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OBJECTIVE AND SUBJECTIVE CHARACTERISTICS OF SLEEP IN CHRONIC INSOMNIA

Doctoral Dissertation

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Declaration:

I declare that the present thesis is my own and that it has been conducted under supervision of PhDr. Jana Kopřivová, Ph.D. I also declare that all the used information resources have been listed in the section References. No part of this dissertation has been submitted for any other degree.

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SUMMARY

Background: Insomnia is one of the most prevalent sleep disorders, negatively impacting the quality of life and increasing the risk of other health problems. Many patients with insomnia underestimate their sleep quantity compared to objective sleep measures. This objective and subjective sleep discrepancy (sleep misperception) occurs in different insomnia subtypes as well as in insomnia with a comorbid psychiatric disorder. Although previous research suggests that the sleep discrepancy reflects specific objective sleep alterations, the results of studies are inconsistent. Moreover, its relation to psychiatric comorbidities is not clear, as well as its role in the insomnia treatment.

Aims: The theoretical part of the present thesis aimed to provide an overview of the recent research on sleep discrepancy in insomnia. The experimental part consists of four studies with the following goals: (1) to explore sleep electroencephalographic (EEG) correlates of sleep discrepancy in insomnia patients (Study 1); (2) to assess the association between sleep discrepancy and psychopathology (Study 2); (3) to examine changes of sleep discrepancy during and after the cognitive behavioural therapy for insomnia (CBT-I; Study 3); (4) to assess whether the additional chronotherapeutic tool can enhance the effect of CBT-I on sleep parameters.

Methods: All four studies included patients with chronic insomnia. Study 1 also included a good sleeper control group (GS). In this study, patients were further divided into three insomnia subtypes according to the objective sleep parameters, and the presence or absence of sleep discrepancy. Subjective and objective (polysomnographic; PSG) sleep parameters were compared between the groups. The sleep EEG activity was further analysed by a power spectral analysis. Study 2 compared the degree of sleep discrepancy between groups of insomnia patients with and without depressive symptoms. In both of these PSG studies, correlational analyses were conducted to examine EEG correlates of sleep discrepancy. Objective sleep measures in Studies 3 and 4 were obtained by actigraphy. Study 3 compared the treatment outcome in patients with different subjective sleep evaluations. Study 4 explored the effect of CBT-I in combination with a blue-light blocking glasses compared to the CBT-I group with clear placebo glasses.

Results: Both PSG studies found a significant association between a reduction of REM sleep proportion and a degree of sleep discrepancy. Reduced REM sleep was also the only common feature found in the two different groups of patients with sleep misperception. A higher degree of sleep discrepancy was observed in insomnia patients with depressive symptoms, and this tendency was associated with depression severity. Studies on CBT-I revealed a significant reduction of sleep discrepancy after the therapy. In Study 3, patients with accurate estimates of sleep at baseline tended to overestimate sleep quantity after the CBT-I. A similar result was found in Study 4. Only patients in the CBT-I group with blue-light blocking glasses tended to overestimate their sleep quantity after the treatment when compared to the CBT-I group with placebo glasses.

Conclusions: The findings are in line with the assumption that insomnia patients with sleep discrepancy show specific sleep alterations, highlight the importance of REM sleep in subjective evaluation of sleep, point out the association between sleep discrepancy and depressive symptoms, and prove the efficacy of CBT-I in reducing sleep discrepancies. The additional chronotherapeutic tool showed promising results by enhancing the effect of CBT-I. Future studies should explore the role of sleep discrepancy in common pathophysiology of insomnia and depression, use more sensitive neurophysiological measures, and also involve patients who overestimate their sleep quantity.

SOUHRN

Úvod: Insomnie je jednou z nejčastějších poruch spánku, která negativně ovlivňuje kvalitu života a zvyšuje riziko rozvoje dalších zdravotních potíží. Jedním z častých fenoménů, objevujících se u pacientů s insomnií, je podhodnocování délky spánku ve srovnání s objektivním měřením. Tato objektivní a subjektivní spánková diskrepance (spánková mispercepce) se může vyskytnout u různých podtypů insomnie, a u insomnie komorbidní s psychiatrickým onemocněním. Ačkoliv studie poukazují na to, že spánková diskrepance reflektuje specifické objektivní změny spánku, jejich výsledky nejsou konzistentní. Stejně tak není jasné, jakou roli hraje tento fenomén ve vztahu insomnie a komorbidní psychopatologie, a jak se mění během léčby.
Cíle: Cílem teoretické části této dizertace bylo poskytnout literární přehled současných studií zaměřených na spánkovou diskrepanci u insomnie. Praktická část je tvořena čtyřmi studiemi, které měly za cíl: (1) nalézt spánkové elektroencefalografické (EEG) koreláty spánkové diskrepance (Studie 1); (2) zjistit, zda existuje vztah mezi spánkovou diskrepancí a psychopatologií (Studie 2); (3) zkoumat změny ve spánkové diskrepanci během a po kognitivně behaviorální terapii insomnie (KBT-I; Studie 3); (4) posoudit, zda chronoterapeutická intervence může zvýšit efekt KBT-I na spánkové parametry (Studie 4).
Metody: Všechny čtyři studie zahrnovaly pacienty s chronickou insomnií. Studie 1 zahrnovala také kontrolní skupinu zdravých dobrovolníků (KS). V této studii byli pacienti s insomnií dále rozděleni do skupin dle přítomnosti či absence spánkové diskrepance, a dle objektivních spánkových parametrů. Následně byly srovnávány rozdíly v subjektivních a objektivních (polysomnografických; PSG) parametrech spánku mezi skupinami. Spánková EEG aktivita byla dále zpracována pomocí spektrální analýzy. Studie 2 porovnávala rozdíl v míře spánkové mispercepce u pacientů s insomnií a depresivními příznaky se skupinou pacientů bez depresivních příznaků. Obě PSG studie zahrnovaly také korelační analýzy pro zkoumání EEG korelátů spánkové diskrepance. Ve Studiích 3 a 4 byl spánek objektivně měřen pomocí aktigrafie. Studie 3 navíc porovnávala efekt KBT-I u pacientů s rozdílným subjektivním hodnocením spánku. Studie 4 srovnávala efekt kombinace KBT-I s večerním užíváním brýlí filtrujících modré světlo, s kombinací KBT-I a placebo brýlemi.
Výsledky: Obě PSG studie prokázaly signifikantní korelaci mezi sníženým množstvím REM spánku a zvýšenou mírou spánkové diskrepance. Snížené množství REM spánku bylo také jediným společným znakem nalezeným u odlišných skupin pacientů se spánkovou mispercepcí. U pacientů s depresivními symptomy byla nalezena signifikantně vyšší míra spánkové diskrepance, která pozitivně korelovala se závažností deprese. KBT-I bylo spojeno se snížením míry spánkové mispercepce. Pacienti, kteří na začátku terapie hodnotili svůj spánek v souladu s objektivním měřením, měli po KBT-I tendenci dobu spánku nadhodnocovat. Stejný výsledek byl nalezen u skupiny absolvující KBT-I v kombinaci s filtračními brýlemi, a nikoliv u KBT-I skupiny s placebo brýlemi.
Závěr: Výsledky dizertace jsou v souladu s předpokladem, že spánková diskrepance reflektuje specifické objektivní změny spánku, vyzdvihují roli REM stádia v subjektivním hodnocení délky spánku, poukazují na vztah mezi spánkovou diskrepancí a depresivními symptomy, a potvrzují efekt KBT-I na snížení míry spánkové diskrepance. Přidaná chronoterapeutická intervence ukázala slibné výsledky posílením efektu KBT-I na kvalitu spánku. Budoucí studie by měly dále prozkoumat roli spánkové diskrepance ve společné patofyziologii insomnie a deprese, použít citlivější neurofyziologické metody, a zahrnout mnohdy opomíjenou skupinu pacientů, kteří kvantitu svého spánku nadhodnocují.

