The sleep phenotype of Borderline Personality Disorder: A systematic review and meta-analysis

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Aim: To delineate the sleep profile of Borderline Personality Disorder (BPD).

Method: A meta-analysis to synthesise findings on the objective and subjective sleep characteristics of BPD.

Results: We identified 32 studies published between 1980 and December 2015. Meta-analysis indicated significant differences between BPD and healthy control groups across objective sleep continuity (sleep onset latency, total sleep time, sleep efficiency) and architecture (rapid eye movement latency/density, slow wave sleep) measures, and self-reported sleep problems (nightmares, sleep quality). Findings were independent of depression (in clinical and community populations), and concomitant psychotropic medication use. There were few significant differences between BPD and clinical (majority depressed) control groups.

Conclusion: BPD is associated with comparable sleep disturbances to those observed in depression. These disturbances are not solely attributable to comorbid depression. Given growing evidence that sleep disturbance may exacerbate emotional dysregulation and suicide risk, treatments for BPD should explicitly address sleep problems. Future studies should utilise prospective designs to ascertain whether (and in which circumstances) sleep problems predate or follow the onset of the disorder.

1. Introduction

Borderline Personality Disorder (BPD) is a chronic mental disorder, which often first manifests in adolescence (Kaess, Brunner, & Chanen, 2014; Winsper et al., 2016; Winsper et al., 2015). It comprises a wide range of debilitating symptoms including mood instability, inappropriate anger, impulsive behaviour, disturbances in self-identity, unstable relationships, abandonment fears, and self-harm (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004).

Studies indicate that individuals with BPD may experience sleep disturbances in comparison to healthy (Bastien, Guimond, St-Jean, & Lemelin, 2008; Benson, King, Gordon, Silva, & Zarcone, 1990) and depressed (De la Fuente et al., 2004) controls. Nevertheless, the sleep profile of BPD has received relatively limited clinical and research attention (Winsper & Tang, 2014). A greater understanding of sleep disturbance in BPD may help enrich treatment protocols, which currently place rather limited emphasis on sleep difficulties (National Institute for Clinical Excellence, 2009; Selby, 2013). Furthermore, elucidating the extent to which BPD is independently associated with sleep problems may provide a more coherent picture of the phenomenological and biological nature of this complex disorder (Goodman, New, Triebwasser, Collins, & Siever, 2010; Simor & Horváth, 2013).

Narrative reviews have considered aspects of sleep in BPD (please see Table 1 for a list of acronyms and associated full names used throughout the review). Hafizi (2013) concluded that BPD is associated with several sleep abnormalities, including disturbances in continuity, reduced Rapid Eye Movement (REM) latency (though findings were inconsistent), and nightmares. Similarly, Simor and Horváth (2013) reported that sleep fragmentation, alterations in Slow Wave Sleep (SWS) – though again results were mixed, shortened REM latency, and dysphoric dreaming are prevalent in BPD. In a narrative synthesis of 12 polysomnography (PSG) studies, Oltmanns and Oltmanns (2015a) reported differences in sleep between BPD patients and healthy controls including less total sleep time

(TST), decreased sleep efficiency (SE), longer sleep onset latency (SOL) and more frequent arousals. The authors concluded that BPD may be independently associated with sleep disturbances.

[Insert Table 1 about here]

While these narrative reviews are informative, inconsistent results across studies make robust conclusions regarding sleep in BPD difficult to formulate (Hafizi, 2013). Inconsistencies are likely to be attributable to small samples sizes resulting in underpowered analyses (Harty, Forkner, Thompson, Stuewig, & Tangney, 2010), and variations in study methodology (e.g., methods of assessment, differing sample characteristics) which may moderate (or mask) pertinent group differences. Finally, the lack of systematic search procedures may have led to the omission of important studies inflating risk of biased conclusions (Cipriani & Geddes, 2003).

To address these issues, we conducted a systematic review and meta-analysis of sleep studies in BPD populations. Our main aim was to quantitatively examine the sleep profile (i.e., continuity, architecture, and nightmares) of individuals with BPD, and determine whether it can be distinguished from that of individuals with other (and no) mental disorders. Specifically, we addressed the following research questions:

- Do individuals with BPD significantly differ from healthy and clinical controls on objective measures of sleep continuity (i.e., SOL, TST, WASO, NWAKE, SE) and sleep architecture (i.e., stage 1 sleep, stage 2 sleep, SWS, and REM)?

- Are effect sizes for group differences in sleep moderated by study features (i.e., co-morbid depression status, sample frame, sleep assessment, gender, age, and study quality)?

- Do individuals with BPD significantly differ from controls on subjective measures of sleep (e.g., nightmares, sleep quality)?

- Is there a subjective-objective discrepancy in the sleep measures of individuals with BPD?

2. Method

2.1. Search and selection of studies

Prior to formulating the protocol, C.W conducted a pilot search to ensure that a systematic review pertaining to the research questions had not been previously published or registered. The Cochrane Database of Systematic Reviews (CDSR), the Centre for Reviews and Dissemination (CRD), and www.pubmed.gov (Sayers, 2008) were accessed for the scoping search. We used MOOSE: Meta-analysis of Observational Studies in Epidemiology (Stroup et al., 2000) and PRISMA: Preferred Reporting Items for Systematic Review and Meta-analysis (Moher et al., 2015) guidelines as a framework. Methods of analysis and inclusion criteria were specified in advance and documented in the protocol (Liberati et al., 2009). We searched Embase, PsycINFO, and PubMed from 1980 (when BPD was first introduced as a disorder in the DSM-III) to December 2015. Search terms were agreed by the authors following consultation of relevant published reviews and the pilot search. We used the grouped terms (borderline* OR BPD OR "emotionally unstable personality disorder") and (sleep* OR nightmare* OR dream* OR REM OR insomnia OR hypersomnia OR

wakefulness OR polysomnograph* OR EEG OR circadian*). Reference lists of retrieved articles and review papers were inspected to enhance search sensitivity. T.L & C.W independently searched 100% of the abstracts for full text retrieval. C.W and M.G independently searched 100% of the full text articles for inclusion in the review.

2.2. Inclusion criteria

First, we defined criteria for studies to be included in the overall review, which were applied to the systematic narrative synthesis and meta-analytic sections. Second, we applied further inclusion criteria for the meta-analysis.

To be included in the overall review studies had to:

1) Be published in English;

2) Be primary research in a peer reviewed journal;

3) Report group comparisons between participants with BPD and healthy or clinical controls on any sleep characteristic, or report associations between BPD symptoms and any sleep characteristic;

4) Be based on adolescent or adult samples (defined as 11 years of age or older);

5) Include a clinical, high-risk (e.g., prison) or community population;

6) Report either an objective (e.g., EEG) or subjective (e.g., self-report) assessment of sleep.

To be included in the meta-analysis, studies had to also:

7) Use a validated assessment of BPD (i.e., questionnaire, structured interview, clinical diagnosis);

8) Report data (e.g., group means, standard deviations) to allow for the calculation of an effect size for group differences;

2.3. Exclusion criteria

1) Case reports;

2) Studies of sleep problems in personality disorders in general rather than BPD
   specifically

2.4. Data extraction

A data extraction form was developed prior to manuscript review. It included: author details,

country of study, sample characteristics (i.e., age, sex, and sample frame), study design, BPD

and sleep assessments, and effect sizes and associated variance for the sleep variables. The

data extraction sheet also included a quality assessment (QA) tool based on the Newcastle-

Ottawa Scale (NOS), which can be adapted for non-randomised cross-sectional and case-

control studies (Wells et al., 2000). The NOS is based on a star system in which each case

control study is assessed on three broad domains: selection (maximum of 4 stars);

comparability (maximum of 2 stars) and exposure (maximum of 3 stars). We used the

adapted scale by Herzog et al. (2013) for cross sectional studies, covering the similar domains

of selection (maximum of 5 stars); comparability (maximum of 2 stars); and outcome

(maximum of 3 stars). The content validity and inter-rater reliability of the NOS have been

established (Wells et al., 2000). As we expected to identify a limited number of relevant

articles, we did not exclude studies according to a study quality threshold. Instead, we used

moderator analysis to determine whether study quality impacted on pooled results.

