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Paradoxical insomnia and subjective-objective sleep discrepancy: A review

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RUNNING HEAD: Paradoxical Insomnia

Author declaration:

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Summary

Paradoxical insomnia is characterized by discrepancy between subjective and objective assessments of sleep and is challenging to diagnosis and treat. Typically, polysomnographic (PSG) findings show significantly longer total sleep time than patients' report of sleep, and the difference between subjective and PSG sleep is greater than that seen in other insomnia subtypes. Subjective-objective sleep discrepancy may also present in different clinical pictures, as marked discrepancies between patients' perception of sleep and objective findings are common in a variety of medical, sleep and psychiatric disorders. However, there is a paucity of literature about the etiology and treatment of sleep discrepancy and paradoxical insomnia. Therefore, the underlying neurophysiological mechanisms of sleep discrepancy and paradoxical insomnia should be further investigated. Additionally, well-controlled clinical trials are needed to establish an evidence based intervention for treatment.

Key words: paradoxical insomnia, review, sleep state misperception, sleep discrepancy

Abbreviations	
CAP: Cyclic alternating pattern CBT: Cognitive behavioral therapy ECT: Electroconvulsive therapy EEG: Electroencephalogram ICSD-2: International Classification of Sleep Disorders, 2nd edition ICSD-3: International Classification of Sleep Disorders, 3rd edition MDD: Major depressive disorder	OSA: Obstructive sleep apnea PSG: Polysomnography PTSD: Post traumatic stress disorder REM: Rapid eye movement

Introduction

Insomnia is a common sleep disorder which affects 10-25% of adults in most countries (1). According to the International Classification of Sleep Disorders, 3rd edition (ICSD-3), insomnia is characterized by subjective complaints of difficulty in initiating and/or sustaining sleep (2) and associated with daytime symptoms such as fatigue, depressed mood, lack of interest in social activity, impairment in work-related performance, and poor quality of life at least three times per week for at least three months (3-6). Insomnia is often complicated by an increased risk of other medical and psychiatric disorders including heart failure, mood disorders, and anxiety and depressive disorders (7-10). There are several guidelines for diagnosis and treatment of insomnia (11-14).

Sleep discrepancy, or the difference between subjective and objective sleep parameters, is commonly experienced in insomnia and often presents with underestimation of total sleep time and **overestimation** of sleep onset latency and wakening after sleep onset. An extreme case of sleep discrepancy is called paradoxical insomnia (15). The prevalence of paradoxical insomnia ranges between 9.2-50% of patients with insomnia (16-18). Patients with paradoxical insomnia do not display the level of daytime sleepiness experienced with sleep deprivation (17), and contrary to complaints of little to no sleep over long periods of time, polysomnographic (PSG) findings show **patients** with paradoxical insomnia have near-normal sleep patterns (18). However, the International Classification of Sleep Disorders, 2nd edition (ICSD-2) emphasized that "physiological abnormalities may exist in the sleep tracing that are too subtle to be detected by recording methods currently in use" (19). In addition to subjective **report of** insomnia **despite** normal **objective measures of sleep**, the diagnosis of paradoxical insomnia requires that the

symptoms are not explained by another medical or mental disorder, and the distress about insomnia is more than what is produced by other sleep disorders (2).

Due to the discrepancy between significant subjective complaints of insomnia and objective findings of sufficient sleep, paradoxical insomnia is among one of the most challenging diagnoses to treat in clinical practice. Additionally, patients become distressed and anxious due to perceived inadequate sleep, which may eventually result in objective sleep disturbance (20, 21). Finally, because insomnia has a high comorbidity with other medical and psychiatric disorders (7, 8) and since paradoxical insomnia is one of most prevalent insomnia subtypes, it is important to gain a better understanding of paradoxical insomnia. However, there is little research addressing this disorder. Therefore, we aim to provide a comprehensive review of paradoxical insomnia.