ABBREVIATIONS

A/O	Accurate/overestimating group
ANCOVA	Univariate analysis of covariance
ARAS	Ascending reticular activating systems
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory
BZ	Benzodiazepines
BZRAs	Benzodiazepine receptor agonists
CAP	Cyclic alternating pattern
CBT-I	Cognitive behavioural therapy for insomnia
EEG	Electroencephalography
ES	Effect size
ESS	Epworth Sleepiness Scale
GABA	Gamma-aminobutyric acid
GLM	General linear model
GS	Good sleepers
HAS	Hyperarousal Scale
ICD	International Classification of Diseases
ICSD	International Classification of Sleep Disorders
INS	Insomnia without depressive symptoms
INS-D	Insomnia with depressive symptoms
ISI	Insomnia Severity Index
MANCOVA	Multivariate analysis of covariance
MI	Misperception index
NIMH	National Institute of Mental Health
NREM	Non-rapid eye movement
NWAKE	Number of awakenings
PARA	Paradoxical insomnia
PSA	Power spectral analysis
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
PSY	Psychophysiological insomnia
PSY/MIS	Psychophysiological insomnia with sleep misperception
QOL	A modified version of the brief World Health Organization Quality of Life questionnaire
REM	Rapid eye movement
SDS	Sheehan Disability Scale
SE	Sleep efficiency
SNP	Single-nucleotide polymorphism
SOL	Sleep onset latency
SWS	Slow wave sleep
TST	Total sleep time
UN	Underestimating group
VLPO	Ventrolateral preoptic area
WASO	Wake after sleep onset

1 INTRODUCTION

Insomnia is among the most prevalent sleep disorders. Its symptoms occur in approximately 30% of the adult population (Ohayon, 2002), and their prevalence is still increasing (Calem et al., 2012). Chronic insomnia may be a disabling condition significantly impacting patients' life. It can lead to several adverse health and socioeconomic consequences, such as increased risk of depression (C. Baglioni et al., 2011), anxiety (Bjorøy, Jørgensen, Pallesen, & Bjorvatn, 2020), cardiovascular disease (Sofi et al., 2014), higher absenteeism (Sivertsen, Overland, Bjorvatn, Maeland, & Mykletun, 2009) or increased risk of injuries (Buysse et al., 2007). Continued investigation of insomnia pathophysiological mechanisms and increasing the effectiveness and availability of its treatment is therefore crucial.

The diagnosis of insomnia is established on patients' subjective complaints with no requirement of the objective measure of sleep. One of the reasons for subjectively based diagnostics of insomnia is a large variability in patients' objective sleep parameters. Many patients do not show any objective impairment of their sleep continuity compared to subjective complaints (Vanable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 2000). Prevalence of this subjective and objective sleep discrepancy (or sleep misperception), ranges between 9.2 and 50 %, depending on the criteria used (Dorsey & Bootzin, 1997; Edinger & Krystal, 2003). Sleep misperception may occur in different insomnia subtypes and insomnia comorbid with psychiatric disorders, such as depression (Rotenberg, Indursky, Kayumov, Sirota, & Melamed, 2000), bipolar disorder (Krishnamurthy et al., 2018), or posttraumatic stress disorder (Ghadami, Khaledi-Paveh, Nasouri, & Khazaie, 2015). It is clinically essential to explore this phenomenon as patients with sleep misperception may develop more extensive objective impairment of sleep (Harvey & Tang, 2012). Moreover, increasing evidence suggests that sleep misperception is not only about wrong perception, but it more likely reflects objective alterations of sleep, which are not captured by traditional sleep measures (Rezaie, 2018). Insights into psychological and neurophysiological mechanisms underlying sleep discrepancy may thus promote the understanding of insomnia disorder and other mental disorders, which are usually accompanied by sleep disturbances (Dolsen, Asarnow, & Harvey, 2014).

Concerning the treatment of insomnia, the efficacy of the first treatment choice, cognitive behavioural therapy (CBT-I), has been proven by previous research (Koffel, Koffel,

& Gehrman, 2015). However, studies describing the mechanism of its impact on sleep are lacking, with a majority of them assessing only subjective sleep parameters (van Straten et al., 2018). A recent meta-analysis has concluded that CBT-I has a rather blunted effect on objective compared to subjective sleep parameters, but there is an urgent need for more studies using objective sleep measures (Mitchell, Bisdounis, Ballesio, Omlin, & Kyle, 2019).

Given the gaps in the insomnia research mentioned above, the aim of this dissertation was to (a) explore objective sleep alterations associated with sleep discrepancy in insomnia patients; (b) assess whether sleep discrepancy might be associated with psychopathology commonly observed in insomnia; (c) examine changes of sleep discrepancy during and after the therapy; (d) assess whether the additional chronotherapeutic tool can enhance the effect of insomnia treatment on subjective and objective sleep parameters.

I. REVIEW OF THE LITERATURE

2 INSOMNIA DISORDER

2.1 Insomnia subtypes

Insomnia disorder is characterized by sustained complaints about poor sleep quality, such as difficulties in initiating and maintaining sleep, or waking up earlier than desired. Daytime consequences of insomnia include attention, concentration or memory impairment, mood disturbances, daytime sleepiness, or behavioural problems (AASM, 2014). Exact diagnostic criteria are presented in Table 1.

A. The patient reports one or more following: 1. Difficulty initiating sleep. 2. Difficulty maintaining sleep. 3. Waking up earlier than desired.
B. The patient reports one or more of the following related to night-time sleep difficulty: 1. Fatigue/malaise. 2. Attention, concentration, or memory impairment. 3. Impaired social, family, occupational, or academic performance. 4. Mood disturbance/irritability. 5. Daytime sleepiness. 6. Behavioural problems (e.g. hyperactivity, impulsivity, aggression). 7. Reduced motivation/energy/initiative. 8. Proneness for errors/accidents. 9. Concerns about or dissatisfaction with sleep.
C. The reported sleep/wake complaints cannot be explained purely by inadequate circumstances (i.e., the environment is safe, dark, quiet, and comfortable) for sleep.
D. The sleep disturbance and associated daytime symptoms occur at least three times per week.
E. The sleep disturbance and associated daytime symptoms have been present for at least three months.
F. The sleep/wake difficulty is not better explained by another sleep disorder.

Table 1. Diagnostic criteria of chronic insomnia according to the International Classification of Sleep Disorders, Third Edition (ICSD 3), (AASM, 2014).

The classification of insomnia has been discussed among clinicians and researchers for decades. The latest third edition of the International Classification of Sleep Disorders (ICSD 3) distinguishes three types of insomnia disorder based on its duration: chronic insomnia, short-term insomnia and other insomnia disorder (Table 2; AASM 2014). This classification had resulted from a reduction of eleven insomnia subtypes described in ICSD 2 (Table 3; AASM 2005). The attempt was to simplify clinical diagnostic as there is a lack of empirical evidence for the insomnia subtypes existence (Bastien et al., 2014). Nevertheless, at least two types of insomnia are important to consider: psychophysiological and paradoxical (sleep misperception) insomnia, which are probably the two of the most prevalent types of insomnia disorder (Edinger JD, 2004).

Chronic insomnia (> 3 months)
Short-term insomnia (< 3 months)
Other insomnia disorder

Table 2. Insomnia subtypes, according to ICSD 3 (AASM, 2014).

Adjustment sleep disorder (acute insomnia)
Psychophysiological insomnia
Paradoxical insomnia (sleep misperception)
Idiopathic insomnia
Insomnia due to mental disorder
Inadequate sleep hygiene
Behavioural insomnia of childhood
Insomnia due to drug or substance
Insomnia due to medical condition
Insomnia not due to a substance or known physiological condition, unspecified
Physiological (organic) insomnia, unspecified

Table 3. Insomnia subtypes, according to ISCD 2 (AASM, 2005).

Psychophysiological insomnia (PSY) is characterized by objectively altered sleep macrostructure and accurate subjective estimates of sleep quantity and quality. On the other hand, paradoxical insomnia (PARA) patients show a substantial discrepancy between their subjective complaints and objective findings on polysomnography (PSG), which usually indicates a standard duration of sleep onset latency (SOL), total sleep time (TST) and/or wake after sleep onset (WASO). Sleep misperception also occurs in insomnia patients with objectively impaired sleep continuity. Therefore, the PSY and PARA subtypes do not exclude each other. These patients show objective sleep alterations on the macrostructure level and a tendency to misperceive their sleep (PSY/MIS) concurrently. As St-Jean and Bastien (2009) proposed, it is difficult to determine the prevalence of this type of insomnia, and the objective and subjective sleep discrepancy should rather be seen as a continuum with an extreme case of sleep misperception (PARA) at its end (Figure 1).