2.5. Meta-analysis

Meta-analysis for each sleep variable was conducted by computing weighted summary

measures using *Stata version 13* (Stata Corp, College Station, TX, USA). Due to

methodological and sample variations, we expected studies to be heterogeneous, and that

sleep disturbances would vary more than expected by chance alone. Thus, we selected the

random-effects model (DerSimonian & Laird, 1986) with the inverse-variance method to

calculate summary estimates. We compared BPD groups to healthy and clinical controls. As

most (but not all) clinical control groups were depressed patients, we repeated the analysis

with depressed only controls as a sensitivity test. In view of the potential effects of

psychotropic medications on sleep (Whitney et al., 1998), we conducted a second sensitivity

test excluding studies in which patients were taking psychotropic or sleep medication at the

time of testing.

The standardised mean difference (SMD) was the principal summary measure. The

SMD is particularly appropriate for the synthesis of studies assessing the same concept (e.g.,

subjective sleep quality) with a variety of continuous scales (e.g., Pittsburgh Sleep Quality

Index, Schlaffragebogen B scale). Heterogeneity was quantitatively assessed using the I-

squared statistic to ascertain whether pooled studies represented a homogeneous distribution

of effect sizes. Evidence regarding publication bias was assessed using funnel plots. Funnel

plots are scatter graphs, which plot the effect sizes of individual studies (horizontal axis)

against a measure of study size (vertical axis). Publication bias (reported in **Table 3**) is

indicated by asymmetrical funnel plots, e.g., if smaller studies showing no statistical effect

remain unpublished there will be a gap in the right (or left for negative effects) bottom side of

the graph (Sterne & Harbord, 2004).

2.6. Subgroup analysis and meta-regressions

We addressed research question two (i.e., study factors associated with heterogeneity) using

subgroup analyses and meta-regression. We selected factors previously suggested to

influence sleep including: comorbid depression status (Holsboer-trachsler & Seffritz, 2000),

age (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004), gender (Mongrain, Carrier, &

Dumont, 2005), sample type (Topf & Thompson, 2001), and sleep assessment tool (Hornung

et al., 2008). We performed subgroup analysis by categorical factors including: comorbid

depression status (0=no history of, or current, depression; 1=no current depression; 2 =

comorbid depression); sample type (0=inpatients; 1=outpatients; 2=prison population); sleep assessment (0=lab polysomnography; 1=less optimal/indirect sleep assessment method); and gender (0=mixed sample; 1 = all females; 2 = all males). We then performed meta-regression to estimate the extent to which each of these variables (and age and QA score entered as continuous variables) explained the observed heterogeneity in pooled SMD estimates (Higgins & Thompson, 2002).

3. Results

3.1. Search results

Of the 972 abstracts scanned, we identified 49 articles for full text retrieval. Interrater agreement was very good (Kappa=0.87). C.W. and T.L discussed any discrepancies, which were largely due to uncertainty regarding whether the study reported relevant sleep variables (e.g., in some cases it wasn't clear whether EEG measures referred to sleep specifically). If there was any doubt over whether an abstract should be included for full text retrieval, we made the decision to include it. Of the 49 full text articles inspected, 32 were identified for inclusion in the review. Interrater agreement was excellent (Kappa=0.90). Of these 32 studies, 20 were also eligible for inclusion in the meta-analysis, 14 (two studies were included in the MA and narrative synthesis) were synthesised narratively (see Figure 1). Reasons for non-inclusion in the meta-analysis included: the reporting of overlapping data (De la Fuente et al., 2004); insufficient data to convert into a common effect size (Bromundt et al., 2013); and the uniqueness of the sleep factor (Dagan, Stein, Steinbock, Yovel, & Hallis, 1998), i.e., it wasn't assessed in a minimum of two studies.

[Insert Figure 1 about here]

See **Table 2** for details and main results of studies. Two studies were prospective (Lereya, Winsper, Tang, & Wolke, 2016; Selby, Ribeiro, & Joiner Jr, 2013), the remainder were cross-sectional. Twenty-four studies (inpatients, n=11; outpatients, n=13) used clinical samples, five community samples (1 nationally representative sample, 1 large community cohort of young adolescents, 1 student sample, 1 older population, and 1 sample recruited for self-reported difficulties with dysregulated behaviours), and three prison populations. Eleven studies reported an objective sleep measure, fifteen a subjective sleep measure, and six both objective and subjective measures. Nearly all studies examining objective sleep measures used polysomnography in laboratory conditions. One study utilised a portable electroencephalogram sleep recording device (Hornung et al., 2008), while two utilised wrist actigraphy (Bromundt et al., 2013; Huỳnh, Guilé, Breton, & Godbout, 2015).

Most studies with clinical control groups included depressed patients with the exception of one group of prisoners with Antisocial Personality Disorder (Lindberg et al., 2003), one group of patients with insomnia (Bastien et al., 2008), and one group of adolescent outpatients with bipolar disorder (Huỳnh et al., 2015). Most studies reported a psychotropic medication wash-out period (usually of 2 weeks) prior to sleep assessment; five did not report a wash-out period (Huỳnh et al., 2015; Plante, Frankenburg, Fitzmaurice, & Zanarini, 2013b; Selby et al., 2013; Simor, Csóka, & Bódizs, 2010; Taherifard, Abolghasemi, & Hajloo, 2015). Adult samples were used in most studies; only three had adolescent samples (Dagan et al., 1998; Huỳnh et al., 2015; Lereya et al., 2016).

[Insert Table 2 about here]

3.2. Quality assessment

The findings from the quality assessment are reported in **Supplementary Tables 1 & 2**.

Scores ranged from 2 to 7 points (out of a possible 9/10). Nearly all studies performed poorly

on representativeness of sample and response rate (which was often not reported in studies, precluding assessment). Most studies performed well on comparability of cases and controls by either matching control groups carefully, or controlling for pertinent confounders.

3.3. Meta-analysis results

3.3.1 Studies with objective sleep assessments

Detailed results (including the Standardised Mean Difference: SMD, confidence intervals, heterogeneity statistics, sensitivity analysis, p-values, and publication bias) are reported in **Tables 3** and **4**. Please see **Figures 2, 3** & **4** for summary forest plots. Below we outline the direction of results.

BPD versus healthy control groups

Individuals with BPD had significantly longer SOL and WASO, and greater NWAKE and REM density than healthy control groups. They also had significantly shorter TST and REM latency, and lower SE than healthy control groups. Groups did not differ in % of REM sleep, and stage-1 or stage-2 sleep (Figure 2).

[Insert Table 3 here]

[Insert Figure 2 here]

There was significant heterogeneity across studies for SOL, TST, NWAKE, SE, REM latency, REM density and stage-2 sleep. In sub-group analyses (Table 4), many significant differences between BPD and healthy groups remained robust across depression status (i.e., no history, no current, comorbid) groups (see Figure 3); differences were mainly robust in inpatient groups (TST, SOL, SE, and REM latency); those assessed with polysomnography (TST, SOL, SE, and REM latency); and mixed sex samples (TST, SOL, SE, REM latency, REM density).

[Insert Table 4 here]

(Insert Figure 3 here]

There were few significant predictors of heterogeneity in the meta-regression analyses. Sleep assessment tool (i.e., the use of polysomnography significantly predicted a negative SMD of TST) and sample type (i.e., the SMD of SOL was significantly greater in the mixed sex sample than the female only sample, and the SMD of NWAKE was significantly greater in the male sex group than the mixed sex samples) significantly accounted for heterogeneity for some of the sleep indices (see Supplementary Table 3).

