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RESEARCH ARTICLE

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Interventions to improve the sleep quality of adults with personality disorder: A systematic review

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Abstract

Poor quality sleep is common for people who have a diagnosis of personality disorder (PD). Core cognitive and behavioral features of PD may cause and perpetuate poor sleep, but to date, no review has collated the evidence on the efficacy of interventions to improve sleep quality for people with PD. Structured searches for interventional studies among adults with PD and reporting validated measures of sleep quality were conducted up to November 2022 in multiple databases. Single-case reports were excluded. Study quality was assessed with standardized risk of bias tools. Unreported data was sought systematically from authors. This review was pre-registered with an international prospective register of systematic reviews (PROSPERO) (CRD42021282105). Of the 3503 identified studies, nine met inclusion criteria, representing a range of psychological, pharmaceutical, and other interventions and outcome measures. Meta-analytic methods were not feasible because of the serious risk of bias in all studies, and results were therefore synthesized narratively. There is limited and low-quality evidence of the effects of a variety of interventions to improve the sleep quality of people living with PD. Further research might consider specifically including people diagnosed with PD in trials of sleep interventions and using sleep outcome measures in trials of established PD treatments.

INTRODUCTION

People diagnosed with personality disorder (PD) commonly describe poor quality sleep, with evidence pointing to a prevalence of insomnia of around 70% (Selby, 2013; Van Veen et al., 2017; Vanek et al., 2021). Because people with PD (also frequently referred to as complex emotional needs [CEN]) make up 30%–50% of all patients in secondary mental health services, sleep disturbance in PD is a common presentation in the psychiatric clinic (Newton-Howes et al., 2010). Furthermore, people given a diagnosis of PD are overrepresented in sleep-disorder clinics (Somma et al., 2018), particularly with parasomnias and sleep–wake phase disorders (Dagan et al., 1996). Recent evidence syntheses have identified worse aspects of sleep quality in people with PD compared with people without PD, which include greater pre-sleep hyperarousal, a longer sleep-onset latency (SOL), a greater number of post-sleep-onset wakings, and poorer sleep efficiency (Winsper

et al., 2017). Moreover, sleep disturbance in this group of people appears independent of cooccurring PTSD (Schredl et al., 2012), depressive symptoms, or substance misuse (Harty et al., 2010).

The cognitive and behavioral factors specific to PD are directly associated with subjective and objective sleep disturbances (Simor & Horváth, 2013). For example, emotional lability, an inconsistent sense of self, difficulties with reality testing, increased nightmare frequency, specific circadian profiles (including phase delay and rest-activity misalignment), and dissociative experiences, among others, are likely to mean that treating sleep disturbance in the context of PD requires a different approach to individuals with disturbed sleep but without personality difficulty (Jenkins et al., 2022; McGowan & Saunders, 2021; Rezaie et al., 2020). Moreover, "sleep-state misperception," a significant discrepancy between subjective sleep quality and objective quality, is thought to be common for people with PD (Bastien et al., 2008; Philipsen et al., 2005). In this sense, approaches to improving the sleep quality of patients with other psychiatric diagnoses cannot be assumed to translate to someone diagnosed with PD.

There are established bi-directional relationships between poor quality of sleep and core aspects of PD (Simor & Horváth, 2013), particularly emotion dysregulation (Vanek et al., 2020), negative affectivity (Kaurin et al., 2022), and many of the features of borderline PD, specifically impulsivity (McGowan et al., 2020; Van Veen et al., 2017), aggression (Dautovich et al., 2021; Krizan & Herlache, 2016), irritability, and self-harming behaviors (Blasco-Fontecilla et al., 2011; DeShong & Tucker, 2019; Kaurin et al., 2022; Ko et al., 2021; Scamaldo et al., 2022) and suicidality (Simmons et al., 2021; Trockel et al., 2015). Moreover, poor quality sleep is also associated with symptoms of dissociation, cognitive symptoms including inattention, lower distress tolerance (Nomamiukor et al., 2018), cooccurring anxiety and depressive disorders, psychotic experiences (Kasanova et al., 2020; Simor et al., 2019), and alcohol and illicit drug use (Chen et al., 2021; Maroti et al., 2011). Qualitative evidence has also demonstrated that poor sleep impacts quality of life (Ma et al., 2019), daily functioning (Selby, 2013), and relationships in people with PD (Wood et al., 2015). Furthermore, the presence of sleep disturbance (Plante et al., 2013a) and dysfunctional cognitions about sleep appear to be associated with poor improvement over time in the symptoms of borderline PD (Plante et al., 2013b). Unsurprisingly, poor sleep is therefore a common contributor to the decision to prescribe psychotropic medication for people with CEN (Martinho et al., 2014; Paton et al., 2015; Plante et al., 2009).