Although PSY and PARA do not fulfil assumptions to be considered insomnia subtypes (Edinger et al., 2011), their differentiation might help to describe the pathophysiological mechanisms of sleep misperception, and thus insomnia itself. Indeed, there are still studies using the insomnia subgroups for this purpose, showing that PARA appears to have different pathophysiology (Liao, Zhu, & Li, 2018; Normand, St-Hilaire, & Bastien, 2016; Spiegelhalder et al., 2012).

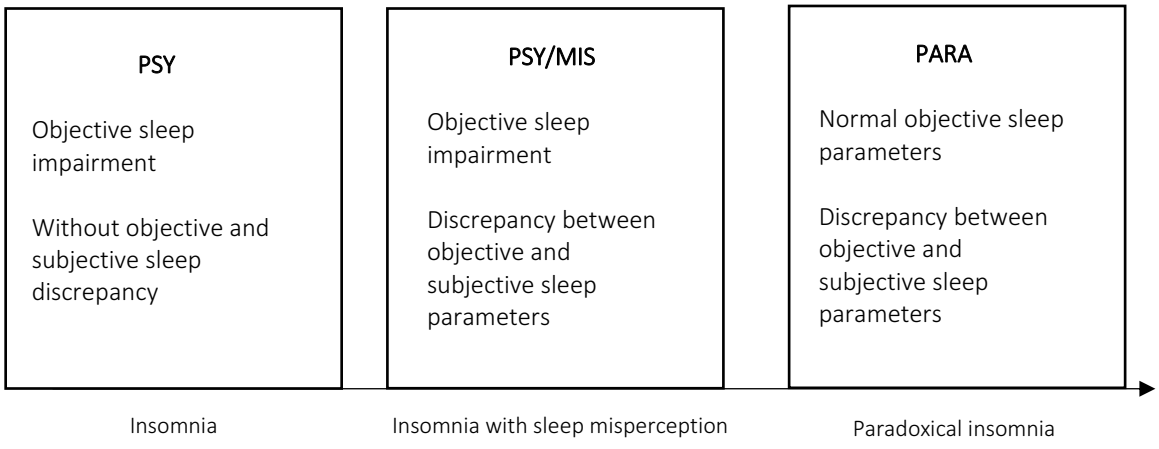

Figure 1. Subtypes of insomnia defined by objective and subjective sleep parameters. Edited from our review (Veldova, Sos, & Koprivova, 2015). PSY: psychophysiological insomnia, PSY/MIS: psychophysiological insomnia with sleep misperception, PARA: paradoxical insomnia.

2.2 Pathophysiology

In recent years, progress has been made in the understanding of the aetiology and pathophysiology of insomnia. Advances in sleep neuroscience have provided new insights into physiological mechanisms that underlie and contribute to insomnia development and maintenance. Although there is still no universal model, several concepts involving different underlying mechanisms of insomnia have been proposed.

2.2.1 Behavioural and cognitive mechanisms

The 3P model by Spielman (1986) describes three different types of factors contributing to insomnia disorder. Predisposing factors, such as genetics (Palagini, Biber, & Riemann, 2014) or personality traits (van de Laar, Verbeek, Pevernagie, Aldenkamp, & Overeem, 2010), can make an individual more vulnerable and susceptible to insomnia. Precipitating factors include significant life events facilitating the acute insomnia onset, such as uncertain family situation, health, work, or school problems (Bastien, Vallieres, & Morin, 2004). Acute insomnia may be therefore considered a normal part of fight or flight stress reaction (Ellis, Perlis, Neale, Espie, & Bastien, 2012) when a person cannot sleep properly because of perceived threat. According to the 3P model, the transition from acute to chronic insomnia is mainly determined by perpetuating factors. These factors involve a variety of cognitive processes (e.g., catastrophizing, worry, dysfunctional beliefs about sleep and insomnia) and behaviours (e.g., napping, increasing time in bed), which significantly contribute to insomnia maintenance (Spielman, Saskin, & Thorpy, 1987). The role of cognitive processes is also highlighted in **the cognitive model** of insomnia proposed by Harvey (2002). This model suggests that insomnia results from excessive worry about poor sleep, which leads to increased arousal and maladaptive behaviours aiming to prolong sleep and minimize the consequences of insomnia. The 3P model has also been complemented by **the neurocognitive model,** which adds neurobiological and neurophysiological findings to insomnia pathophysiology (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997b). The model suggests that insomnia may lead to conditioned cortical arousal, which manifests as increased electroencephalographic (EEG) activity during sleep.

2.2.2 Hyperarousal concept of insomnia

Insomnia is often considered a hyperarousal disorder caused by increased somatic, cortical, emotional, and cognitive activation. In acute insomnia, this activation occurs as a triggered response to threat-related stimuli. This flight-or-fight response is characterized by the activation of the autonomic nervous system and enhanced adrenergic and hypothalamic-pituitary-adrenal axis activity (Charmandari, Tsigos, & Chrousos, 2005). Hyperarousal has also been found in patients with chronic insomnia who tend to worry and ruminate about sleep problems and its consequences, and are likely to develop conditioned arousal, causing the sleep-related environment to become stressful stimuli. Subsequent maladaptive behaviour contributes to the perpetuation of insomnia (Riemann et al., 2010).

A presence of hyperarousal in insomnia has been proved in various research domains. Studies have demonstrated enhanced heart rate variability, enhanced sympathetic activity and reduced parasympathetic activity in insomnia patients during sleep (Maes et al., 2014) as well as endocrine changes (Vgontzas et al., 2001), enhanced global cerebral glucose metabolism (Nofzinger et al., 2004) and reduced gamma-aminobutyric acid (GABA) in patients with insomnia (Plante, Jensen, Schoerning, & Winkelman, 2012). At the level of the central nervous system, studies using power spectral measures of high-frequency EEG activity showed enhanced cortical activation in insomnia patients during sleep or sleep onset (Cervena et al., 2004; St-Jean, Turcotte, Perusse, & Bastien, 2013). These findings were further supported by a study using a more sensitive measure, i.e., a high-density EEG, which revealed a global increase in high-frequency EEG activity and locally increased alpha activity in sensory and motor areas during deep NREM sleep in insomnia patients compared to good sleepers (Figure 2; Riedner et al., 2016).

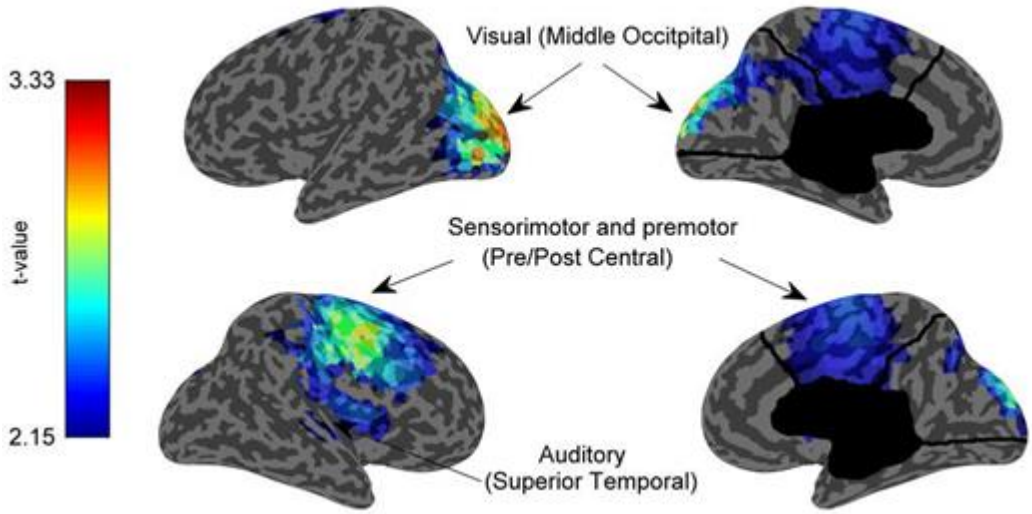

Figure 2. Increased alpha EEG activity during deep NREM sleep in patients with insomnia compared to good sleepers measured by 256 channel high-density EEG (Riedner et al., 2016). Increased alpha EEG activity in sensory and motor areas in patients with insomnia compared to good sleepers during the deep non-rapid eye movement sleep (NREM). The inflated cortical map presents the t-values of the brain areas showing significant differences ($p < .05$) between insomnia patients and good sleepers revealed by a nonparametric mapping cluster test. Areas with the most significant differences are visual (Brodmann areas 18, 19), somatosensory (2, 3, 7, 40), motor (4) and premotor (6) areas. Increased alpha activity is also present in auditory and language areas (22, 40).