BPD versus clinical controls

Individuals with BPD did not significantly differ from all clinical control groups or depressed only groups on any of the sleep indices (see Table 3 and Figure 4).

[Insert Figure 4 here]

There was significant heterogeneity across studies for SE, SWS, and REM latency. Sub-group analyses for these indices indicated some subtle differences according to study factors (full results on request from authors). For SE, BPD individuals who had never been depressed, and outpatients, had significantly higher SE than CCs. In contrast, BPD individuals who were prisoners had significantly lower SE than CCs. For SWS, BPD individuals without co-morbid depression and those who were prisoners had significantly less slow wave sleep than CCs. For REM latency, BPD patients who had never been depressed had longer REM latencies than CCs, while REM latency was significantly shorter in BPD individuals than controls in the prison population.

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There were few significant predictors of heterogeneity in the meta-regression analyses. Sample type (i.e., the SMD of SE was significantly lower in the prison population than the inpatient groups) and study quality (i.e., the SMD of SE was higher in the studies of higher quality) significantly moderated effect size for some of the sleep indices (full results on request from authors).

3.3.2. Studies with self-reported sleep assessments

Studies considering self-reported sleep characteristics examined nightmares, sleep quality, SOL, TST, and SE. See **Table 5** and **Figure 5** for a summary of the meta-analysis findings.

[Insert Figure 5 here]

[Insert Table 4 here]

Individuals with BPD reported significantly more nightmares, significantly poorer sleep quality, and significantly longer SOL than controls. Groups did not differ in self-reported TST or SE. There was significant heterogeneity across studies for nightmares, sleep quality, TST, and SE. We did not conduct meta-regressions due to the small number of studies. Sub-group analysis indicated that BPD patients without co-morbid depression, and both inpatients and outpatients experienced significantly more nightmares than controls. Subjective sleep quality was worse in BPD patients without co-morbid depression, and in both inpatients and outpatients (full results on request from authors).

3.4. Summary of results from the narrative synthesis

We narratively summarised results from studies that could not be quantitatively combined. Below we outline the pattern of findings. Please refer to **Table 2** for more details on individual study results.

3.4.1. Circadian rhythm abnormalities

The circadian rhythm (or "body clock") is an endogenously-driven roughly 24-hour cycle, which tells our bodies when to sleep, and regulates other physiological process. Three studies indicated irregularities in circadian rhythms in BPD. Individuals with BPD appear more susceptible to delayed sleep phase syndrome: DSPS (Dagan et al., 1998); experience greater variability in Relative Amplitude, i.e., the distribution of activity between day and night (Bromundt et al., 2013); and experience wider sleep variability between weekdays and weekends (Huỳnh et al., 2015).

3.4.2. Nightmares

Findings from the narrative synthesis were consistent with the meta-analysis, demonstrating significant associations between nightmares and BPD in clinical (Lloyd, Overall, Kimsey, & Click Jr, 1983) and community (Claridge, Davis, Bellhouse, & Kaptein, 1998; Lereya et al., 2016) populations. Furthermore, although preliminary, there was evidence for a prospective association between nightmares and BPD symptoms over both short (Selby et al., 2013) and long (Lereya et al., 2016) periods of time. These studies suggest that nightmares may exacerbate daytime dysregulation contributing to worsening symptoms (Selby et al., 2013), and possibly the development of BPD (Lereya et al., 2016).

3.4.3. Self-reported sleep-quality and maladaptive sleep cognitions

Also consistent with the meta-analysis, there was evidence for a significant association between subjective poor sleep quality/insomnia symptoms and BPD in clinical (Bromundt et al., 2013; Sansone, Edwards, & Forbis, 2010), prison (Harty et al., 2010), and community (Oltmanns, Weinstein, & Oltmanns, 2014; Selby, 2013) populations. Plante, Frankenburg, Fitzmaurice, and Zanarini (2013a) found that maladaptive cognitions about sleep (i.e., perceived consequences of insomnia; worry/helplessness about insomnia; expectations about

sleep; and attitudes about sleep medication) distinguished those who continued to meet BPD criteria from those who didn't.

4. Discussion

We examined the specific sleep profile of individuals with BPD by pooling the results of studies comparing BPD patients to healthy and clinical controls. Further, we conducted sub-group analyses to determine the extent to which group differences were moderated by pertinent study factors. Studies not suitable for meta-analysis were synthesised narratively.

4.1. Summary of findings

4.1.1. Do individuals with BPD significantly differ from healthy and clinical controls on objective sleep measures, and are pooled results moderated by study factors?

The meta-analysis indicated robust differences between BPD and HC groups across sleep continuity measures (i.e., SOL, TST, SE, WASO, and NWAKE). Our results extend previous reviews (Hafizi, 2013; Oltmanns & Oltmanns, 2015a) by quantitatively demonstrating significant differences between BPD and HC groups across all sleep continuity measures. Sub-group analyses indicated some variations according to study-based characteristics.

Differences were robust in sub-groups of BPD patients without any history of (SOL, TST, and SE) or concurrent (NWAKE) depression. This lends some support to the previous suggestion that sleep continuity problems are *independently* associated with BPD, rather than being a consequence of comorbid depression (Oltmanns & Oltmanns, 2015a). Subgroup analysis also demonstrated that differences between BPD and HC groups were significant in studies utilising polysomnography (rather than wrist actigraphy or other non-lab-based devices), and in inpatient populations. The more robust findings in polysomnography studies are perhaps unsurprising as this technique is often considered as the gold standard of sleep assessment methods (Hornung et al., 2008). The more robust findings in inpatient groups

could be partly attributable to the severity of BPD symptoms, which may be associated with more pronounced objective sleep problems (Bastien et al., 2008).

Results were less consistent for sleep architecture. BPD groups did not significantly differ from HCs in stage 1 or 2 sleep, or percentage of REM sleep. They did, however, demonstrate significantly less SWS. This result contrasts with previous reviews, which did not identify a difference in SWS between BPD and HCs (Oltmanns & Oltmanns, 2015a), or suggested a potential increase in SWS in BPD (Simor & Horváth, 2013). Discrepant conclusions across reviews are likely partly due to a lack of power in individual studies (i.e., null findings, which when combined in meta-analysis reveal significant associations).

Also in contrast to conclusions from previous reviews (Oltmanns & Oltmanns, 2015a), we found that patients with BPD experienced significantly shorter REM latency than HCs. This finding was robust across several sub-groups, including depression status, sample type, and sex. Our finding of increased REM density in BPD is consistent with previous reports on BPD (Oltmanns & Oltmanns, 2015a), while reduced REM latency and increased REM density have also been robustly associated with depression (Palagini, Baglioni, Ciapparelli, Gemignani, & Riemann, 2013). Findings from the sub-group analyses (i.e., significant abnormalities in BPD groups with no history of, or current, depression), however, support that abnormalities in REM latency and density may not be directly attributable to comorbid depression in BPD.

Comparisons with clinical controls (CCs)

Meta-analyses of objective sleep studies indicated no overall significant differences between BPD groups and CCs across sleep measures. For most of the sleep indices, heterogeneity across studies was non-significant precluding sub-analysis. However, where sub-group analysis was indicated, it revealed some potential subtle differences between groups. BPD patients who had never been depressed and outpatient groups had significantly higher SE

than CCs. This could be attributable to the severity of BPD (i.e., outpatients and those without a history of depression could have a less severe form of BPD). We also found that BPD patients without current comorbid depression and prisoners experienced significantly less SWS than CCs. This may be associated with increased alcohol consumption in patients with BPD (Lester, Rundell, Cowden, & Williams, 1973) or an overlap between BPD and psychotic symptoms (De la Fuente et al., 2004). Our hypotheses remain tentative, however, as these findings were not robust across the majority of BPD groups.