Addressing poor quality of sleep is advised by the National Institute of Health and Clinical Excellence (NICE) guidance on the treatment of borderline PD, which is constituted by sleep hygiene and short-term hypnotic medication use (National Institute for Health and Care Excellence, 2018). Previous review articles of specific interventions in psychiatric and general populations, including the NICE first-line insomnia treatment "cognitive behavioural therapy for insomnia" (CBTi), have not identified studies applied to people with PDs (Jansson-Fröjmark & Norell-Clarke, 2016; Wu et al., 2015). Given the frequency and impact of poor sleep and the anticipation that improving sleep may offer an avenue of relief for some of the distressing and dysfunctional experiences had by people with a diagnosis of PD (Winsper et al., 2017), a review of interventions was deemed clinically important. This systematic evidence synthesis looks to review the literature on the efficacy of interventions of all and any nature, including pharmacological, psychological, behavioral, and social, among others, on sleep outcomes in PD.

METHODS

We formulated the following research question: Which interventions improve the quality of sleep of people diagnosed with PD? Simple searches of PubMed, Google Scholar, PROSPERO, and the Cochrane Library did not reveal previous or in-progress systematic reviews with a similar aim. This review was conducted and reported following the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines (Moher et al., 2009), and the study protocol was pre-registered with PROSPERO, an international prospective register of systematic reviews, with identification number CRD42021282105.

Patient and public involvement and engagement

The clinical importance and rationale for this study were discussed with lived-experience clinicians from Central and North West London NHS Foundation Trust's "Complex Emotional Needs Pathway," who also offered suggestions for the discussion of this article.

Selection criteria

Studies were included if they described original data on the effects of an intervention on individuals aged 18 and

over with an established diagnosis of PD made by a structured clinical assessment or trained clinician, including all PD subtypes, trait domain qualifiers, and clusters by DSM or ICD criteria. There were no eligibility limitations based on psychiatric comorbidity or concurrent medication use. Case studies were excluded, but there were no other limitations placed on the interventional study design. While the technical difference between comparative and non-comparative studies is important to note, observational cohort studies that prospectively followed the effect of an unallocated intervention were also included. Studies must have reported either "hard" outcomes, including objective measures of sleep quality like polysomnography (PSG), actigraphy, sleep electroencephalography (EEG), and routine data outcomes like insomnia diagnoses or hypnotic medication prescriptions, or "soft" subjective measures of sleep quality validated in adults. During preliminary searches, it became clear that a number of reports measured sleep outcomes using subscales or highly abbreviated sleep quality outcomes, sometimes limited to single sleep items taken from other psychopathology, often depression, rating scales. We decided that limiting this review to studies with sleep measures validated to assess sleep quality was a pertinent approach to avoid a large number of studies with critical measurement bias. Studies were limited to the English language, and there was no limitation on publication dates.

Search strategy

Two authors independently conducted an advanced search strategy that was developed to search EMBASE, MEDLINE and PsychINFO via Ovid, Web of Science, CINAHL, and the Cochrane Collaboration databases for the three facets of the research question: "personality disorder," "sleep outcomes," and "interventions." Search terms constituting each facet were developed in conjunction with the Yale MeSH analyzer (https://mesh.med.yale.edu/). Grey literature was explored by screening the first 500 results from a simple Boolean search in Google Scholar using the terms "Sleep AND 'Personality Disorder'," and pre-prints from July 2021 to November 2022 were searched in medRxiv with the same search term. One author undertook hand searches of key journals in the field: *Sleep*, *Sleep Research*, *Sleep Medicine*, *Journal of Personality Disorders*, and *Personality and Mental Health*, from 2010 to September 2022. Furthermore, we made a specific effort to find reports of people with a PD diagnosis treated with CBTi from articles highlighted in systematic reviews of patients with psychiatric disorders treated with CBTi, which were identified by searches in PubMed and Google Scholar of "CBTi AND mental AND systematic." The citations of included articles were reviewed by backwards snowballing. Searches were re-run before final analysis on November 20, 2022. A full search strategy is reported in the review protocol.