2.2.3 Sleep and wake regulation

Hyperarousal on different levels may inhibit sleep regulatory processes from occurring in insomnia patients naturally. Insomnia might be associated with a dysregulation of sleep and wake brain networks, which are essential for normal sleep regulation. According to **the "flip-flop switch" model** of Saper, Chou, and Scammell (2001), sleep and wake states occur based on reciprocal inhibition between the sleep-promoting and wake-promoting brain regions. During sleep, the activation of the ventrolateral preoptic area (VLPO) neurons leads to decreased activation of wake-promoting areas (locus coeruleus, nucleus dorsalis raphae, nucleus tuberomammillaris, nucleus tegmentalis laterodorsalis, nucleus tegmentalis pedunculopontinus) via inhibitory neurotransmitters, such as GABA. According to the animal model of insomnia induced by acute stress in rats (Cano, Mochizuki, & Saper, 2008), insomnia may be caused by decreased ability of the VLPO to inhibit the ascending reticular activating systems (ARAS) responsible for arousal. It is believed that the activation of limbic system (i.e., emotional

activation) prevents inhibition of the ARAS. At the same time, the ARAS cannot inhibit the VLPO because of the homeostatic and circadian pressure. Thus, both sleep and wake-promoting systems are activated at the same time, which may lead to sleep disruption.

The homeostatic sleep drive and circadian rhythmicity are the two crucial mechanisms that regulate arousal and sleep-promoting systems in the human brain. **The two-process model** proposes that a homeostatic process (Process S) interacts with a process controlled by the circadian pacemaker (Process C), both modulated by physiological and behavioural variables. The coordination of both processes is crucial for normal sleep (Borbely, Daan, Wirz-Justice, & Deboer, 2016). It has been proposed that insomnia is associated with a deficiency in the Process S, leading to a less prominent homeostatic sleep drive (Pigeon & Perlis, 2006), as well as circadian rhythm dysfunction (Lack, Gradisar, Van Someren, Wright, & Lushington, 2008) further promoted by maladaptive behaviour, such as irregular sleep schedule or excessive time in bed.

2.2.4 Integration

The above-described models suggest that several biological and psychological factors are involved in the development and maintenance of insomnia. In summary, individuals with increased genetic risk (Palagini et al., 2014), abnormalities in neurobiological processes (Saper et al., 2001) or specific personality characteristics, such as perfectionism and neuroticism (van de Laar et al., 2010), are more likely to develop insomnia which is usually triggered by psychosocial stressors (Bastien et al., 2004). These traits may determine the occurrence of physiological hyperarousal, emotional, cognitive, and behavioural processes, usually as a reaction to stress-related stimuli, which increases the risk for developing and maintaining chronic insomnia (Levenson, Kay, & Buysse, 2015).

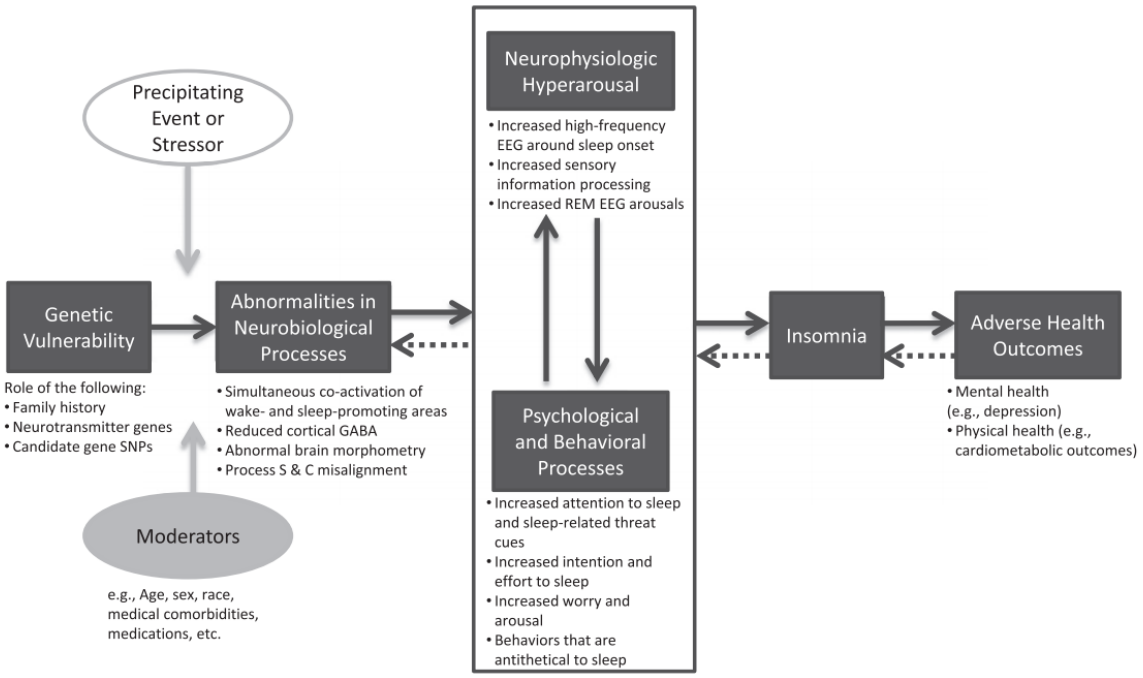

Figure 3. Model of the pathophysiology of insomnia by Levenson et al., 2015. GABA: gamma-aminobutyric acid; SNP: single-nucleotide polymorphism

2.3 Treatment

2.3.1 Cognitive behavioural therapy for insomnia

The first treatment choice for chronic insomnia should be CBT-I (Riemann et al., 2017). CBT-I aims to target maladaptive sleep habits, unhelpful beliefs and thoughts about sleep, and hyperarousal (Buysse et al., 2011; Morin & Espie, 2003). The description of CBT-I components is provided in the following paragraphs.

Psychoeducation aims to provide relevant information about normal sleep, age-related changes in sleep, and sleep hygiene rules. Sleep hygiene is a set of recommendations that may help to increase sleep quality. It involves information about environmental factors (e.g., light, noise) and behaviours (e.g., physical exercise, substance use), possibly interfering with the quality of sleep.

Relaxation techniques are usually involved in the CBT-I package, aiming to reduce both physiological and cognitive arousal, i.e., physical tension and intrusive thoughts at bedtime. Several techniques can be trained, such as progressive muscle relaxation, autogenic training, or imagery training.

The goal of **sleep restriction therapy** is to reduce the time spent in bed, increase the homeostatic sleep drive (process S), and to establish a regular sleep-wake schedule to support circadian rhythms (process C). Patients are recommended to spend the same amount of time in bed, as was their average total sleep time during the previous week. Sleep quality is monitored weekly by sleep diaries, and the recommended sleep window is adjusted based on sleep efficiency (SE). Time in bed is increased by 15-30 minutes (when SE is ≥ 85-90 %), remains the same, or is decreased by 15-30 minutes (when SE is < 85-80%; Riemann et al., 2017).

Stimulus control therapy is a set of recommendations aiming to strengthen the bed and sleep association by reducing time spent in bed awake (Bootzin, 1972). These recommendations involve: (a) leaving the bed if cannot fall asleep, coming back to bed when feeling sleepy; (b) avoiding naps during the day; (c) using the bed and bedroom only for sleep and sex.

Cognitive therapy aims to identify, challenge, and change dysfunctional and faulty beliefs about sleep, insomnia, and its consequences (Morin & Espie, 2003). A typical dysfunctional belief about insomnia consequences is, for example: "*If I do not sleep for 8 hours, I will not be able to work tomorrow. It will be a disaster*". The outcome of cognitive strategies should be a decrease in negative emotions affecting sleep quality.