In a previous review comparing the endophenotypes of BPD and Major Depressive Disorder (MDD), it was concluded that BPD and depressed patients exhibit differing sleep profiles (Goodman et al., 2010). Our quantitative synthesis of all available studies does not support this conclusion. Overall, BPD and depressed patients appear to share similar disturbances in sleep continuity and architecture, which may suggest potential overlapping biological processes in this domain.

4.1.2. Do individuals with BPD significantly differ from controls on subjective measures of sleep?

Converging evidence demonstrates that inpatients, outpatients, and community populations with BPD (or BPD symptoms) report significantly more nightmares than HCs. Again, associations appear independent of comorbid depression. Complex associations between trauma, nightmares, and BPD are indicated (Claridge et al., 1998; Lereya et al., 2016; Semiz, Basoglu, Ebrinc, & Cetin, 2008). Recent prospective evidence suggests that nightmares could exacerbate reactions to early trauma increasing risk of subsequent BPD (Lereya et al., 2016). These intriguing findings are consistent with contemporary developmental theories for BPD, which highlight the potentiation of emotional and behavioural dysregulation across development (Crowell, Beauchaine, & Linehan, 2009).

4.1.3. Is there a subjective-objective discrepancy in the sleep findings for BPD?

Consistent with the objective sleep measures, patients with BPD reported significantly poorer sleep quality and significantly longer SOL than HCs. In contrast to the objective results, they did not differ from HCs on subjective measures of TST and SE. Previously, it was suggested that individuals with BPD (common to those suffering from paradoxical insomnia) show marked discrepancies between subjective and objective sleep measures (Bastien et al., 2008), i.e., they report worse sleep quality than HCs while objectively demonstrating similar sleep patterns (Bastien et al., 2008; Philipsen et al., 2005). Our findings do not support that individuals with BPD are likely to over-estimate their sleep problems. They are, however, likely to engage in maladaptive cognitions about their sleep, which could potentially impede recovery (Plante et al., 2013a).

4.1.4. Strengths and Limitations

Our review has strengths. First, we utilised systematic search procedures to reduce risk of bias and ensure comprehensive coverage of the literature. Second, by utilising meta-analysis (and thus increasing statistical power) we were able to reveal patterns in the research literature that could not be detected through narrative synthesis. This has enabled us to move knowledge forward regarding the specific sleep profile of BPD.

It is also important to highlight the considerable limitations of the available evidence and hence the meta-analytical approach we took. First, although interest in sleep and BPD is growing (Oltmanns & Oltmanns, 2015a), there were a limited number of studies eligible for inclusion in the meta-analysis. Consequently, we had limited power to detect significant moderators of effect size in the meta-regression analyses, making it difficult to definitively ascertain the independent effects of study factors. This was also apparent in the sub-group analyses, where some categories were represented by very few studies. Relatedly, while studies were heterogeneous and varied in quality, we could not exclude any based on the

quality assessment due to the limited numbers. Our metaregression analysis did not (overall) indicate any major effect of study quality on effect size. However, as most studies scored poorly in terms of representativeness and response rate, this could impact on the generalisability of our results.

Second, we undertook multiple analyses to explain the observed heterogeneity across studies, which could have resulted in type I errors. Nevertheless, we pre-planned and grounded our analyses (and research questions) within the extant literature to avoid data dredging and reduce the probability of false positives (Thompson & Higgins, 2002).

Third, some of our findings may have been subject to publication bias. This could have resulted in an over estimation of group differences, especially for TST where the funnel plot indicated that bias could have played a role.

Fourth, while it is interesting that many of the objective sleep findings were robust independent of comorbid depression, the exclusion of BPD patients with depression will have limited the generalisability of the results from these clinical subgroups, as comorbid depression is relatively common in BPD (Oltmanns & Oltmanns, 2015a; Zimmerman & Mattia, 1999). Nevertheless, significant sleep- BPD associations were also found in community (Oltmanns et al., 2014; Selby, 2013) and prison (Harty et al., 2010) studies following statistical adjustment for depression symptoms. Further, sub-analyses demonstrated that BPD patients both with and without comorbid depression evinced similar sleep disturbances. This converging evidence supports the independence of sleep-BPD associations across diverse populations.

Fifth, some studies concentrated on the effects of comorbid or past history of depression (Akiskal, Yerevanian, Davis, King, & Lemmi, 1985), or just controlled for depression symptoms (Oltmanns et al., 2014), rather than a full range of psychiatric disorders. Bipolar disorder and post-traumatic stress disorder, for example, have been

associated with both BPD (Zimmerman & Mattia, 1999) and sleep disturbance (Kobayashi, Boarts, & Delahanty, 2007; Plante & Winkelman, 2008). Nevertheless, the majority of studies did exclude participants with other psychiatric disorders (e.g., psychotic disorder, bipolar disorder, substance abuse, PTSD) or controlled for other psychiatric symptoms, e.g., substance abuse, PTSD (Harty et al., 2010; Selby et al., 2013), adding support to the independence of observed associations between BPD and sleep disturbance.

Sixth, although most studies included in our meta-analysis incorporated a medication wash-out period, in most cases this lasted for a maximum of two weeks. Thus, it is possible that medication taken prior to the study may have had a residual impact on the sleep disturbances assessed (Battaglia, Ferini-Strambi, Smirne, Bernardeschi, & Bellodi, 1993). In a study by Cowdry and Gardner (1988), for example, BPD patients receiving benzodiazepines experienced an increase in serious dyscontrol.

Finally, due to a lack of prospective studies, we were unable to ascertain from the extant literature whether sleep problems predate the development of BPD or are a consequence of the disorder. There is limited evidence to support that sleep problems may be evident before the development of the disorder and contribute to its aetiology (Lereya et al., 2016; Selby et al., 2013). There is also tentative evidence to suggest that ongoing sleep problems can contribute to the chronicity of the disorder (Plante et al., 2013b). It should be noted, however, that associations between sleep disturbance and BPD are likely to be bi-directional, as a number of symptoms and behaviours (e.g., impulsivity, substance abuse, chaotic lifestyle) central to BPD are known to disrupt sleep patterns (Fleischer, Schäfer, Coogan, Häßler, & Thome, 2012; Winsper & Tang, 2014).

4.1.5. Clinical and research implications

Results from this review provide clues regarding the biopsychosocial basis of BPD. It appears that BPD may have an independent association with a range of sleep problems (many of them

objectively measured) not accounted for by concomitant depression (or other psychiatric disorders). This conclusion is supported by converging evidence from clinical studies utilising BPD patients with and without comorbid Axis I disorders (e.g., Battaglia et al., 1993; De La Fuente et al., 2001), and larger studies controlling for the effects of depression symptoms and substance dependence on BPD-sleep associations (Harty et al., 2010; Oltmanns et al., 2014; Selby, 2013).

BPD and depressed patients appear to share similar objective sleep disturbances, which could indicate an overlap in biological processes. Harty et al. (2010) suggest that sleep problems in BPD may represent a more general impairment in self-regulation, and that sleep and impaired psychological self-regulation in BPD are underpinned by a common biological substrate. It has been proposed that sleep disturbances and emotional dysregulation are transdiagnostic processes which are aetiological and maintaining factors for a range of psychopathologies (Fairholme et al., 2013). As hypothesised in the "emotional cascades" theory of BPD, daytime dysregulation may spill over into the night-time increasing risk of sleep disturbance, which in turn may further contribute to dysregulation in a positive feedback loop (Selby et al., 2013). Increased amygdala responsiveness to dream stimuli (Nielsen & Stenstrom, 2005) leading to hyper-activity of the limbic system (Yoo, Gujar, Hu, Jolesz, & Walker, 2007) may represent one potential physiological mechanism contributing to this cycle.

