scope of our umbrella review. Third, biases that might have been caused by the respective method characteristics of individual component studies, such as sex, age, race/

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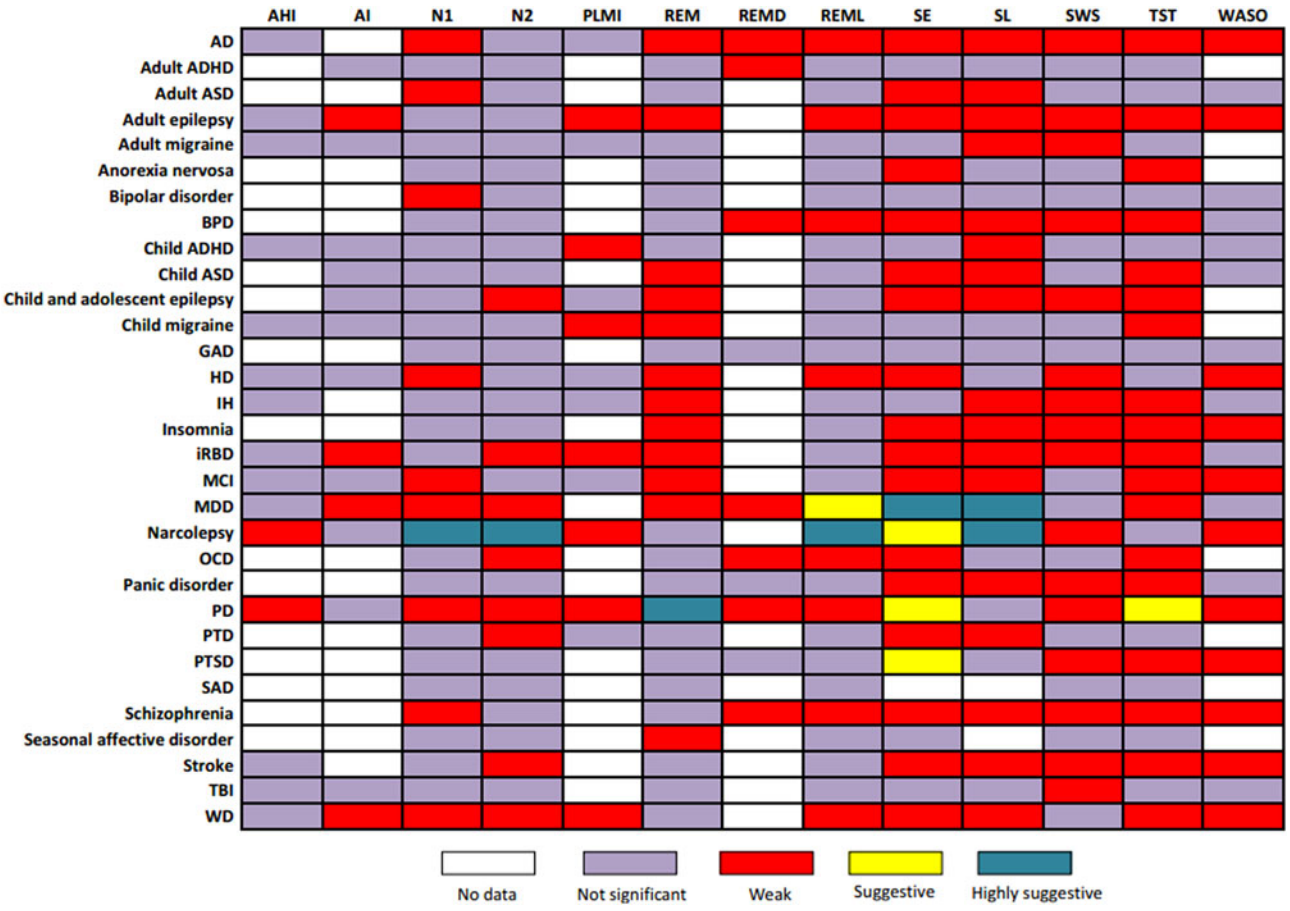

Fig. 3. Credibility of polysomnographic alterations in 27 neuropsychiatric diseases. **Abbreviations of disease names:** AD, Alzheimer's disease; ADHD, attention deficit hyperactivity disorder; ASD, autism spectrum disorder; BPD, borderline personality disorder; GAD, generalized anxiety disorder; HD, Huntington's disease; IH, idiopathic hypersomnia; iRBD, idiopathic rapid eye movement sleep behavior disorder; MCI, mild cognitive impairment; MDD, major depressive disorder; OCD, obsessive compulsive disorder; PD, Parkinson's disease; PTD, persistent tic disorder; PTSD, posttraumatic stress disorder, SAD, social anxiety disorder; TBI, traumatic brain injury, WD, Wilson's disease. **Abbreviations for sleep parameters:** AHI, apnea hypopnea index; AI, arousal index; PLMI, Periodic limb movement index; REM, rapid eye movement sleep; REMD, rapid eye movement sleep density REML, rapid eye movement sleep latency; SE, sleep efficiency; SL, sleep latency; SWS, slow wave sleep; TST, total sleep time; WASO, wake time after sleep onset.

ethnicity, socioeconomic status effects, and genetic causes of diseases, were not fully assessed in our umbrella review, due to insufficient information (i.e. not performing analyses stratified by sex or other factors) reported in the majority of the component studies. Fourth, our umbrella review did not include all neuropsychiatric diseases. For instance, PSG changes in multiple sclerosis and multiple system atrophy are lacking because meta-analyses for these topics were not found in our literature search. Thus, the evidence map of PSG characteristics is still incomplete. Fifth, in our study selection process, we encountered more than one meta-analysis on the same topic that included some, but not all, of the same studies. In these instances, we included the most up-to-date meta-analysis that contained the most studies. It also should be noted that, in addition to newly identified original case–control studies, different meta-analyses on the same topic may use different eligibility criteria and different search terms that results in differences in included studies. This means that not all relevant data across meta-analytic studies were considered. However, we cannot offer a way to address this concern, though it has been previously noted (Correll et al., 2021; Dragioti et al., 2019; Kim et al., 2020) and may be resolved in future as the methodology for umbrella reviews continues to evolve.

Despite these limitations, this umbrella review mapped PSG characteristics across 27 neuropsychiatric diseases. Out of 321 identified PSG comparisons, evidence from the pooled analyses was highly suggestive for increased SL and decreased SE in MDD, increased N1 percentage, and decreased N2 percentage, SL and REML in narcolepsy, and decreased REM sleep percentage in PD. Evidence from the pooled analyses was suggestive for decreased REML in MDD, decreased SE in PTSD and in narcolepsy, and decreased TST and SE in PD. We cannot state that other PSG comparisons supported by weak evidence are not meaningful, but they have uncertainties that need to be resolved. Although the credibility of evidence of PSG characteristics in the 27 neuropsychiatric diseases varied across different PSG variables and different diseases, the current findings provide a starting point that may guide advances in sleep research and improve the understanding of sleep features in neuropsychiatric diseases. Critically, no two diseases had the same altered sleep patterns, suggesting that specific sleep profiles may be an important dimension for marking distinct disorders. Further well-designed studies with large sample sizes and accurate assessment of potential biases are needed to confirm and expand these findings.

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Conflict of interest. We declare no competing interests.

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