2.3.2 Pharmacotherapy

Despite the recommendation of guidelines, the most common treatment of insomnia is pharmacotherapy. Around 60 % of insomnia patients receive benzodiazepines (BZ) or benzodiazepines-related medication from their general practitioners (Hoebert et al., 2012). Major drug classes used in Europe include already mentioned BZ and benzodiazepine receptor agonists (BZRAs), antidepressants, antipsychotics, antihistamines, phytotherapeutic substances, and melatonin (Riemann et al., 2017).

A meta-analysis by Winkler, Auer, Doering, and Rief (2014) has shown that BZ and BZRAs are effective in the short-term treatment (maximum four weeks) of insomnia and that their efficacy is more robust than that of sedative antidepressants. However, according to the insomnia guideline by Riemann et al., (2017), BZ, BZRAs and also sedative antidepressants may be prescribed for short-term treatment of insomnia if the first-choice treatment (CBT-I) is not efficient or available. On the other hand, antipsychotics, antihistamines, melatonin, and phytotherapy are not recommended for insomnia patients due to low to insufficient quality of evidence, according to the guideline.

2.3.3 Other therapeutic approaches

Small to moderate effects on sleep parameters were found for light therapy in the treatment of insomnia (van Maanen, Meijer, van der Heijden, & Oort, 2016), which is often used in the treatment of circadian rhythm disorder or seasonal affective disorders. Recent research showed that blocking a nocturnal blue light may be promising for insomnia patients (Shechter, Kim, St-Onge, & Westwood, 2018). Several therapeutic approaches have been suggested in the field of alternative medicine, for example, acupuncture, aromatherapy, homeopathy, or yoga. Nevertheless, due to poor quality of original studies, it is not possible to draw definite conclusions according to the authors of meta-analyses (Ernst, Lee, & Choi, 2011; Cheuk, Yeung, Chung, & Wong, 2012) and the insomnia guideline (Riemann et al., 2017).

3 OBJECTIVE AND SUBJECTIVE SLEEP DISCREPANCY

3.1 Definition

Insomnia diagnosis is based exclusively on subjective complaints because of a great variability in objective sleep parameters and the possible presence of more subtle objective alterations which are not detectable with traditional objective sleep measures (Parrino, Ferri, Bruni, & Terzano, 2012). Another reason is an often present discrepancy between subjective sleep evaluation and objective findings either by actigraphy (Van Den Berg et al., 2008) or PSG (Morgenthaler et al., 2006). However, not all insomnia patients show this mismatch. Some patients overestimate the quality of their sleep compared to objective findings, which is sometimes called ´positive sleep misperception´ (Trajanovic, Radivojevic, Kaushansky, & Shapiro, 2007) or positive sleep discrepancy (Kay, Buysse, Germain, Hall, & Monk, 2015). Moreover, sleep discrepancy may also occur in healthy individuals (Bianchi, Wang, & Klerman, 2012), albeit a stronger tendency to underestimate sleep quantity is observed in individuals experiencing insomnia (Castelnovo et al., 2019).

There have been two approaches to sleep misperception phenomenon. Some authors see a mismatch between sleep parameters as a continuum and consider it a characteristic trait of most of the insomnia patients. The extreme cases of sleep misperception, such as PARA, lies at the end of this continuum (Manconi et al., 2010). Other authors consider PARA (sleep misperception) a distinct diagnostic entity (Edinger & Krystal, 2003). The absence of validated quantitative criteria of sleep misperception or PARA undoubtedly contributes to the unreached consensus in the scientific community. In a recent review, Castelnovo et al. (2019) provided a critical overview and analysis of available definitions used in sleep misperception research. They concluded that the current quantitative definition of objective and subjective sleep discrepancy or PARA is insufficient due to the different criteria used in many studies. These criteria include a different range of discrepancy between sleep parameters (i.e., a different amount of time used as a cut off for a significant sleep misperception) and also different sleep parameters used (TST, SOL, or SE). The authors conclude that there is no clear recommendation on how to select patients for research on sleep misperception. However, they support the use of TST over the SOL when identifying patients with significant sleep misperception due to ICSD definitions, which usually contain information about the amount of sleep obtained during the

night instead of the amount of wakefulness (AASM, 2005). Another reason is the fact that patients with significant sleep discrepancy and PARA sufferers typically complain about sleeping just for few hours or not sleeping at all (Castelnovo et al., 2019).

It is important to note that the sleep discrepancy may reflect an objective sleep impairment and can also negatively impact the insomnia progression. These patients can become distressed and anxious about their inadequate sleep, which may promote an objective sleep disruption (Harvey, 2002; Mercer, Bootzin, & Lack, 2002). Despite its relevance in insomnia disorder, the number of studies focusing on its role in the aetiology and treatment of insomnia is still small (Rezaie, 2018).

3.2 Aetiology

3.2.1 Psychological factors

Certain behaviours and cognitions around sleep onset may present an ideal base for sleep misperception. For example, ***clock monitoring*** while trying to fall asleep may lead to worry, enhanced arousal, and contribute to the overestimation of SOL (Tang, Anne Schmidt, & Harvey, 2007). Specific cognitive processes, especially worry and selective attention, may bias our perception of sleep onset latency (Mercer et al., 2002). Indeed, the earlier literature has shown that the more information a person processes in a particular unit of time, the longer the time is perceived (Thomas & Cantor, 1976).

Due to many methodological differences across studies, definite conclusions cannot be made about ***personality predisposition*** to sleep misperception (van de Laar et al., 2010). Research on personality traits in insomnia often lacks accurate diagnostic and subsequent differentiation of insomnia subtypes. However, one common trend has been found. Patients with insomnia often show signs of neuroticism, internalisation, anxiety, and perfectionism (van de Laar et al., 2010). Patients with sleep misperception showed a higher level of neuroticism compared to patients with objective insomnia (PSY) and healthy subjects (Dorsey & Bootzin, 1997). Authors Fernandez-Mendoza et al. (2011) suggested two phenotypes of insomnia: (a) patients with sleep misperception, depressive mood, rumination, anxiety, worries, and

insufficient coping with stress; and (b) insomnia with objective sleep impairment, cognitive deficit especially in information processing or attention.

Other studies focused on the possible association between sleep misperception and wrong time estimation. The results suggest no significant differences between insomnia and healthy subjects in the ability to estimate a certain amount of time during the night (Tang & Harvey, 2005). Thus, sleep misperception cannot be explained simply by a wrong estimation of time (Tang & Harvey, 2005). Moreover, a study by Mercer et al. (2002) showed that patients with insomnia more often reported being awake although PSG recording showed NREM 2 or REM sleep stage, while their time estimation was as accurate as in healthy control group (Mercer et al., 2002). It is, therefore, more probable that ***sleep is perceived as wake*** by patients with sleep misperception. Underlying mechanisms may be revealed by a rapidly developing sleep neuroscience research.

3.2.2 Brief awakenings

Transient awakenings, i.e., brief (3 – 30 s) awakenings hardly detected by conventional sleep stage scoring, have been shown as one of the possible factors contributing to sleep discrepancy. The EEG indicator of unstable sleep and brief awakenings is a cyclic alternating pattern (CAP) occurring during the NREM sleep stage. CAP is usually present in physiological sleep when sleep stages are changing or during motor activity. However, a higher amount of CAP indicates unstable and poor quality sleep (Terzano et al., 2001), which has been shown in patients with insomnia (Chouvarda et al., 2012) as well as in patients with insomnia and sleep misperception (Parrino, Milioli, De Paolis, Grassi, & Terzano, 2009). These patients showed a higher overall rate of CAP during NREM 1 and NREM 2 sleep stage compared to good sleepers (GS). These findings indicate increased neural activation throughout the night in patients reporting objective and subjective sleep discrepancy (Parrino et al., 2009).

3.2.3 Arousal of the autonomic nervous system

Insomnia is often associated with enhanced arousal of the autonomic nervous system (physiological arousal) during sleep, which may also contribute to sleep misperception (Maes

et al., 2014). Physiological arousal may worsen sleep discrepancy by increasing mentation during sleep onset period (Bonnet & Arand, 1992) that enhances the probability of perceiving sleep as wakefulness (Mercer et al., 2002).