More prospective cohort studies from childhood onwards are now needed to ascertain whether sleep problems are a marker, cause, or consequence of BPD, and to elucidate the aetiological pathways involving sleep, dysregulation and BPD. Studies incorporating a range of psychopathological outcomes (e.g., BPD, depression, psychosis, bipolar disorder, anxiety) simultaneously may help shed further light on the specific nature of sleep problems in BPD across development (Harvey, 2015). Such studies should be complemented by experimental

and micro-longitudinal (McCall et al., 2010) designs to investigate the proximal impact of sleep disruptions on BPD symptoms at the biological (Yoo et al., 2007) and phenotypical (Anderson & Platten, 2011) level. It should be highlighted that the BPD diagnosis (in the categorical form used in many of the included studies) comprises a heterogeneous group of individuals. Indeed, the requirement for five out of nine symptoms means that there are numerous ways to meet the BPD diagnosis (Linehan, Cochran, & Kehrer, 2001). Hafizi (2013) suggests that specific types of sleep problem may be associated with specific BPD symptoms (e.g., nightmare disorder with self-harm and suicide, short sleep length with affective instability). Thus, to further our understanding of sleep disturbance in this complex disorder, future studies may examine associations between specific sleep problems and individual symptoms (or symptom clusters) of BPD.

Sleep problems may not only worsen symptom course in BPD (Selby et al., 2013) but also heighten risk of suicide (Balestrieri et al., 2006; Winsper & Tang, 2014) and self-harm (Semiz et al., 2008) in this vulnerable population. Hence, mental health professionals should routinely assess the degree to which patients with BPD are experiencing sleep difficulties. Sedative-hypnotic regimens should be approached with caution (National Institute for Clinical Excellence, 2009), as patients with BPD may be likely to use medications in excess, thus exacerbating suicide risk (Plante et al., 2013a; Winsper & Tang, 2014).

This review highlights several potential treatment approaches for sleep problems in BPD. Cognitive Behaviour Therapy for insomnia (CBT-I) is an effective treatment (Harvey, 2015), which may help reduce maladaptive sleep related cognitions and potentially aid recovery in BPD (Plante et al., 2013a). Considering the high incidence and potential aetiological role of nightmares in BPD (Selby et al., 2013), imagery rescripting and rehearsal (IRR) and imaginal confrontation with nightmare contents (ICNC) may be other evidence-based approaches for some patients (Hansen, Höfling, Kröner-Borowik, Stangier, & Steil,

2013). Timed light exposure, planned, regular sleep schedules, and interpersonal and social rhythms therapy (Harvey, 2015) are further options for disturbed circadian rhythms in BPD (Bromundt et al., 2013; Dagan et al., 1998; Huỳnh et al., 2015).

In view of the centrality of dysregulation to BPD and sleep disturbance (Marwaha, Balbuena, Winsper, & Bowen, 2015), an integrated treatment approach focusing on promoting sleep hygiene (Huỳnh et al., 2015; NICE, 2009) and reducing daytime rumination (Selby et al., 2013) and emotional dysregulation (Blum et al., 2008) is warranted.

Table 1. List of acronyms and full name used throughout the review	
Acronyms	**Full name**
BPD	Borderline Personality Disorder
CBT-I	Cognitive Behaviour Therapy for Insomnia
CC/s	Clinical Control/s
CDSR	Cochrane Database of Systematic Reviews
CRD	Centre for Reviews and Dissemination
DSPS	Delayed Sleep Phase Syndrome
EEG	Electroencephalogram
HC/s	Healthy Control/s
ICNC	Imaginal Confrontation with Nightmare Contents
IRR	Imagery Rescripting and Rehearsal
MOOSE	Meta-analysis of Observational Studies in Epidemiology
NWAKE	Number of Awakenings
NOS	Newcastle Ottawa Scale
PRISMA	Preferred Reporting Items for Systematic Review and Meta-analysis
PTSD	Post-Traumatic Stress Disorder
PSG	Polysomnography
QA	Quality Assessment
REM	Rapid Eye Movement
SE	Sleep Efficiency
SMD	Standardised Mean Difference
SOL	Sleep Onset Latency
SWS	Slow Wave Sleep
TST	Total Sleep Time
WASO	Wake After Sleep Onset

Table 2. Details and Main Results of Studies Reporting on the Sleep Characteristics of Individuals with BPD diagnosis or symptoms

Author/ Synthesis (Design)	Year/ Country	% female	Sample (N, controls)	Sample frame	Age (SD) In years	Sleep assessment /medication	Co-morbidity	BPD assessment tool	Main results
(Akiskal et al.) MA	1985 USA	50% 43% 43% 38%	24 BPD 30 depression 14 HCs 16 other PDs	Outpatients (Sleep disorders centre)	36 (11) 46 (12) 36 (16) 45 (13)	Polysomnography/ No psychoactive drugs for 2 weeks	*Excluded:* Definite or probable depressive episodes during the preceding year; narcolepsy and sleep apnea	DSM-III criteria for BPD	BPD patients had significantly shorter REM latency than healthy and PD controls. They did not significantly differ in REM latency from depressed controls.
(Asaad, Okasha, & Okasha) MA	2002 Egypt	60% 60% 60%	20 BPD 20 depression 20 healthy controls	Outpatients	27.04 (7.53) 27.83 (7.65) 27.8 (4.62)	Polysomnography; Questionnaire/ No psychotropic drugs for 2 weeks	*Excluded:* Past or current affective disorder or other psychiatric disorder; sleep apnea	ICD-10 "emotionally unstable PD"	BPD patients significantly differed from HCs on SOL (longer), SE (lower), SWS (less), REM % (greater), REM latency (shorter) & REM density (higher). BPD patients significantly differed from depressed controls on SOL (shorter), SE (higher), NWAKE (less), REM latency (longer), REM density (lower).
(Bastien et al.) MA	2008 Canada	100% 67% 73%	12 BPD 15 good sleepers 15 psycho* insomnia 15 para* insomnia	Outpatients	33.3 (10.7) 34.1 (9.9) 36.6 (7,1)	Polysomnography; Sleep diary; ISI/ No psychotropic drugs or sleep promoting medication	*Excluded:* Axis I psychiatric disorder or alcohol/drug abuse during past year; other sleep disorder, e.g., apnea	DSM-IV criteria for BPD DIB-R (≥7)	BPD patients had significantly longer SOL, less TST, and lower SE than good sleepers (GS). BPD patients had more stage 4 sleep than controls with insomnia. Subjective reports of sleep difficulties did not differ between patients with BPD and GS. Controls with insomnia had significantly poorer subjective sleep than BPD patients and GS.
(Battaglia et al.) MA	1993 Italy	60% 60%	10 BPD 10 healthy controls	Inpatients	25.4 (4.5) 24.8 (3.5)	Polysomnography/ 2 weeks without medication	*Excluded:* Past or current depression, bipolar disorder, cyclothymia, dysthymia, schizophrenia	DSM-III criteria for BPD	BPD patients had significantly longer SOL, more WASO and number of awakenings, lower SE, and shorter REM latency than HCs.
(Bell, Lycaki, Jones, Kelwala, & Sitaram) MA	1983 USA	67% 56%	8 primary BPD 11 primary depression	Inpatients	29.7 (3.9) 36.5 (13.2)	Polysomnography/ 2 weeks without medication	All had co-morbid depression	DSM-III criteria for BPD	There were no significant differences in SOL, SE, REM % or latency, TST or WASO between BPD and depressed only groups.
(Benson et al.) MA	1990 USA	0% 0%	10 BPD (veterans) 15 HCs	Inpatients	32.7 (7.6) 30.5 (7.6)	Polysomnography/ 2 weeks medication free	No history of, or co-morbid, major depressive disorder	DSM-III criteria for BPD	BPD patients had significantly less TST, more stage 1 sleep and less stage 4 sleep than HCs. The groups did not differ in REM latency.
(Bromundt et al.) N	2012 Switzerland	100%	14 BPD 10 healthy controls	Outpatients	30.1 (6.0) 25.7 (4.8)	Wrist actimetry; PSQI; MEQ; SPAQ/ 10/14 BPD patients on stable medication	Not reported (though BPD group had significantly higher depression and anxiety symptoms); no sleep apnea	DSM-IV criteria for BPD BPI (>8)	BPD patients exhibited a wider range in Relative Amplitude (RA) than HCs (p=0.019). BPD patients with high RA had regular sleep-wake cycles, while those with low RA group had irregular rhythms and more BPD symptoms. Morning LT significantly improved daytime alertness (F [1, 12] = 9.33, p = 0.010), and attenuated atypical depression scores (*t* [13] = 2.55, p = 0.024) in patients with BPD.
(Claridge et al.) N	1998 Canada	100%	60 women (study1) 66 women (study2) 203 women (study3)	Student population	20.4 (range=18-30 years) 21.2	Nightmare Distress Scale (13 item self-report); Dream and Nightmare Enquiry/ not reported	Not reported	STB	Study 1: BPD symptoms were significantly correlated with ND (*r*=0.42, p< 0.001) and remained a significant predictor of nightmares following control for sexual abuse and neglect. Study 2: BPD symptoms were significantly correlated with adult (*r* =0.31, *p*< 0.01), but not childhood (*r* =0.07) nightmare content. Study 3: ND was significantly positively correlated with BPD symptoms (*r*=0.49, *p*< 0.001) following control for abuse and neglect.
(Dagan et al.) N	1998 Israel	48%	52 adolescents mixed diagnoses 11 BPD	Inpatients	17.4 (2)	Sleep wake habits Interview/ all on medication	4 of the 5 BPD patients with DSPS had no co-morbidities	MCMI PDQ-R	50% of adolescent patients with Delayed Sleep Phase Syndrome had BPD (Odds Ratio=7.67; 95% CI=1.70, 34.48).