Historical account

The International Classification of Sleep Disorders-I-Revised (1997) adopted the diagnosis of sleep state misperception **to describe** patients who complain of poor sleep despite normal sleep quality and duration indices in PSG (22). Other terms such as “pseudo insomnia” or “insomnia without objective findings” have also been used to describe the condition. In 2005, the ICSD-2 changed the term to “paradoxical insomnia” from sleep state misperception and divided chronic insomnia into 11 subtypes, among the most frequent being paradoxical insomnia, psychophysiological insomnia and idiopathic insomnia (19). In addition to the incongruence between subjective and objective sleep assessment, ICSD-2 emphasized the presence of significant impairment in daily function of patients with paradoxical insomnia. Due to a lack of evidence to support separate groups, both the Diagnostic and Statistical Manual of Mental

Disorders, Fifth Edition and the ICSD-3 removed the distinction of insomnia subtypes, (2, 23) emphasizing the comorbid quality of insomnia and highlighting its need for independent clinical consideration. By this definition, insomnia is not seen as secondary to other medical and psychiatric disorders but as a comorbid disorder warranting targeted consideration (23).

Identifying paradoxical insomnia as a subtype of insomnia has been somewhat controversial for two main reasons. First, sleep discrepancy is found in both healthy people and patients with insomnia, and paradoxical insomnia is considered by some to be an exaggeration of sleep discrepancy without an identified cut-off (24). However, paradoxical insomnia appears to have a distinct pathophysiology, suggesting further research is warranted to examine the etiology and treatment of this subtype (16). Second, the heterogeneity of research findings has hampered efforts to propose specific objective criterion for the discrepancy between subjective sleep **and** PSG **for the** diagnosis of this subtype of insomnia (20). **Newly proposed methods in** quantifying the **amount** of discrepancy in sleep estimation **could aid in gaining consensus** for paradoxical insomnia as a subtype of insomnia. For example, Manconi et al. (2010) introduced the misperception index to estimate the differences between patients with insomnia and those with normal sleep (15), and they **stipulated a** high misperception index (≥0.9) **as indicating** paradoxical insomnia. PSG criteria have also been set for paradoxical insomnia including a difference **from self-report** of 60 min or more for total sleep time or a difference of at least 15% for sleep efficiency (25), and Turcotte et al. suggested that an individual could be diagnosed with paradoxical insomnia if they meet **both of these** criteria on two of four nights (26). However, it may require up to a few weeks to establish a reliable estimate of sleep patterns, and it is unclear if two of four nights would suggest a reliable pattern. For example, while sleep discrepancy has been found to be consistent in younger adults, Kay et al. found that night-to-night variability in

sleep discrepancy is common in older adults, and the greatest night-to-night variability is found among older adults with sleep complaints (27). This suggests it is necessary to obtain a larger sample of average sleep and assess night-to-night variability in this population. While it seems the combination of the standard criteria by Edinger et al. (25) and Turcotte et al (26) provide a foundation for the research diagnosis of paradoxical insomnia in a sleep laboratory, there is still a need for more investigation regarding application of these diagnostic criteria in clinical settings.

Method

We searched the PubMed database using the keywords “paradoxical insomnia,” “sleep state misperception” “subjective insomnia, and “subjective-objective sleep discrepancy.” We extracted 40 articles published between 1990 (the decade of sleep medicine expansion and PSG acceptance) (28) and 2016 (the year the review was conducted) in peer-reviewed journals.

Results

Clinical features

As stated above, the common clinical feature for paradoxical insomnia is self-report of insufficient sleep quality or quantity with little objective evidence of short sleep from PSG or actigraphy (2). Patients usually complain of sleeping only a few hours (i.e., 2-4) or of not sleeping at all, and they rarely report having an adequate night of sleep (15). They frequently indicate heightened awareness of environmental stimuli and **mental activity** while trying to sleep (17). However, they are usually obtaining greater sleep than they recount and do not experience the same level of impairment associated with sleep deprivation (2). It is important to note that special considerations may need to be taken for certain populations. Although comparing subjective and objective findings of sleep is an effective way of diagnosing

paradoxical insomnia, McCall and Edinger (1992) suggest that neither PSG nor actigraphy is required to diagnose paradoxical insomnia. They reported two cases of extreme paradoxical insomnia and concluded that complaints of total subjective insomnia were physiologically impossible. **In such cases** a diagnosis of paradoxical insomnia would be warranted through clinical interview alone if the clinical complaint is extreme (29).