Unpublished and missing data

Unless data was already publicly available, the authors of articles that were suspected of harboring unpublished data were contacted by e-mail by one author on up to three occasions at fortnightly intervals from May 2022. Studies were suspected to harbor unpublished data if they made reference to including participants with PD, assessed for Axis II diagnoses, and collected sleep outcome measures, but that subgroup analyses relating to the PD population were not reported.

Quality assessment and data extraction

Search results from each database were downloaded into the desktop version of EndNote and re-uploaded to the Covidence platform (https://www.covidence.org/). Two reviewers independently undertook abstract screening and full text review. Disagreements in whether a screened abstract progressed to the next stage were resolved by two screening authors' consensus, and disagreements on the outcome of full-text reviews were rationalized by full working-group consensus. Two authors independently assessed the quality and risk of bias at the individual study level, with the Cochrane Collaboration's Revised Risk of Bias tool 2 (RoB2) for randomized studies, "Risk of Bias in Non-Randomised Studies of Interventions" (RoBINS-I) for non-randomized studies (Sterne et al., 2016; Thomson et al., 2018), and other study types with the appropriate National Institute of Health (NIH) Quality Assessment Tool (Tran et al., 2021). The disagreement was resolved by all-author consensus. We planned to use the "Grading of Recommendations Assessment, Development and Evaluation" (GRADE) tool to demonstrate confidence in the evidence reported.

We extracted the following information from qualifying studies into a custom data extraction spreadsheet: basic article identifiers, the number of participants with PD and the number and nature of any controls, the diagnostic method of PD, co-occurring psychiatric diagnoses, a summary of the intervention, sleep outcome measure and effect sizes, secondary outcomes, and timing of assessment outcomes.

RESULTS

In total, 3501 articles were identified through the search strategy, and 318 duplicates were removed. Two additional studies were identified by hand search, and no additional studies were identified by other means. After 3185 underwent title and abstract screening, 97 were reviewed in full. Ten studies were identified as harboring relevant unreported data, of which three had no contact information or contact attempts bounced. One study reported a single applicable case and was therefore not contacted. Three authors did not return contact after successive attempts. Three corresponding authors returned a reply in which one was unable to provide data because of restrictions placed on the dataset, one provided additional clarification that excluded the study on the grounds of an inappropriate outcome measure, and one study that provided original data on two eligible participants clarified that their data was imputed and missing original scores, meaning it could not be used in synthesis.

Nine remaining studies (Bromundt et al., 2013; De la Fuente et al., 2002; Ellis et al., 2019; Petrov et al., 2019; Roepke et al., 2017; Schennach et al., 2019; Slotema et al., 2019; Steingrímsson et al., 2022; Weinhold et al., 2017) met criteria and were included in the evidence synthesis (Figure 1).

Study characteristics

Nine included studies were published between 2002 and 2022 and were undertaken in seven countries: three in Germany, two in the United States, and one in each of Belgium, the Netherlands, Switzerland, and Sweden. One randomized controlled trial was identified. Two other studies compared their intervention with a control group given treatment as usual or no treatment, and the remaining six studies were observational cohorts, or case series, and did not have comparator groups.

There was significant clinical heterogeneity; no two interventions were comparable. Four studies reported on psychological interventions: narrative exposure therapy, eye movement desensitization and reprocessing (EMDR), image rehearsal therapy for nightmares, and behavioral therapy for insomnia. Two pharmaceutical agents were studied: doxazosin (an alpha-1 adrenergic antagonist) and carbamazepine (an anticonvulsant). Other studies examined the effects on sleep outcomes of morning light