Some studies have also reported significantly increased 24-hour metabolic rate in insomnia patients misperceiving their sleep compared to controls, suggesting that the state of physiological hyperarousal might be a whole day problem instead of just sleep period (Bonnet & Arand, 1997). Nonetheless, more attention is given to the cortical hyperarousal when studying sleep discrepancy and insomnia in general. It has been suggested that increased activation of the cortex reflects the conditioned cortical arousal, a crucial maintaining factor of insomnia, which is associated with enhanced sensory and information processing (Neurocognitive model of insomnia; Perlis et al., 1997).

3.2.4 Arousal of the central nervous system

Studies comparing primary insomnia with GS have found several differences in cortical activation during sleep reflected by the elevated activity in different frequency bands at central sites, analyzed by a power spectral analysis (PSA). Interestingly, studies dividing patients into PSY and PARA subtypes have also shown significant differences in cortical activity between these two groups. For example, larger absolute spectral amplitudes in alpha, sigma, and beta frequency bands during NREM 2 and NREM 4 sleep were found in the PARA group compared to PSY group and GS (Krystal, Edinger, Wohlgemuth, & Marsh, 2002; Spiegelhalder et al., 2012). These findings suggest higher cortical activation in PARA compared to PSY, which interferes with normal sleep and reflects a state of hyperarousal. Contrary to these results, St-Jean et al. (2013) have found decreased absolute cortical activation in NREM sleep in PARA compared to PSY and GS as well as an increase in relative activation in REM sleep.

Feige et al. (2008) have shown that the time spent awake and in REM sleep predicted a subjective amount of perceived wake time in insomnia. It has been hypothesized that REM sleep may be pathologically close to the state of wakefulness in insomnia patients, as evidenced by correlation with subjective wake time and increased amount of arousals during REM sleep compared to NREM sleep, and thus perceived as awake (Feige et al., 2008; Riemann et al., 2012). On the other hand, a study by Perusse et al. (2015) has shown that the more time insomnia

patients (PSY and PARA) spent in REM sleep, the better was their subjective evaluation of sleep quality and quantity (TST, SE). This correlation was more robust in PARA compared to the PSY group. One of the possible causes of these contrary results might be the fact that most of the studies differ in subdividing insomnia subtypes. Some of them gathered PSY, PSY/MIS, and PARA into one group (Maes et al., 2014), or excluded the PSY/MIS group aiming to compare only PSY and PARA (St-Jean et al., 2013). Despite the inconsistent results, studies using PSA suggest that the activity of the different frequency bands during REM and NREM sleep stages might contribute to the objective and subjective sleep discrepancy (Bastien et al., 2014). Moreover, it seems that PARA and PSY groups may have different cerebral asymmetry patterns (St-Jean, Turcotte, & Bastien, 2012). PSA studies should, therefore, distinguish insomnia subtypes based on the presence of sleep misperception (Bastien et al., 2014).

In summary, hyperarousal is one of the factors which explain the pathophysiology of insomnia and the sleep discrepancy phenomenon (Riemann et al., 2010). Moreover, it also seems to play an important role in the relationship between insomnia and psychiatric disorders (Riemann, Krone, Wulff, & Nissen, 2020).

3.3 Sleep discrepancy and psychopathology

Insomnia is often comorbid with other neuropsychiatric disorders, such as schizophrenia, depression, anxiety, or alcohol dependency (Chouinard, Poulin, Stip, & Godbout, 2004; Ohayon, Caulet, & Lemoine, 1998; Ohayon & Lemoine, 2002). Research shows that insomnia may worsen the course of illness and decrease the overall quality of life in patients with psychiatric disorders (Brissos et al., 2013). Furthermore, pre-existing chronic insomnia is an independent risk factor for the development of several psychiatric disorders, such as depression (C. Baglioni et al., 2011). On the other hand, patients with depressive disorder often show sleep problems typical for insomnia, such as subjective and objective sleep discrepancy.

3.3.1 Insomnia and depression

It is now well known that insomnia and depression are in a close bidirectional relationship. Sleep disturbances are among the most common complaints in depressed patients

often accompanied by objective sleep alterations, such as shortened REM sleep onset latency, prolonged first REM sleep episode, increased REM density, and alterations of slow-wave sleep (SWS) activity (Riemann, Berger, & Voderholzer, 2001). On the other hand, insomnia may be a predictor of depression onset (C. Baglioni et al., 2011) and a risk factor for its development (Mallon, Broman, & Hetta, 2000). Patients with persistent insomnia are about three and a half risk for depression development compared to individuals without insomnia complaints (Mallon et al., 2000). Perlis et al. (2006) found that elderly patients with persistent insomnia were approximately six times more likely to develop the first episode of the major depressive disorder compared to individuals without insomnia. A meta-analysis of Baglioni et al. (2011) suggested that non-depressed patients with insomnia are at a twofold risk of developing depressive disorder compared to those without sleep complaints. This predicting effect of insomnia is similar in different age groups, such as children, adolescents, adults, and elderly individuals (C. Baglioni et al., 2011).

Psychophysiological mechanisms underlying the predictive effect of insomnia are still not known, although certain overlap is present when comparing the pathophysiology of insomnia and depression. For example, both disorders are usually triggered by psychosocial stressors, and both show significant signs of hyperarousal (Riemann et al., 2020). A comprehensive neuropsychobiological model that suggests a possible pathway between insomnia and depression (and psychopathology in general) is illustrated in Figure 4. Nevertheless, not all insomnia patients develop depression. Both insomnia and depression can also occur independently in some cases. Both disorders may, therefore, share some underlying genetic, psychological, and neurobiological alterations, but whether or not depression occurs may depend on subsequent triggers, such as psychosocial stressors, lifestyle, coping strategies, and early treatment (Riemann et al., 2020).