In addition to underestimating sleep duration and quality, Trajanovic and colleagues suggested the concept of positive sleep discrepancy, in which patients overestimate their sleep duration but experience daytime sleepiness and fatigue (30). The authors proposed sleep as a spectrum with negative and positive sleep discrepancy at each end. Reynolds and colleagues noted that many people with primary insomnia report greater difficulty in sleep latency, sleep duration, and sleep efficiency than is measured with PSG. Therefore, they hypothesized paradoxical insomnia may simply lie at the extreme end of the primary insomnia spectrum (31).

It has also been proposed that paradoxical insomnia could **be so extreme to be of** delusional **proportions**. Khazaie and colleagues described a 60-year-old woman with paradoxical insomnia who was not responsive to electroconvulsive therapy (ECT) and other benzodiazepine drugs but was finally successfully treated with olanzapine 2.5 mg twice per day**. They concluded that there may be a subtype of paradoxical insomnia in which patients present with delusional features that can be treated with antipsychotic agents** (32). Another study using one night of PSG in adults with primary insomnia in Korea reported that the prevalence of paradoxical insomnia was 26.4%, and they were younger on average than patients with insomnia who did not meet criteria for paradoxical insomnia. They also found no significant difference between individuals with paradoxical insomnia and other insomnia subtypes with regard to gender, body

mass index, disease, and alcohol use (33). Due to the controversy related to distinguishing paradoxical insomnia as a distinct subtype of insomnia, future research is needed to further understand the clinical features of paradoxical insomnia.

Etiology

Although the causal mechanisms of paradoxical insomnia are not fully understood, there are a couple of hypotheses to explain the **phenomenon**. One suggested explanation is the role of personality traits (34,35). Patients with paradoxical insomnia have higher scores on the neuroticism, hypochondriasis, conversion-hysteria and psychasthenia scales of the Minnesota Multiphasic Personality Inventory compared to matched controls (34). However, there is no consensus as to why these personality traits **should be** associated with paradoxical insomnia. Some suggested that these traits may escalate anxiety and arousal (20), which is consistent with the suggestion that sleep discrepancy is due to a "prodromic or transitional state of sleep dysfunction" between normal sleep and objective insomnia (34).

Other studies examining neurophysiological correlates have indicated that paradoxical insomnia is associated with differences in sleep microstructure which objective measures of sleep may be unable to detect (36). Specifically, Harvey and Tang (2012) suggested increased cortical arousal, abnormal neuronal circuitry and frequent brief nighttime awakenings as etiological factors for paradoxical insomnia (20). In another study, Krystal and colleagues compared electroencephalogram (EEG) frequency spectra of paradoxical insomnia and insomnia. Results found reduced delta and increased alpha, sigma and beta non-rapid eye movement EEG activity in patients with paradoxical insomnia compared to both healthy controls and those with objective insomnia, suggesting patients with paradoxical insomnia may experience increased arousal in

sleep (37). Another study assessed EEG event-related potentials to investigate information processing during wake and sleep in insomnia and demonstrated increased cortical arousal in paradoxical insomnia compared to primary insomnia. In addition, they suggested that sleep misperception might occur during rapid eye movement (REM) sleep (26). Another study consistent with this conclusion found that patients with greater REM sleep were more likely to report greater amounts of wakefulness (38). St-Jean et al. (2013) compared REM and non-REM spectral analysis on two consecutive nights among patients with paradoxical insomnia, patients with psychophysiological insomnia and healthy controls (39). The results revealed that patients with paradoxical insomnia have lower activity and slower frequency in REM than controls (39).