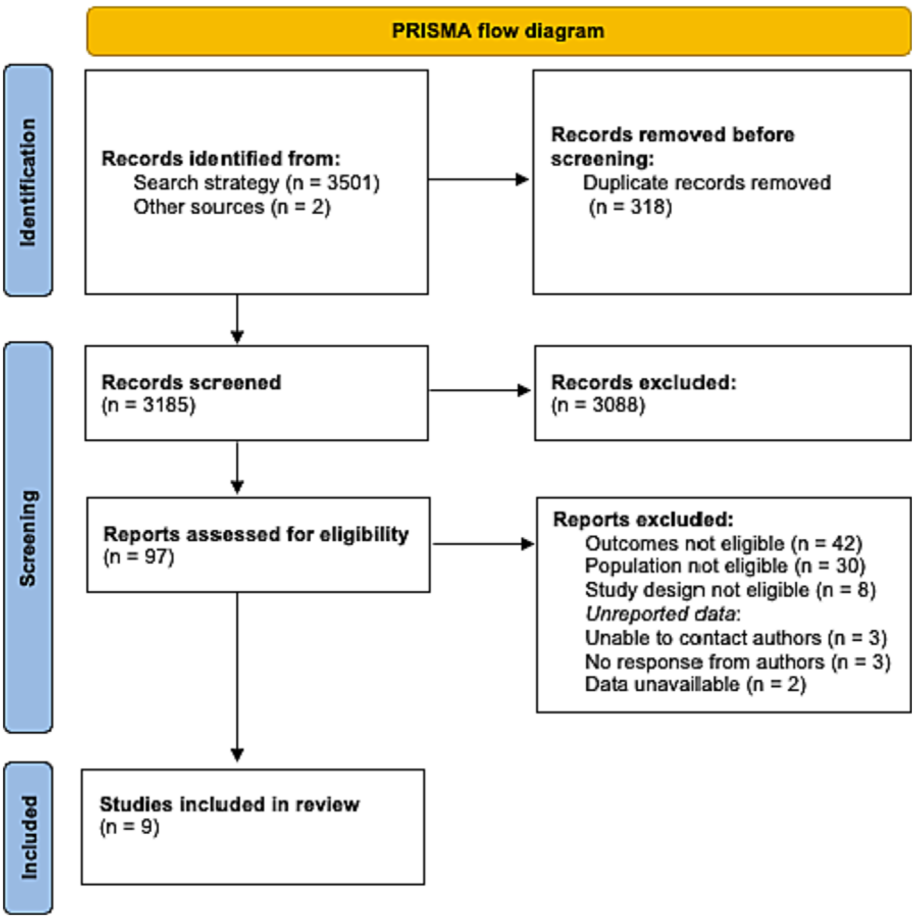

FIGURE 1 Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram.

therapy, weighted blankets, and another examined an inpatient psychiatric admission that included psychotherapy not specific to improving sleep quality, and hypnotic or sedative medication use where clinically indicated and which were "applied temporarily, but tapered off and stopped as soon as the psychopathological condition had improved" (Schennach et al., 2019). The interventions described in six studies targeted sleep specifically, and in the other three treatments did not target sleep specifically but measured sleep outcomes. Sample sizes were small, varying between three and 268, median $n = 24$, and the total number of reported PD patients across studies was 515. "Borderline Personality Disorder" was the most common diagnosis among the included participants, followed by "Personality Disorder," and fewer than 10 patients with obsessive–compulsive PD (OCPD), avoidant PD (AvPD), or "mixed Personality Disorder" were represented. One study included female participants only (Bromundt et al., 2013); otherwise, studies did not restrict recruitment to one gender. Two studies reported on patients with co-occurring PTSD (Slotema et al., 2019; Weinhold et al., 2017). No studies were identified using other terminology, like CEN. Two studies considered patients with both PD and PTSD (Slotema et al., 2019; Weinhold et al., 2017); one study reported all psychiatric comorbidities of its 3 participants (Ellis et al., 2019); one study highlighted if participants specifically had a history of disordered eating (10/16 participants) or attention deficit disorder (5/16 participants) (Bromundt et al., 2013); and within other studies, comorbidity of PD with other psychiatric disorders was not reported or participants with comorbidity (usually psychosis) were actively excluded.

Four studies reported on subjective outcomes only; three reported objective outcomes only; and two reported both objective and subjective outcomes for their participants. Three subjective self-report sleep quality scales were used by the included studies: two studies used the Pittsburgh Sleep Quality Index (PSQI), three used the Insomnia Severity Index (ISI), and one used the PSQI Addendum (PSQI-A) for PTSD. Two studies reported PSG data, and one study reported each of the other objective sleep quality outcome measures, actigraphy, and sleep electroencephalography (EEG). One study also reported summarized sleep diary data, and another reported population-level prescriptions of sedative medication.

Quality and risk of bias

Table 1 summarizes the included studies, along with risk of bias assessments. One randomized trial was assessed as having a high risk of bias by the RoB2, and of two non-randomized comparative studies assessed with the ROBINS-I tool, neither was deemed to have a low risk of bias. Likewise, of six open label studies assessed with the "NIH Quality Assessment Tool for Pre-Post studies without control groups," all but one presented a "Fair quality," representing an intermediate risk of bias—the remaining study returned a poor quality.