Synthesis: MA=meta-analysis; N=narrative; HC=Healthy control; *psycho= psychophysiological; *para= paradoxical; DIB-R=Revised Diagnostic Interview for Borderlines; STB=Borderline Personality Scale; MCMI=Millon Clinical Multiaxial Inventory; PDQ-R=Personality Diagnostic Questionnaire Revised

Study	Year Country	%	Sample	Setting	Age M (SD)	Measure/ Medication	Comorbidity/Exclusion	Diagnostic criteria	Findings
(De la Fuente, Bobes, Vizuete, & Mendlewicz) **MA**	2001 France	70% 75% 70%	20 BPD 20 depression 20 HCs	Inpatients	32.4 (6.9) 38.5 (7.6) 35.5 (5.8)	Polysomnography/ 10 days medication free	*Excluded:* Other Axis I disorders	DSM-III criteria for BPD DIB-R (≥7)	BPD patients had significantly less TST, longer SOL, and a greater % of wakefulness than HCs. BPD patients had a longer duration of REM sleep, less stage 3, stage 4 and SWS than both control groups. BPD patients had more stage 2 sleep than depressed controls.
(De la Fuente et al.)* **N**	2004 France	70% 70% 75% 75%	20 BPD 20 RBD 20 MD 20 HCs	Inpatients	32.4 (6.9) 36.6 (7.6) 35.9 (3.4) 35.5 (5.8)	Polysomnography/ 10 days medication free	*Excluded:* Other Axis I disorders	DSM-III criteria for BPD DIB-R (≥7)	Patients with BPD experienced significantly less stage 3 sleep and SWS, and significantly longer REM duration than patients with RBD. They did not significantly differ on SOL; TST; stage 1 or 2 sleep; or REM latency.
(Harty et al.) **N**	2010 USA	30%	513 jail inmates	Prisoners	32 (10)	5 item index self-report index/ not reported	Not reported (though analysis controlled for depression symptoms and substance dependence)	PAI-BOR scale	BPD symptoms were significantly correlated with self-reported sleep problems (r=.43). This significant correlation held following control for cognitive, affective and physiological depression (r=.20), and substance dependence (r=.39).
(Hornung et al.) **MA**	2008 Germany	100% 100%	15 BPD 15 HCs	Inpatients	26.1 (6.1) 25.6 (6.8)	Portable recording device; sleep questionnaire/ no medication 3 days	3 of the BPD patients had co-morbid depression	DSM-IV criteria for BPD	BPD patients reported significantly poorer sleep quality and less restorative value of sleep in comparison to HCs. They did not significantly differ in objective measures of SOL, TST, WaSO, sleep stage or REM latency.
(Huỳnh et al.) **MA/N**	2015 Canada	83% 67% 65%	18 BPD 6 BD 20 HCs	Outpatients	16 (1.1) 16.7 (1.0) 14.7 (1.0)	Actigraph/ most patients on psychotropic medication	*Excluded:* Euthymic (i.e., current depression symptoms), mania, psychotic features, bipolar	DSM-IV criteria for BPD DIB-R (≥8)	On school/work days, adolescents with BPD spent a significantly lower % of time asleep than bipolar controls (p=0.04). Time awake in bed did not significantly differ between BPD & HCs. On schedule-free days, youths with BPD and bipolar disorder spent more time in bed than HCs (*ns*). Adolescents with BPD demonstrated more inter-daily total sleep time variability than bipolar and HC groups.
(Lahmeyer et al.) **MA**	1987 USA	53% 50%	21 BPD 20 depression	Outpatients	29.0 (3.4) 35.2 (4.8)	Polysomnography/ 14 days without medication	*Excluded:* Major affective disorders and schizophrenia	DSM-III criteria DIB (≥7)	BPD patients had significantly less stage 1 sleep than depressed controls and significantly greater SE. The 2 groups did not differ on TST, SOL or REM (&, density and latency) indices.
(Lereya et al.) **N**	2016 UK	51.4%	6,050	Community population of young adolescents	11.8	Mother reported/ not reported	Not reported, but controlled for emotional and behavioural dysregulation within the analysis	UK-CI-BPD	Chronic nightmares significantly predicted BPD (OR=1.67; 95% CI=1.18, 2.38). This association was mediated by daytime emotional and behavioural dysregulation (β =0.016, *p*<0.001). Associations between early emotional temperament (β=0.001, *p*=0.018); abuse (β=0.015, *p*=0.018); maladaptive parenting (β=0.002, *p*=0.021), and subsequent BPD were all mediated by nightmares.
(Lindberg et al.) **MA**	2003 Finland	0% 0% 0%	10 ASPD 6 ASPD with BPD 11 HCs	Prisoners (charged with violent crimes)	28.1 (3.40) 34.8 (4.45) 32.5 (3.44)	Polysomnography/ 2 weeks medication free	*Excluded:* Axis I disorder (other than drug/alcohol abuse), other personality disorders	DSM-IV criteria for BPD	Groups did not differ on TST, SOL, SWS, stage 1 sleep or REM measures. The BPD group had significantly more awakenings and significantly lower SE than ASPD and HCs. BPD and ASPD groups had significantly more stage 4 sleep than HCs.
(Lloyd et al.) **N**	1983 USA	66%	27 BPD; 178 mixed disorders	Outpatients	20-59 years	MMPI-168/ not reported	Not reported	DSM-III criteria	Outpatients with BPD were significantly more likely to report nightmares every few nights than controls (p< .01).
(Mahmood, Tripodi, Vaughn, Bender, & Schwartz) **N**	2012 USA	100%	475 BPD 330 controls	Prisoners	33.5 (8.5)	One item: "have trouble sleeping in prison"/ not reported	Not reported though BPD was correlated with anxiety and depression	SCID-II	Female prisoners with BPD were more likely to experience self-reported sleep problems following control for demographic variables; though the difference did not quite reach statistical significance: OR=1.33; 95% CI = 0.90, 1.94.
(McNamara et al.) **MA**	1984 USA	100% 70% 100%	10 BPD 10 depression 10 HCs	Inpatients (2 outpatients)	28.1 (3.8) 27.4 (5.1) 27.8 (4.5)	Polysomnography/ 2 weeks medication free	BPD patients had variety of Axis I co-morbidities (6 had major depression)	DIB (≥7)	The BPD group significantly differed from HCs on TST (less), SE (less), and REM latency (shorter) and REM density (greater).
(Oltmanns et al.) **N**	2014 USA	57%	633 older adults	Community sample	62.3 (2.8)	ISI (self-report questionnaire)/ not reported	Not reported, but controlled for depression within the analysis	SIDP-IV MAPP	BPD pathology was significantly predictive of insomnia symptoms following control for major depression, body mass index, race, gender, and other personality disorders: β=0.23, p<0.001.