In a **PSG** study comparing patients with paradoxical insomnia to controls, **patients** with paradoxical insomnia reported significantly shorter subjective sleep duration and longer subjective sleep latency. **Further, the** cyclic alternating pattern (CAP) rate was greater **for the patients with paradoxical insomnia** in the **time interval** between objective and subjective sleep onset despite no **other** significant differences in PSG between the two groups. Additionally, when consecutive objective awakenings were separated by **short bouts of** non-REM sleep with repetitive EEG arousals, they were subjectively perceived as a single period of wakefulness **in patients with paradoxical insomnia.** Patients with insomnia were also found to have significantly greater CAP rate in stage 1 and stage 2 but not in slow wave sleep (40). **Consistent with these findings, recent research has shown a significant relationship between subjective-objective discrepancy in sleep onset latency and glucose metabolism in the areas of the brain that are related to sensory processing and consciousness (41).** Thus, while traditional measures of sleep architecture such as sleep efficiency and sleep stage percentages would suggest that patients **meeting criteria for** paradoxical insomnia underestimate their sleep

time, evidence of micro-arousals and **altered glucose metabolism** would suggest **that patients with paradoxical insomnia may be accurately describing an impaired aspect of their sleep that is missed by traditional objective measures.** Further research using fine-grained assessment methods is needed to further address the electrophysiology of paradoxical insomnia.

Paradoxical Insomnia and other sleep disorders

Insomnia and obstructive sleep apnea (OSA) have high comorbidity, with 50-55% of patients with OSA presenting with complaints of insomnia (42). Considering insomnia is commonly associated with sleep misperception (43), patients with OSA are possibly at risk for comorbid paradoxical insomnia. McCall (1995) compared PSG findings and subjective sleep of patients with and without OSA and found that both tend to overestimate sleep onset latency, but patients with OSA demonstrate greater overestimation (44). Additionally, Pinto and colleagues (2009) compared **PSG and subjective sleep perception** among patients with sleep breathing disorders, insomnia, comorbid insomnia and sleep breathing disorders and healthy controls. They reported that subjective sleep duration in patients with sleep breathing disorders was similar to the control group, and those with sleep breathing disorders tended to overestimate sleep duration **relative to PSG.** However, patients with insomnia only and with comorbid insomnia and sleep breathing disorders had the greatest sleep discrepancy **between PSG and subjective report** but underestimated sleep duration. Overestimation of sleep in the sleep breathing disorders only group was attributed to positive sleep discrepancy (45).

Another study specifically focused on sleep discrepancy in patients with OSA **and found** a significant relationship between sleep discrepancy **and dreaming**. The **investigators found that for those OSA patients with** greater sleep discrepancy, **the greater discrepancy could be**

attributed to the perception of dreams as wakefulness. However, there was no association between OSA severity and sleep discrepancy (46). Despite evidence of sleep discrepancy in OSA patients, the causal links between sleep discrepancy and OSA are not well understood (47).

Although insomnia has high comorbidity with other sleep disorders including periodic limb movement disorder, circadian rhythm disorder and restless legs syndrome (24), there is little known regarding the comorbidity of paradoxical insomnia with these disorders. However, the possibility of concurrent paradoxical insomnia may be clinically relevant, as sleep discrepancy may overshadow other comorbid sleep disorders. For example, benzodiazepines may affect respiratory symptoms negatively (48) and should be prescribed with precaution in patients with comorbid OSA. On the other hand, OSA treatment with continuous positive airway pressure in combination with cognitive behavioral therapy (CBT) can improve sleep discrepancy and sleep quality (49). Therefore, more investigation is needed to assess sleep misperception in other sleep disorders.

Paradoxical insomnia and psychiatric disorders

Insomnia has been found to be comorbid with many other psychiatric disorders including schizophrenia, depression, anxiety and alcohol dependency (50-53). Therefore, insomnia should be included in the assessment of psychiatric patients.

Evidence indicates insomnia is associated with worsening course of illness and poorer quality of life in patients with psychiatric disorders such as schizophrenia (54, 55). Insomnia may be a prodromal symptom of schizophrenia and is associated with increased likelihood of worsening symptoms and relapse (56). Notably, evidence shows patients with schizophrenia often overestimate sleep duration. Comparing subjective and objective sleep quality in patients with

schizophrenia, Bian and colleagues (2016) reported that while most patients (56.8%) had an accurate subjective assessment of their sleep, 38% overestimated sleep and 7% underestimated sleep. It has been suggested that negative symptoms of schizophrenia may affect perception, leading to overestimation of sleep duration (57). Given the prevalence of sleep discrepancy, paired objective and subjective assessment of sleep may be necessary in patients with schizophrenia.