Summary of findings

In light of significant methodological differences, a small number of studies describing unique interventions and using different incomparable outcome measures, and a high risk of bias in all studies, use of the GRADE tool was not possible, and we opted to synthesize findings through a narrative rather than meta-analytic approach.

Psychological

There was mixed evidence of an effect on sleep quality from studies investigating psychological interventions. Weinhold et al. (2017) provided 4 weeks of once or twice weekly Narrative Exposure Therapy to 13 participants with BPD and co-occurring PTSD, demonstrating significant reductions in PSG measured SOL from a mean of 30 to 24 min compared with an observed increase in SOL in eight treatment-as-usual controls at 4 weeks. Controls were not followed to the 6-month follow-up point, and the active arm had lost any benefit to sleep quality from the intervention, aside from maintaining benefits to a reduced number of sleep arousals on PSG, with SOL at 6 months more than double baseline values and PSQI scores being similar. In another study of 47 patients with cooccurring PD and PTSD, treatment with EMDR in addition to treatment as usual demonstrated significant decreases from 17.6 to 10.7 (from moderately severe to subthreshold insomnia) on the 28-point self-reported Insomnia Severity Scale, accompanied by improvements in PTSD symptomatology and dissociative experiences (Slotema et al., 2019). The length of EMDR treatment varied considerably in this study, from two to 15 sessions.

Petrov et al. (2019) delivered behavioral therapy for insomnia and gradual hypnotic medication withdrawal over eight individual sessions in as many weeks to eight patients with OCPD and 15 patients without OCPD, both groups having had regular hypnotic use for more than 6 months. Perceived improvements in quality of sleep as measured by the self-reported ISI and a general sleep quality measure were reported by participants with and without OCPD post-treatment and at 1 year, despite a

TABLE 1 Summary of included studies.

Author and date	Participants	Intervention	Population size	Outcome	Results	Risk of bias assessment
Randomized control trials						*RoB-2*
De la Fuente et al., 2002	BPD	Carbamazepine	10 active arm 10 control arm	Sleep EEG pre and post treatment	No significantly differing improvements in sleep architecture compared with controls.	High risk of bias
Non-randomized comparative trials						*ROBINS-I*
Bromundt et al., 2013	Females with BPD	Morning light therapy	14 active arm 10 control arm*	Actigraphy pre and post treatment.	Phase advance of rest-activity cycle. Earlier waking and less total sleep time post intervention. No effect on onset latency or efficiency.	Critical risk of bias
Weinhold et al., 2017	BPD and PTSD	Narrative exposure therapy	13 active arm 8 control arm	PSQI and PSG, pre, post, and at 6 months.	Reductions in sleep-onset latency post-treatment only compared with TAU. Improvements not sustained at 6-month follow-up.	Serious risk of bias
Open-label/case series studies						*NIH Quality Assessment Tool for pre-post studies*
Ellis et al., 2019	OCPD and/or AvPD or BPD	Image rehearsal therapy for nightmares	3	ISI pre and post treatment (1, 2)	Reductions in ISI from 10 to 6; 22 to 9; and 19 to 13.	Poor
Petrov et al., 2019	OCPD	Behavioral therapy for insomnia	8	ISI, diaries and PSG, pre, post, and at 1 year	Subjective improvements post-treatment maintained alongside reduced hypnotic use at 1 year follow-up.	Fair
Roepke et al., 2017	BPD	Doxazosin	33	PSQI-A pre and post treatment	A significant improvement in PSQI-A at Week 12 was similar to those who had discontinued treatment.	Fair
Schennach et al., 2019	PD	Treatment as usual in psychiatric hospital	101	PSQI on admission and discharge	PD patients reported clinically important albeit statistically non-significant PSQI improvements.	Fair

TABLE 1 (Continued)

Author and date	Participants	Intervention	Population size	Outcome	Results	Risk of bias assessment
Slotema et al., 2019	PD and PTSD	EMDR	47	ISI pre and post treatment	Significant group mean ISI reductions from 17.6 to 10.7 after varying lengths of treatment.	Fair
Steingrímsson et al., 2022	PD	Weighted blankets	268	Sedative medicine use at 1 year	Sleep medication use did not change significantly after 1 year having collected a weighted blanket.	Fair