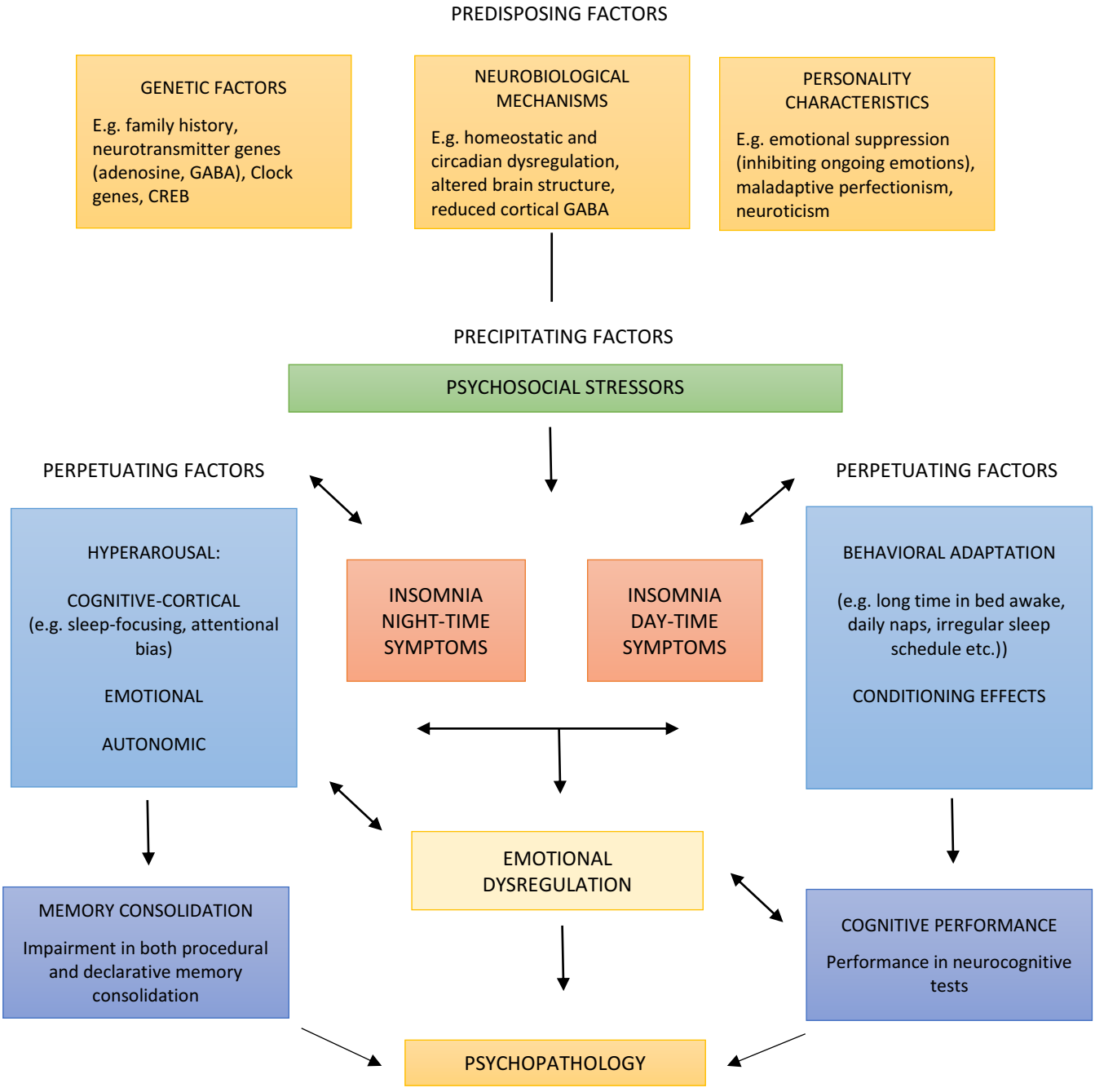

Figure 4. A comprehensive model of the link between insomnia and psychopathology (i.e., depression) edited from Riemann et al. (2020). The model integrates various fields of research (i.e., genetics, neurobiology, personality research, psychophysiology) to provide a complex understanding of the mechanisms involved in insomnia aetiology and the role of insomnia in the development of psychopathology.

3.3.2 Sleep discrepancy and depression

Similar alterations in objective sleep measures may be present in both insomnia and depression. For example, TST, SE, and SWS time may be reduced, and SOL increased in both insomnia and depression compared to healthy controls, as measured by PSG (Benca, Obermeyer, Thisted, & Gillin, 1992). Furthermore, similarly to insomnia, objective sleep parameters do not always correspond with subjective complaints about sleep in depressive patients. Sleep discrepancy has been reported in patients with major (Perlis et al., 2006; Rotenberg et al., 2000) and moderate depressive disorder (Matousek, Cervena, Zavesicka, & Brunovsky, 2004), and was shown to be associated with severity of depression. The more severe depression, the higher the degree of sleep discrepancy (Tsuchiyama, Nagayama, Kudo, Kojima, & Yamada, 2003). Nevertheless, not all depressed patients underestimate their sleep (Edinger & Fins, 1995), and not all insomnia patients with sleep discrepancy or PARA show depressive symptoms (Moon, Song, & Cho, 2015).

Table 4 summarizes studies involving measures of sleep discrepancy and depression in individuals with insomnia or insomnia symptoms. Including criteria for the presented studies were: (a) distinguishing insomnia with sleep discrepancy or PARA, and usage of depressive symptoms measurement, and/or (b) exploring the association between sleep discrepancy and depressive symptoms in insomnia patients. Inconsistent findings are present regarding the depressive symptoms in PARA or insomnia patients with sleep discrepancy. It seems that a significant association between depressive symptoms and sleep discrepancy has more commonly been reported in studies that did not distinguish PARA and PSY subtypes (Bliwise, Friedman, & Yesavage, 1993; Fernandez-Mendoza et al., 2011; Williams, Kay, Rowe, & McCrae, 2013). There are several possible explanations of such contrary results. Studies that included PARA patients involved only the extreme cases of sleep discrepancy and excluded the rest of the sleep discrepancy continuum. Some of the studies used a cut-off score of Beck Depression Inventory (BDI) to exclude insomnia patients with more severe depression (Turcotte, St-Jean, & Bastien, 2011), which may have biased the results. Despite that, it seems that sleep discrepancy may be important not only to understand insomnia disorder, but it may also help to clarify the relation between insomnia and depression.

Authors, year	Subjects	Mood measures	Results
Liao et al. (2018)	63 PSY, 63 PARA, 63 GS	SAS, SDS	Both PSY and PARA showed higher anxiety and depression levels than GS. PARA had a higher level of depression and a slightly lower anxiety level than PSY.
Herbert, Pratt, Emsley, and Kyle (2017)	42 subjects with insomnia symptoms	Visual analogue scales measuring cognitive/physiological arousal, sleep effort, current state mood	Cognitive arousal and bad mood upon awakening were independently predictive of the total sleep time misperception index.
Normand et al. (2016)	24 PSY, 17 PARA, 29 GS	BDI, BAI	Both PSY and PARA showed higher levels of anxiety and depression than GS. There was no difference between PSY and the PARA group.
Moon et al. (2015)	66 PI with SM, 184 PI without SM	BDI, HDS, HAS	No difference in depression and anxiety symptoms between groups.
Perusse et al. (2015)	39 PSY, 27 PARA, 47 GS	BDI, BAI	PSY and PARA showed higher levels of anxiety and depression, but the scores were not clinically significant. There was no difference between PSY and PARA.
Bastien, Turcotte, St-Jean, Morin, and Carrier (2013)	30 PSY, 28 PARA, 30 GS	BDI, BAI	Both PSY and PARA showed higher levels of anxiety and depression, but the scores were not clinically significant. There was no difference between PSY and PARA.
Dittoni et al. (2013)	76 PI patients divided into a group with SM and group without SM	BDI, SAS	No difference in depressive symptoms, PI with SM showed lower anxiety severity.
St-Jean et al. (2013)	26 PSY, 21 PARA, 21 GS	BDI, BAI	PSY and PARA showed higher scores on BDI and BAI scale compared to GS, and there was no difference in depression or anxiety level between PSY and PARA.
Williams et al. (2013)	152 older adults with and without insomnia symptoms	BDI	Depressive symptoms were related to higher objective and subjective sleep discrepancy.
St-Jean et al. (2012)	17 PSY, 14 PARA, 19 GS	BDI, BAI	Both PSY and PARA reported higher scores on BDI and BAI scale compared to GS, and there was no difference in depression or anxiety level between PSY and PARA.
Fernandez-Mendoza et al. (2011)	1 741 PI patients divided into groups with and without SM	MMPI	PI with SM showed a profile indicating depression and rumination.
Sanchez-Ortuno et al. (2011)	332 subjects with insomnia	IDD, POMS	PARA group was consistently associated with lower daytime impairment. Mood disturbance was characteristic of insomnia associated with mental disorder and idiopathic insomnia.
Turcotte et al. (2011)	26 PSY, 26 PARA, 26 GS	BDI	Both PSY and PARA showed a higher score on BDI than GS, and there was no difference between PSY and PARA.

Tang and Harvey (2004)	20 subjects with PI with SM and 20 PI without SM	BDI, BAI	No difference in anxiety and depression symptoms between groups.
Vandeputte and de Weerd (2003)	917 adults from sleep disorders center	BDI	More than half of the patients in PSY (60%), PARA (63%), PLMD (53%), and inadequate sleep-wake hygiene (63%) group showed some form of depression (score ≥ 10).
Krystal et al. (2002)	30 PI (objective vs. subjective insomnia), 20 GS	BDI, STAI	No difference between groups in anxiety and depression symptoms
Edinger et al. (2000)	64 PI divided into subjective and objective insomnia groups, 61 GS	BDI, STAI	Subjective insomnia tended to report higher anxiety, lower mood, and more dysfunctional sleep-related cognitions than those with objective insomnia.
Vanable et al. (2000)	104 adults with ICSD insomnia diagnoses	MMPI	A positive correlation between sleep time underestimation and cognitive rumination, physiological symptoms of anxiety, catastrophic expectation, but not with a scale for depression.
Bonnet and Arand (1997)	9 PI with SM and 9 GS	MMPI, POMS	Patients with SM reported increased tension, confusion, anger, and depression (POMS) compared to GS, with no difference in the Depression scale of MMPI.
Edinger and Fins (1995)	173 adult insomnia patients	ICSD diagnosis	Insomnia with depression was among the insomnia subgroups most prone to show gross underestimates of TST.
Bliwise et al. (1993)	71 elderly subjects with poor sleep	GDS, STAI-S, MMSE	Depressed mood was associated with a tendency to underestimate the length of sleep.
Borkovec, Grayson, Obrien, and Weerts (1979)	11 pseudoinsomnia patients, 8 idiopathic insomnia patients	MMPI	No significant difference in Depression scale score between groups.