Insomnia is among the most frequent symptoms reported in patients with major depressive disorder (MDD) (58). Subjective sleep disturbances have been found to be a risk factor for first onset and recurrent depressive episodes in both adolescent and adult populations (58-61). Significant sleep discrepancy has also been reported in patients with MDD (60). One research study concluded that sleep discrepancy in MDD was associated with severity of depression, age, and personality (62).

Sleep disturbance is a central focus in the diagnosis of post-traumatic stress disorder (PTSD) and is considered both an underlying mechanism and hallmark symptom of PTSD (63-65). Insomnia is a common complaint in patients with PTSD with an estimated 70% of patients experiencing insomnia symptoms (63). However, studies using objective measurements of sleep showed there is no difference in sleep parameters between patients with PTSD and controls (66, 67). This finding has been attributed to paradoxical insomnia (68). In addition to some physiological mechanisms, such as high levels of sympathetic nervous system activation and dysfunction of the hypothalamic-pituitary-adrenal axis seen in PTSD (69), paradoxical insomnia is associated with memory impairment (70), anxiety and ruminative thoughts (20). Therefore, non-pharmacological interventions, such as CBT, are **justified** for treatment of insomnia in patients with PTSD (71).

Although complaints of insomnia in psychiatric disorders are very common, the subtype of insomnia is rarely taken into consideration (Table 1). Therefore, we recommend clinicians pay specific attention to both subjective and objective evidence when assessing insomnia in psychiatric patients.

Paradoxical insomnia and other disorders

Sleep misperception has been found to be present in several medical disorders including rheumatoid arthritis (72), irritable bowel syndrome (73, 74) and some neurological disorders (74, Table 2). Hirsch and colleagues (1999) studied sleep discrepancy in patients with rheumatoid arthritis and reported positive sleep discrepancy (72). In contrast, patients with irritable bowel syndrome reported negative sleep discrepancy, which was attributed to an exaggerated response to internal and external stimuli (73). The finding was replicated by Heitkemper and colleagues (2005) who compared subjective and objective indices in women with irritable bowel syndrome (74).

Many neurological disorders are associated with insomnia, and there is an association between sleep quality and health related quality of life in these patients (75). Although there is **little research** assessing insomnia subtypes in these disorders, patients with epilepsy have been found to experience sleep discrepancy. Because inadequate sleep can potentially provoke seizures, Ng and Bianchi emphasized the objective assessment of patients' self-report of sleep disturbances in epilepsy (76). These studies underscore the relevance of considering sleep discrepancy in medical disorders.

Treatment of paradoxical insomnia

Existing evidence-based medicine protocols for treatments of insomnia do not provide clear guidance on the treatment of paradoxical insomnia (11-14). However, paradoxical insomnia treatment can be classified in two broad categories of pharmacological and non-pharmacological treatment, as summarized in Table 3. The short-term use of benzodiazepine and non-benzodiazepine receptor agonists can increase the likelihood that sleep will be identified as sleep by a patient with insomnia (77). Additionally, the use of atypical antipsychotics such as quetiapine and olanzapine may also be beneficial (14). Khazaie and colleagues proposed the hypothesis that fixed idea features are present in patients with paradoxical insomnia, and subsequently conducted a clinical trial comparing the efficacy of risperidone and olanzapine in the treatment of paradoxical insomnia. They found improvement in sleep quality with the two drugs, but greater efficacy was seen by olanzapine. The benefit was attributed to higher affinity to H1 receptors (78). **However, adverse side effects of atypical antipsychotic agents, such as metabolic syndrome, should be taken into consideration (79), and the presence of fixed ideas should be carefully assessed and identified before using antipsychotic medications**. Additionally, if paradoxical insomnia is due to differences in sleep microstructure as opposed to fixed ideas, then benzodiazepine drugs, especially on a long-term schedule, would not be appropriate. Compared to healthy controls, chronic use of benzodiazepines was found to be associated with a reduced ability to habituate to afferent stimuli during sleep and lower level of CAP time resulting in increased number of awakenings (80). Therefore, optimal treatment of paradoxical insomnia depends on its clinical presentation.