Abbreviations: AvPD, avoidant personality disorder; BPD, borderline personality disorder; EEG, electroencephalography; EMDR, eye movement desensitization and reprocessing therapy; ISI, Insomnia Severity Index; OCPD, obsessive–compulsive personality disorder; PD, personality disorder; PSG, polysomnography; PSQI, Pittsburgh Sleep Quality Index; PSQI-A, Pittsburgh Sleep Quality Index PTSD Addendum; TAU, treatment as usual.
*Ten participants without diagnoses of PD, and not administered the intervention were used as control arm.

substantial reduction in the abstinence of hypnotic medication in all but four individuals at 1-year follow-up. Self-completed sleep diaries and PSG, however, did not identify significant changes in any sleep–wake measure in either group post-treatment, and at 1 year, there were significant deteriorations in total sleep time (TST), sleep efficiency (SE), and time spent awake after sleep onset (WASO) among the OCPD compared with the no-OCPD group. Authors speculate that these deteriorations in the OCPD group at 1 year might represent greater reactivity to the PSG sleep lab environment, as demonstrated by a return to a baseline higher score on pre-sleep state and trait anxiety inventories in the OCPD group but not the no-OCPD group, as well as the increasing uncertainty introduced by even smaller group sizes after a 25% and 33% drop-out rate (OCPD $n = 6$; no-OCPD $n = 10$ at the 1-year time point). Finally, a small open label review of four 1-h small group sessions of imagery rehearsal therapy for nightmares, reported that three patients with OCPD and AvPD, OCPD, and AvPD and BPD, respectively, each with multiple other psychiatric comorbidities, reported improvements pre-post intervention in nightmare severity, suicidal ideation, and subjective sleep quality measured by the ISI from 10 to 6, 19 to 13, and 22 to 9 (Ellis et al., 2019). There may have been other patients among the 20 total treated with PD in this study who remained unreported.

Pharmacological

In two studies of pharmaceutical interventions, there was limited evidence of benefit to sleep outcomes. De la Fuente et al. (2002) demonstrated that 400–800 mg of carbamazepine over 32 days increased the proportion of slow-wave sleep (SWS) on EEG in 10 BPD patients without significantly affecting REM latency or REM%, as it had also been demonstrated to do in people without PD given the drug in other studies. These observed changes were also demonstrated in the study's 10 control BPD participants given placebo; SWS increased from 4.14% to 11.81% in the carbamazepine group (REM% 25.89–20.89) and 8.64%–13.3% in controls (REM% 25.5–21.87), significantly different in both groups by time but not between groups. Furthermore, while improvements in SOL and sleep maintenance might also have been expected given previous reports of this benefit observed in non-BPD participants of other trials, this was not found among either cases or controls.

Thirty-three BPD patients with distressing nightmares treated open-label with doxazosin by Roepke et al. (2017) reported significant improvements in PSQI-A sleep scores at 4 weeks and further improvement by 12 weeks of treatment. Among all patients in the study, there were discontinuation rates of one-quarter by Week 12, and those discontinuing medication reported similar improvements in sleep from baseline as those who continued treatment.

Other interventions

Using actigraphy outcomes in a study of morning bright light therapy, Bromundt et al. (2013) demonstrated that 13 women with BPD who slept longer and awoke later than controls at baseline woke earlier and slept less overall after the intervention, now equivalent to the sleep pattern of controls. Increased sleep latency and poor sleep efficiency, two other parameters by which the BPD group

significantly differed from non-BPD participants at baseline, did not change. The authors also reported that symptoms of daytime alertness improved and self-reported atypical depression reduced, although other secondary outcomes of typical depression did not.

Using health-register data from a region of western Sweden, Steingrímsson et al. (2022) followed up on the sleeping medication prescriptions of 1785 adults with mental disorders, of whom 268 had been diagnosed with PD, over 1 year after having collected a weighted blanket from a public pharmacy. While prescriptions of sleep-aid medication reduced for those with anxiety and unipolar affective disorders, there was no change in use among patients with psychotic or PDs. This study was unable to capture psychiatric comorbidity, preferencing psychotic and affective disorders over PD, and so it is likely that many other patients with PD cooccurring with other psychiatric disorders were not captured in this work. In another pre-post study of psychiatric inpatient admissions, Schennach et al. (2019) report on 101 people with PD who, having started with the second highest group mean PSQI score (10.3; a score >5 is generally accepted to represent sleep disturbance; Buysse et al., 1989), representing some of the poorest sleep among diagnostic groups on admission, enjoyed the greatest benefits to their subjective sleep quality on discharge with a 1.7 (±3.8) point reduction. Importantly, confounding data like sedative medication use and length of admission were unavailable.