*Overlapping sample with De La Fuente 2001; RBD=Recurrent Brief Depression; PAI-BOR=Personality Assessment Inventory;*Sample from McNamara (1984); MMPI-168=Minnesota Multiphasic Personality Inventory; ISI=Insomnia Severity Index; SF-A=Schlaffragebogen A; DBAS-1 Dysfunctional Beliefs About Sleep; UK-CI-BPD=UK Childhood Interview for DSM-IV BPD; SCID-II=Structured Clinical Interview for DSM-IV; SIDP-IV=Structured Interview for DSM–IV PDs; MAPP=Multi-Source Assessment of Personality Pathology

Study	Year Country	%	Sample	Setting	Age	Measure/medication	Exclusions/comorbidity	Diagnostic instrument	Findings
(Philipsen et al.) **MA**	2005 Germany	100% 100%	20 BPD 20 HCs	Outpatients	28.6 (6.9) 28.6 (7.1)	Polysomnography; SF-A, PSQI/ 2 weeks medication free	*Excluded:* Any other psychiatric disorder within six months of the sleep assessment; sleep apnea	DSM-IV criteria for BPD, SCID-II	BPD patients experienced significantly less stage 2-sleep than HCs. They did not differ on other objective sleep measures (SOL; TST; SE; SWS; REM). Self-reported sleep quality was significantly worse in BPD patients.
(Plante et al.) **N**	2013a	83% 80%	105 recovered BPD; 118 non-recovered BPD	Outpatients	41.2 (5.4) 44.2 (6.0)	DBAS-16/ Some using sleep medications	Non-recovered group had range of co-morbidities including major depression, panic disorder and PTSD	SCID-I DIB-R DIPD-R	Following adjustment for age, sex, depression, anxiety and primary sleep disorders, non-recovered BPD patients reported significantly more maladaptive sleep-related cognitions than the recovered group: p=0.0009.
(Plante et al.) **MA**	2013b	83% 80%	105 recovered BPD; 118 non-recovered BPD	Outpatients	41.2 (5.4) 44.2 (6.0)	PSQI/ Some using sleep medications	Non-recovered group had range of co-morbidities including major depression, panic disorder and PTSD	SCID-I DIB-R DIPD-R	Non-recovered BPD patients had significantly worse sleep quality, longer SOL, increased odds of using sleep medication, and more daytime dysfunction than recovered BPD patients.
(Reynolds et al.) **MA**	1985 USA	100% 70% 70% 100%	10 BPD (retrospective)* 10 BPD (prospective) 10 depression 10 HCs	Inpatients	28.1 (3.8) 25.4 (6.4) 27.4 (5.1) 27.8 (4.5)	Polysomnography/ 2 weeks medication free	All BPD had co-morbid affective disorder	DIB (≥7)	The BPD group (prospective) had significantly less TST, significantly longer SOL and significantly lower SE than HCs. They did not significantly differ from depressed patients on sleep assessments.
(Sansone et al.) **N**	2010 USA	74.6%	76 outpatients	Outpatients	41.6 (13.8)	PSQI/	*Excluded:* Psychotic illness	PDQ-4 SHI	Poor sleep quality was significantly associated with BPD symptoms (p< 0.05)
(Schredl et al.) **MA**	2012 Germany	100% 100%	27 BPD (18 no PTSD) 20 HCs	Outpatients	29.3 (7.7) 24.3 (3.8)	Polysomnography; SF-B, LISST/no medication for 3 months	*Excluded:* Lifetime schizophrenia or bipolar I disorder, current drug/alcohol abuse or major depression	IPDE	BPD patients experienced significantly more awakenings and time awake, shorter REM latency, and greater REM density than HCs. BPD patients reported much lower sleep quality and feelings of being refreshed than HCs. BPD patients also reported significantly more parasomnias and nightmares than HCs.
(Selby et al.) **MA/N**	2013a USA	66%	16 BPD patients 31"dysregulated" controls	Community and undergraduate	Not reported	Questionnaire/ Some participants taking psychotropic medication	Not reported though associations controlled for MDD diagnosis	SCID-II	BPD patients experienced more frequent nightmares. BPD diagnosis interacted with baseline trait rumination to prospectively predict number of nightmares. Daytime emotional dysregulation prospectively predicted nightmares. These associations remained following adjustment for sleep quality, depression and Post-Traumatic Stress Disorder.
(Selby) **N**	2013 USA	Not reported	5,692 adults	Community	Not reported	Questionnaires/ not reported	Not reported though associations adjusted for Axis I disorders	IPDE	BPD was significantly associated with chronic sleep disturbances (i.e., difficulty initiating sleep, difficulty maintaining sleep, and waking earlier than desired). BPD symptoms interacted with sleep problems to predict social, emotional, cognitive, and self-care impairment.
(Semiz et al.) **MA**	2008 Turkey	45% 43%	88 BPD 100 HCs	Inpatients	21.7 (3.6) 22.3 (3.9)	VDAS; PSQI/ No medication for 4 weeks	*Excluded:* Psychotic or mood disorder or PTSD in the past year	SCID	BPD patients experienced significantly more nightmares and dream anxiety, and significantly poorer sleep quality and daytime functioning than controls. BPD patients with nightmare disorder experienced greater psychopathology (i.e., self-harm, substance abuse, dissociation) than those without nightmare disorder.
(Simor et al.) **MA**	2010 Hungary	83% 83%	23 BPD 23 HCs	Outpatients	NR	VDAS; DQQ/ 6/23 BPD patients on antidepressants	*Excluded:* Other Axis II disorder or PTSD	SCID-II	BPD patients experienced significantly more dream disturbances, in terms of nightmares; bad dreams; night-terror symptoms, and dream anxiety. Dream disturbances were positively correlated with neuroticism.
(Taherifard et al.) **MA**	2015 Iran	0%	50 BPD 50 ASPD 50 HCs	Outpatients	28.5 (4.33) 29.72 (5.07) 25.88 (3.97)	PSQI/not reported	Not reported	IDI for BPD	BPD patients had significantly lower sleep quality than controls.

*Sample from the McNamara (1984) study thus results on prospective sample used

Table 3. Meta-analysis results comparing the objective sleep characteristics of BPD patients to healthy and clinical controls