There is also evidence for non-pharmacological treatment for patients with paradoxical insomnia. A case series of patients with paradoxical insomnia reported improvement in symptoms through behavioral sleep education including a video explaining PSG and exploring the discrepancy

between PSG findings and self-report of sleep onset latency and total sleep time (81). Using psychoeducation to treat paradoxical insomnia may provide a safer, non-pharmacological treatment option for paradoxical insomnia.

Considering patients with paradoxical insomnia may have an inaccurate perception of sleep, behavioral approaches have shown success by giving feedback about the discrepancy between subjective and objective measures and training patients to distinguish between sleep and wakefulness (82, 83). Additionally, Lund and colleagues reported that CBT for insomnia is effective in both improvement of sleep quality measured by PSG and self-report of sleep estimation that consequently led to improvement in sleep discrepancy (84). These results were also replicated in another study of older adults with paradoxical insomnia (85). Therefore, CBT for insomnia may be considered an effective treatment for patients with paradoxical insomnia.

Finally, Case and colleagues reported the efficacy of ECT in remission of MDD and extreme paradoxical insomnia in a report of a 48-year-old woman (86). However, the results have not been replicated in other studies. Conversely, Khazaie and colleagues found no response to ECT in a 60-year-old woman with paradoxical insomnia (32). **While the presence of comorbid severe treatment resistant MDD in patients with paradoxical insomnia may justify the use of ECT, it is not appropriate as a first line of treatment should not be considered in the routine management of** paradoxical insomnia.

Specific evidence-based guidelines are needed for the treatment of paradoxical insomnia. However, current evidence suggests that pharmacological and CBT treatments may be efficacious, but further research is needed to develop specific evidence-based guidelines.

Limitations

The study has several limitations. There is inconsistent use of a consensus definition of paradoxical insomnia in the literature, and this clouds our interpretation of the published findings. In some cases, we included studies in which there is no exact diagnosis of paradoxical insomnia. Consistent terminology and diagnostic criteria will be necessary to create a body of evidence necessary to reach a consensus in future reviews.

Conclusion

Paradoxical insomnia is a complex disorder and may not be attributed to a single factor. Our review highlights the controversy of defining paradoxical insomnia as a subtype of insomnia. Although previously defined as the subjective misperception of sleep as wakefulness, more recent research has demonstrated neurophysiological correlates of paradoxical insomnia which suggests current sleep measures may not be sensitive enough to detect the differences in sleep microstructure. More fine-grained measures, such as EEG spectral analysis, CAP and event-related potentials may be necessary to better understand the etiology of paradoxical insomnia. Additionally, the lack of randomized controlled trials has prevented the development of evidence-based treatment guidelines. However, some pharmacological and CBT interventions appear promising. Future research combining the expertise of neurophysiologists and psychiatrists is needed to further develop our understanding of the etiology and treatment of paradoxical insomnia.

Practice points

1. Paradoxical insomnia is diagnosed by comparing subjective versus objective sleep time.
2. A fixated belief about lack of sleep may signify paradoxical insomnia.
3. Features of paradoxical insomnia may co-occur with psychiatric disorders.

Research agenda

1. An epidemiological study to determine prevalence of paradoxical insomnia in the general population is needed, along with studies of its associated factors in the clinical setting.
2. The underlying neurophysiological mechanisms of paradoxical insomnia should be further investigated using neuroimaging, electroencephalogram and other techniques.
3. Well-controlled clinical trials should be conducted to establish an evidence-based intervention for treatment for paradoxical insomnia.