Subjective–objective differences

Two studies reported both objective and subjective outcomes. In Weinhold et al. (2017), neither PSG nor total PSQI score showed meaningful improvement over time or between groups, (aside from the aforementioned reductions in sleep on-set latency in the intervention group post-intervention, unstained at 6 months). Reporting of breakdown PSQI scores might have facilitated a better comparison between PSG-derived objective sleep–wake measures and subjective change in these facets. On the other hand, regarding Petrov et al. (2019), specific breakdowns of sleep diary data permit a comparison of pre- and post-behavioral therapy for insomnia among people who do and do not meet criteria for an OCPD diagnosis. At baseline, both groups underestimated their TST and sleep efficiency and overestimated their sleep on-set latency, but only the OCPD group overestimated the amount of time they were awake after sleep onset. Post-intervention, both groups accurately perceived their TST and SE and continued to overestimate their SOL. At 1 year, the OCPD group continued to accurately perceive their TST, SE, and now also their SOL, and for the non-OCPD group, there was continued accuracy of perception of TST and SE but continued overestimation as at other time points of their SOL. These results might tentatively appear to demonstrate benefits to the accuracy of perception of sleep state following behavioral therapy for insomnia and withdrawal of hypnotic medication for both those with and without OCPD.

DISCUSSION

This systematic review is the first to collate evidence on the effects of interventions on sleep outcomes among adults with a diagnosis of PD. Given a broad search strategy, we were able to identify and report on several small and non-randomized studies. We also employed a robust approach to seeking unreported data.

Evidence quality and study heterogeneity are such that no firm conclusions can be drawn from this evidence synthesis. However, individual studies did demonstrate effects on several sleep quality outcomes from a collection of interventions. While most studies demonstrated some improvement in their reported outcomes, there were no particular patterns of effects on sleep quality outcomes comparable between studies. No study with subjective measurements reported a worsening of sleep quality post-intervention. No study using self-reported subjective sleep measures reported breakdown scores, which might have given a better sense of how sleep is improved following intervention and offered an opportunity for narrative comparison with objective outcomes. Different patterns of outcomes are likely to reflect different mechanisms of action of the intervention, methodological limitations, differences in baseline sleep profiles, or other confounding factors, particularly physical and psychiatric comorbidity and medication.

Diagnostic groupings were too small to adequately evaluate whether one category of PD responded more or less favorably to treatment than others. To date, while a systematic review has synthesized studies describing the sleep phenotype of individuals with borderline PD (Winsper et al., 2017), those with other previously formulated A or C clusters of PD do not appear to have been studied and are likely to represent different dimensional personality profiles (Kim et al., 2021) and herein baseline sleep profiles (Hintsanen et al., 2014), which are also likely to differentially affect response to intervention. Case in point: around core unifying criteria, the new ICD-11 and DSM-V alternative models of PDs will now better position work on PD to consider trait domains, translatable with the Big 5 personality traits, which have a long-standing evidence base in sleep research.

Moreover, subjective sleep-state misperception—the experience of having slept for significantly shorter or longer than observed by objective measures—may be more common in people with PD (Bastien et al., 2008; Philipsen et al., 2005), particularly OCPD (Ruiter et al., 2012) and given the signal from Petrov and colleagues that behavioral therapy for insomnia and supported withdrawal from hypnotic medication may improve the accuracy of perception of sleep-state, future work should look at both subjective and objective outcome measures, given that subjective sleep quality appears, more than objective quality to be associated with dysfunction and emotion regulation in some studies (Jenkins et al., 2022).

There were a number of persistent methodological concerns in these studies, not least small sample sizes, few studies with comparator groups, and a high risk of bias throughout. In the majority of included studies (5/9), patients with PD were not the primary focus of interest and formed a subgroup from a larger body of patients with mental illness or experienced PD as cooccurring with PTSD. This is a surprising finding given the frequency with which sleep problems are reported by people given a diagnosis of PD in clinical settings.