Sleep characteristic	Versus healthy controls						Versus clinical controls					
	Pooled SMD	Lower CI	Higher CI	Hetero-geneity	P value for group difference	Publication bias[b]	Pooled SMD	Lower CI	Higher CI	Hetero-geneity	P value for group difference	Publication bias[b]
Sleep onset latency (Sensitivity analysis[a])	**0.626** (0.653)	**0.229** (0.214)	**1.023** (1.091)	67.2%* (69.8%)	**0.002** (0.004)	No	-0.072 (-0.107)	-0.420 (-0.480)	0.277 (0.266)	40.6% (44.7%)	0.687 (0.574)	No
Total sleep time	**-0.556** (-0.674)	**-1.090** (-1.227)	**-0.022** (-0.122)	74.0%* (71.2%)	**0.041** (0.017)	Yes	0.279 (0.326)	-0.104 (-0.091)	0.662 (0.742)	44.1% (48.8%)	0.153 (0.125)	No
Wake after sleep onset	**0.653** (N/A)	**0.073** (N/A)	**1.233** (N/A)	54.0% (N/A)	**0.027** (N/A)	Some indication	-0.389 (N/A)	-1.005 (N/A)	0.226 (N/A)	46.3% (N/A)	0.215 (N/A)	No
Number of awakenings	**0.874** (N/A)	**0.132** (N/A)	**1.616** (N/A)	80.5%* (N/A)	**0.021** (N/A)	No	0.762 (N/A)	-1.361 (N/A)	2.885 (N/A)	93.2%* (N/A)	0.482 (N/A)	No
Sleep efficiency	**-1.015** (-1.169)	**-1.555** (-1.660)	**-0.476** (-0.678)	74.0%* (62.5%)	**<0.001** (<0.001)	Slight indication	0.179 (0.250)	-0.623 (-0.627)	0.981 (1.127)	84.5%* (85.7%)	0.662 (0.576)	Slight indication
REM latency	**-0.812** (N/A)	**-1.154** (N/A)	**-0.470** (N/A)	55.0%* (N/A)	**<0.001** (N/A)	Some indication	0.039 (N/A)	-0.409 (N/A)	0.488 (N/A)	69.3%* (N/A)	0.864 (N/A)	No
REM %	0.219 (N/A)	-0.122 (N/A)	0.560 (N/A)	46.3% (N/A)	0.207 (N/A)	No	0.078 (N/A)	-0.269 (N/A)	0.426 (N/A)	36% (N/A)	0.659 (N/A)	No
REM density	**0.741** (N/A)	**0.263** (N/A)	**1.218** (N/A)	56.3%* (N/A)	**0.002** (N/A)	No	-0.206 (N/A)	-1.540 (N/A)	1.129 (N/A)	90.8%* (N/A)	0.763 (N/A)	No
Stage 1 sleep	0.297 (N/A)	-0.010 (N/A)	0.604 (N/A)	38.0% (N/A)	0.058 (N/A)	No	-0.303 (N/A)	-0.655 (N/A)	0.050 (N/A)	37.9% (N/A)	0.092 (N/A)	No
Stage 2 sleep	-0.181 (N/A)	-0.569 (N/A)	0.208 (N/A)	60.7%* (N/A)	0.361 (N/A)	No	0.299 (N/A)	-0.095 (N/A)	0.693 (N/A)	49.5% (N/A)	0.137 (N/A)	Slight indication
Slow wave sleep	**-0.529** (N/A)	**-0.904** (N/A)	**-0.154** (N/A)	47.8% (N/A)	**0.006** (N/A)	No	-0.418 (N/A)	-1.189 (N/A)	0.353 (N/A)	77.4%* (N/A)	0.288 (N/A)	No

* Significant heterogeneity across studies; [a] Sensitivity analysis excluding studies with patients on psychotropic medication at the time of testing; [b] publication bias estimated with funnel plots; NA= All studies excluded patients on psychotropic medication; SMD=Standardised Mean Difference; CI=Confidence Interval

Table 4. Sub-group analysis results comparing the objective sleep characteristics of BPD patients to healthy and clinical controls

Subgroup	Sleep characteristic (SMD and 95% Confidence Intervals reported)						
Depression status	**TST**	**SOL**	**NWAKE**	**SE**	**REM latency**	**REM density**	**Stage 2 sleep**
No past (or current) depression	**-1.31** (**-2.19, -0.42**)	**1.63** (**0.56, 2.69**)	0.28 (- 0.36, 0.92)	**-1.59** (**-2.18, -1.00**)	**-1.44** (**-2.44, -0.44**)	**0.74** (**0.04, 1.44**)	0.39 (-0.58, 1.36)
No current depression	-0.14 (-0.67, 0.38)	0.24 (-0.09, 0.57)	**1.28** (**0.14, 2.41**)	-0.66 (-1.38, 0.06)	**-0.58** (**-0.89, - 0.26**)	0.48 (-0.69, 1.66)	**-0.47** (**-0.92, -0.03**)
Co-morbid depression	**-1.68** (**-2.41, -0.94**)	**1.11** (**0.44, 1.78**)	N/A	**-1.32** (**-2.01, -0.63**)	**-0.77** (**-1.41, -0.12**)	**1.35** (**0.37, 2.33**)	-0.17 (-0.80, 0.45)
Sample type							
Inpatients	**-0.99** (**-1.79, -0.20**)	**0.78** (**0.33, 1.23**)	0.70 (-0.21,1.60)	**-1.48** (**-2.06, -0.90**)	**-0.64** (**-1.10, -0.19**)	0.81 (-0.20, 1.82)	0.23 (-0.29, 0.75)
Outpatients	-0.22 (-0.76, 0.32)	0.59 (-0.19, 1.36)	0.46 (- 0.09, 1.01)	-0.54 (-1.09, 0.01)	**-0.89** (**-1.45, -0.32**)	0.67 (-0.12, 1,45)	**-0.47** (**-0.89, -0.05**)
Prison populations	0.52 (-0.50, 1.53)	-0.09 (-1.09, 0.91)	**4.63** (**2.70, 6.57**)	**-3.12** (**-4.62, -1.63**)	**-1.47** (**-2.59, -0.34**)	N/A	**-1.17** (**-2.27, -0.07**)
Sleep assessment							
Polysomnography	**-0.85** (**-1.36, -0.34**)	**0.73** (**0.22, 1.24**)	N/A	**-1.17** (**-1.66, -0.68**)	**-0.87** (**-1.22, -0.52**)	N\A	N\A
Portable device	0.41 (-0.13, 0.95)	0.19 (- 0.35, 0.73)	N/A	0.30 (-0.39, 0.99)	-0.09 (-0.96, 0.77)	N\A	N/A
Sex							
Mixed	**-0.96** (**-1.91, -0.01**)	**1.09** (**0.58, 1.59**)	0.28 (-0.36, 0.92)	**-1.10** (**-1.93, -0.26**)	**-1.21** (**-1.87, -0.56**)	**0.74** (**0.04, 1.44**)	-0.13 (-0.61, 0.36)
Female	-0.17 (-0.83, 0.50)	0.08 (-0.33, 0.49)	0.62 (-0.05, 1.29)	**-0.53** (**-0.92, -0.14**)	**-0.45** (**-0.78, -0.12**)	0.74 (-0.19, 1.66)	-0.35 (-0.87, 0.17)
Male	-0.41 (-2.20, 1.38)	-0.09 (-1.09, 0.91)	**4.63** (**2.70, 6.57**)	**-3.12** (**-4.62, -1.63**)	-0.90 (-1.84, 0.05)	N/A	-0.05 (-2.18, 2.09)

Significant group difference in bold face; negative figures represent that the BPD group experienced less TST, SE, and REM latency than HCs, positive figures indicate that the BPD group experienced more SOL & REM density

Table 5. Meta-analysis results comparing the self-reported sleep characteristics of BPD patients to controls

	Pooled SMD	Lower CI	Higher CI	Hetero-geneity	P value for group difference	Publication bias[b]
Nightmares (sensitivity analysis [a])	**1.07** (1.32)	**0.43** (0.73)	**1.71** (1.91)	**81.8*** 62.5	**0.001** <0.001	No
Sleep quality	**1.99** (2.28)	**0.96** (1.24)	**3.03** (3.32)	**95.8*** (89.0*)	**<0.001** (<0.001)	No
Sleep onset latency	**0.35** (0.32)	**0.10** (-0.19)	**0.60** (0.84)	**7.9** (36.1)	**0.006** (0.209)	No
Total sleep time	-0.35 (-0.60)	-0.76 (-1.23)	0.06 (0.03)	61.7%* 56.5	0.095 0.062	Yes
Sleep efficiency	-0.76 (-1.18)	-1.65 (-1.79)	0.12 (-0.58)	85.8* (23.4)	0.09 (<0.001)	Yes

* Significant heterogeneity across studies; Significant group difference in bold face; [a] Sensitivity analysis excluding studies with patients on psychotropic medication at the time of testing; [b] publication bias estimated with funnel plots; SMD=Standardised Mean Difference; CI=Confidence Interval

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