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Table 1- Comorbidity of subjective-objective sleep discrepancy and psychiatric disorders

Authors	Study Type	Participants	Measures	Major Results
Hurwitz, et al. 1998[57]	Cross sectional study	18 Vietnam combat veterans with PTSD and 10 healthy volunteers	2 night PSG and sleep history	No detectable sleep disturbances in PSG
Tsuchiyama, et al. 2003[53]	Cross sectional study	23 patients with MDD	5 consecutive day PSG and sleep diary	Subjective-objective sleep discrepancy was found, and it was influenced by depression severity, age and personality.
Kline, et al. 2003[58]	Prospective study	26 MVC with PTSD, 76 MVC without PTSD, 19 patients without MVC	48 h Actigraphy, and mini sleep questionnaire	Lack of detectable sleep disturbance in actigraphy record.
Ghadami, et al. 2015[59]	Cross sectional study	32 patients with chronic PTSD	Two consecutive night actigraphy,	Incongruent objective and subjective results.
Bian, et al. 2016[48]	Cross sectional study	148 inpatient with schizophrenia	PSG, and sleep questionnaire	Positive sleep discrepancy

MDD: Major depressive disorder, MVC: Motor vehicle collision, PTSD: Post-traumatic stress disorder

Table 2. Subjective-objective sleep discrepancy and comorbid medical disorders

Authors	Study Type	Participants	Measures	Major Results
Hirsch, et al. 1994[64]	Cross sectional study	19 patients with rheumatoid arthritis and 19 healthy controls	Two consecutive nights PSG and self-report questionnaire	Overestimation of sleep and objective sleep fragmentation.
Elsenbruch, et al. 1994[65]	Cross sectional study	15 patients with irritable bowel syndrome(IBS) and 15 healthy controls	PSG and Pittsburgh sleep quality index	Subjective poor sleep quality and the absence of objective sleep disturbance in IBS.
Heitkemper, et al. 2005[66]	Cross sectional study	36 women with irritable bowel syndrome and 38 healthy women	Two consecutive nights PSG and Pittsburgh Sleep Quality Index	Under estimation of sleep quality in patients with severe irritable bowel syndrome.
Neg,and Bianchi , 2014 [68]	Retrospective study	64 patients with epilepsy, 50 patients with obstructive sleep apnea and 50 patients with insomnia	Retrospective chart review of PSG and self-report	Similar overestimation of sleep duration among patients with epilepsy and other groups.

PSG: Polysomnography

Table 3. Treatment studies for subjective-objective sleep discrepancy

Authors	Study Type	Participants	Treatment	Outcome
Downey and Bonnet 1992[82]	Clinical trial	10 subject with insomnia in both control, and training group, and 10 subject with insomnia in training group	Group	Significant improvement in subjective sleep latency, correctness of estimates of sleep versus wakefulness and perceived ability to fall asleep
Case et al., 2008 [86]	Case report	Single case study with comorbid depression	Electroconvulsive therapy	Remission of depression and PI
Tang and Harvey 2004[83]	Clinical trial	20 patients with insomnia (shown discrepancy), 20 patients with insomnia (no demonstration of discrepancy)	Behavioral experiment	Improvement of subjective perception of SOL and reduction of sleep related anxiety and preoccupation.

Khazaie et al (2010)[32]	Case report	Single case study	Olanzapine (2.5 mg twice per day	Remission of PI
Geyer et al., 2011[81]	Case series	Four participant case series	Behavioral sleep education explaining PSG and the discrepancy between PSG findings and subjective report	Improvement in self-reported sleep onset latency, total sleep time, and Insomnia Severity Index scores
Khazaie et al., 2013[78]	Clinical trial	29 patients with PI	Either 8 weeks of olanzapine 10 mg daily (n=14) or 8 weeks of risperidone 4 mg daily (n=15).	Improvement of sleep quality with both drugs, but greater efficacy was seen with olanzapine.
Lund et. al 2013[84]		60 adult with co morbid insomnia	CBT	Decreased N1, improvement in sleep quality, and SOSD
Kay et. al 2015[85]	Clinical trial	63 adult with insomnia, and 51 healthy control	CBT_I	Change in WASO discrepancy

CBT: Cognitive behavioral therapy PI: Paradoxical insomnia SOSD: subjective-objective sleep discrepancy WASO: wake after sleep onset