This review only included studies using objective or validated subjective measures of sleep disturbance. Many studies screened for this review utilized sleep quality outcomes using unvalidated subscales of more established assessment tools, particularly for mental health conditions or quality of life. For example, Ziegenhorn et al. (2009) conducted a small randomized-controlled trial of Clonidine in BPD utilizing three selected morning-sleep items from an established assessment tool, and Yip et al. (2021), examining the effects of electroconvulsive therapy in people with PD and treatment-resistant depression, analyzed four selected sleep items from a depression assessment tool. The concern with using subscales is that these Likert-type items have not previously been assessed for their construct validity and therefore their suitability to represent a coherent sleep concept as a continuous variable. Additionally, a large number of pharmaceutical trials in PD screened in this review recorded a range of broadly related sleep outcomes, usually as side effect monitoring, including sedation and daytime somnolence, with unvalidated outcome measures. Atypical antipsychotics and sedating antidepressant medications are commonly prescribed off-label for specific symptoms like emotional regulation, agitation, and impulsivity, albeit no apparent evidence has demonstrated efficacy of these medications for sleep outcomes in this specific population.

Such a small number of studies meeting inclusion criteria was surprising, given that a wealth of interventional studies have aimed to evaluate their impacts on the quality of sleep of people with mental health problems by a range of methods and invariably employ robust sleep quality outcomes (Wu et al., 2015). However, a notable absence was people with PD from many of these studies, particularly those of CBTi, the recommended first-line intervention for insomnia by several international organizations (Hertenstein et al., 2022). These studies appear to commonly exclude people with a history of psychotic illness, substance misuse, or PD. Albeit, a recent protocol for CBTi delivered to people with PD by mobile app has recently been published (van Trigt et al., 2022). Despite the evidence and the clinical frequency of sleep disturbance reported by people with PD, many further studies do not assess for Axis II diagnoses or do not report on any participants with cooccurring PD in their populations (Talbot et al., 2014). There have been longstanding difficulties in researching personality pathology, perhaps in part because of limited clinical and research funding compared with its prevalence (Zimmerman & Gazarian, 2014).

Another notable absence was the lack of studies evaluating sleep outcomes from established treatments for PD, including dialectic behavior therapy, psychodynamic psychotherapies, or commonly used off-license treatments like atypical antipsychotics (Cochrane Developmental, Psychosocial and Learning Problems Group, et al., 2022). Sleep outcome measures could be measured in routine or new treatment approaches to core symptoms of PD, and in so doing, include sleep quality as a factor in explanatory modeling. Moreover, to date, no study using dimensional models of personality pathology has assessed sleep quality or response to targeted treatments, and this would be a natural next step for research in this field along with the integration of objective and subjective measures and better assessment and control of confounding mental state disorders, among other contributory factors like hypnotic and psychotropic medication and substance misuse. While sleep outcomes were recently considered for a set of specialist consortium-recommended patient-reported outcome measures for PD clinical trials (Prevolnik Rupel et al., 2021), other outcomes were ultimately given priority.

CONCLUSIONS

Disturbed sleep is a common and concerning problem for people with PD and has an impact on quality of life as well as bidirectional relationships with many core features of PD, including emotional dysregulation, self-harming behaviors, dissociative episodes, drug and alcohol use, and co-occurring anxiety and depressive

symptoms. Improving the sleep of people with PD may therefore hypothetically offer an avenue to improving quality of life and mental health outcomes. This review set out to collate the evidence on the effect of any intervention on sleep quality outcomes in people with PD. The overall quality of evidence available is poor, and to date, it is based on a handful of comparative trials and case series with high risks of bias. No clear conclusion can be drawn other than that further research is indicated. Interventions to improve sleep quality for people with PD could also focus on causal mechanisms, including sleep-related cognitive distortions, pre-sleep hyperarousal, changes in cooccurring mental illness, sleep-state misperception, sleep regularity, and circadian profiles, among others. Trials of CBT for insomnia delivered to people with CEN are glaringly absent and should be a priority for future studies. Moreover, the inclusion of people living with PDs in future trials of interventions to improve sleep quality is recommended, as well as the inclusion of validated sleep outcomes in trials of established PD treatments.

AUTHOR CONTRIBUTIONS

All authors meet ICJME criteria for authorship. All authors were involved in the development of the research question and search strategy, interpreting results, and reading, editing, and agreeing to the final version of this article. Jacob D King and Shee Cheng conducted the search strategy and risk of bias assessments. Jacob D King drafted the initial manuscript.

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CONFLICT OF INTEREST STATEMENT

JM is an associate editor of *Personality and Mental Health*. Other authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

All process data relating to this systematic review is available from the corresponding author on reasonable request.

ETHICAL STATEMENT

Ethics panel approval for this piece of work was deemed not to be necessary by the National Health Service - Health Research Authority self-complete decision tool.

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