Table 4. Depressive symptoms in insomnia patients with sleep misperception. BAI: Beck Anxiety Inventory, BDI: Beck Depression Inventory, GDS: Geriatric Depression Scale, GS: good sleepers, HAS: Hospital Anxiety Scale, HDS: Hospital Depression Scale, ICSD: International Classification of Sleep Disorders, IDD: inventory to diagnose depression, MMPI: Minnesota Multiphasic Personality Inventory, MMSE: Mini-Mental State Exam, PARA: paradoxical insomnia, PI: primary insomnia, PLMD: periodic limbic movement, POMS: Profile of Mood States, PSY: psychophysiological insomnia, SAS: Self-Rating Anxiety Scale, SDS: Self-Rating Depression Scale, SM: sleep misperception, TST: total sleep time, STAI: State-Trait Anxiety Inventory, STAI-S: State Anxiety Scale of the State-Trait Anxiety Inventory. Note: subjective insomnia, pseudoinsomnia refer to insomnia with sleep misperception

3.3.3 REM sleep instability

Only a small number of studies has focused on underlying mechanisms of sleep discrepancy and depression relationship. The REM sleep instability hypothesis presents a possible common pathway to insomnia and depression in general (Riemann et al., 2012). This

concept follows PSG findings indicating that insomnia is often associated with a reduced amount of REM sleep and increased EEG arousals during this sleep stage (Feige et al., 2008). An overall hyperarousal may lead to a moderate reduction of REM sleep and its fragmentation in insomnia. The REM sleep fragmentation may cause an alteration of sleep perception through a REM sleep mentation being experienced as wake-like and focused on patients' main concerns instead of experiencing dreams, leading to a higher objective and subjective sleep discrepancy (Feige et al., 2018). Considering the relationship between insomnia and depression, Riemann et al. (2020) proposed that a sustained REM sleep reduction and fragmentation in insomnia may disturb emotion regulation processes and interfere with a limbic and paralimbic system. Indeed, the experiment by Wassing et al. (2019) has shown that interrupted REM sleep was associated with a dysregulation of amygdala activity in individuals with insomnia. When insomnia continues to persist together with the REM sleep alterations, a REM sleep rebound presented by a shorter REM sleep latency, increased REM density (sleep alterations often associated with depression), and enhanced response to negative stimuli may develop, which further facilitate the onset of depression (Riemann et al., 2020). These suggestions raise a question of whether the subjective and objective sleep discrepancy, together with the REM sleep alterations in insomnia patients, might be predictors of subsequent development of depressive disorder.

3.4 Sleep discrepancy and CBT-I

Although several studies have proven the long-term effect of CBT-I in individual or group form (Koffel et al., 2015; Morin et al., 2006), studies describing the exact mechanism of its effect on sleep are lacking. Moreover, because of the variability across subjective and objective sleep parameters in insomnia, there is not an exact definition of the optimal treatment outcome (Morin, 2003). Most of the studies measured the outcome by sleep diaries without objective sleep measures (van Straten et al., 2018). Nevertheless, to understand the mechanisms of CBT-I and the insomnia disorder itself, it is crucial to assess objective sleep quality as well. This chapter reviews the most relevant research on CBT-I and its effect on objective and subjective sleep parameters and sleep discrepancy measures.

3.4.1 Effect of CBT-I on sleep parameters

To date, studies assessing both subjective and objective sleep quality have shown a stronger impact of CBT-I on subjective compared to objective sleep measurements (Okajima, Komada, & Inoue, 2011). Findings of a recent meta-analysis indicate high effectiveness of CBT-I in improving subjective sleep continuity measures (SE, WASO, and SOL) and decreasing insomnia severity based on self-reported questionnaires, with the smallest effect on TST (van Straten et al., 2018).

Although objective sleep alterations often characterize insomnia in comparison with normal sleepers (C. Baglioni, Regen, W., Teghen, A., Spiegelhalder, K., Feige, B., Nissen, Ch., Reimann, D., 2014), the effect of CBT-I on objective sleep measures is much less explored. A recent meta-analysis by Mitchell et al. (2019) reported only five studies involving PSG measurement, concluding that these studies provide insufficient evidence for CBT-I impact on objective sleep parameters. Studies using actigraphy revealed a small effect on SOL and moderate negative effect on TST. The reduction of TST was the most robust effect found in this review (Mitchell et al., 2019), probably as a consequence of sleep restriction therapy aiming to reduce time spent in bed and consolidate sleep (Kyle et al., 2014). In line with these findings, our pilot study also showed decreased insomnia severity and increased subjective sleep quality, although the objective TST measured by actigraphy tended to be shorter in insomnia patients after CBT-I (Veldova, Buskova, & Koprivova, 2019). However, it should be noted that only a limited number of studies explored other sleep parameters than those associated with sleep continuity (SOL, TST, SE, and WASO), such as changes in sleep architecture or cortical activity during sleep (Cervena et al., 2004), which might underlie changes of subjective sleep parameters.

3.4.2 Effect of CBT-I on sleep discrepancy

One of the possible explanations of CBT-I efficacy might be the correction of sleep discrepancy. Kay et al. (2015) showed a significant reduction of subjective overestimation of WASO and SOL after CBT-I in a group of older adults with insomnia. A study involving older adults with comorbid insomnia showed that CBT-I decreased a negative sleep discrepancy of SOL and TST with a minimum effect on objective sleep parameters measured by PSG (Lund,

Rybarczyk, Perrin, Leszczyszyn, & Stepanski, 2013). The change in sleep discrepancy was associated with a reduction of the NREM 1 sleep stage after the treatment (Lund et al., 2013). A recent study by Dzierzewski et al. (2019) confirmed the correction of sleep and wake subjective estimations in older adults with insomnia receiving CBT-I. The authors also reported the association between self-reported overall sleep quality and changes in sleep and wake time discrepancies. Another recent study explored the impact of 14 days inpatient CBT-I programme involving education about sleep misperception, demonstrated a significant improvement in sleep duration estimation (Cronlein et al., 2019). Other therapeutic techniques, apart from CBT-I, may also be beneficial. For example, a behavioural experiment of Tang and Harvey (2004) showed promising results of reduction in sleep misperception by allowing participants to compare their self-reported estimates with actigraphic recordings.

Although CBT-I seems to be effective in correcting sleep discrepancies, the number of studies is sparse with no study assessing sleep discrepancy during CBT-I to see the progress in time. Moreover, most of the studies involved older adults, albeit insomnia is common also in the middle age working adults. Another gap in the CBT-I research is related to the exploration of the treatment response in different insomnia subtypes. Since not all patients with insomnia show negative sleep discrepancy (Te Lindert et al., 2020), the treatment outcome cannot be explained by the correction of sleep misperception in all individuals experiencing insomnia. Studies exploring treatment response in insomnia subtypes with different sleep perceptions are missing to clarify the treatment outcome.

3.4.3 Additional therapeutic tools

Despite the good-quality evidence proving the efficacy of CBT-I (Morin et al., 2006), only about 60 % of treated patients show a clinically significant response, and only about 40 % of these patients fully remit (Morin et al., 2009). Considering also the blunted impact of CBT-I on objective sleep measures (Okajima et al., 2011), it is essential to examine other therapeutic interventions that could further enhance the CBT-I efficacy. It is also a clinically relevant need to examine alternative interventions to CBT-I or sleep restriction as in some patients the sleep restriction therapy can lead to sleep deprivation and related adverse side effects, such as increased sleepiness or significantly reduced sleep duration (Kyle et al., 2014).

Since insomnia is associated with circadian rhythm disruption, interventions aiming to support circadian rhythm might be beneficial for insomnia patients and may support the overall CBT-I outcome. Albeit CBT-I focuses on a regular sleep schedule and sleep hygiene, a set of behavioural and environmental recommendations (Morin et al., 2006) related to caffeine and alcohol use, or physical activity (Irish, Kline, Gunn, Buysse, & Hall, 2015), it usually does not include any particular intervention to reduce the impact of light on patients' sleep. Only a set of recommendations on how to alleviate the negative impact of artificial light from night exposure to screen on sleep quality is usually provided. Specifically, patients should avoid the night time screen exposure as the light can have an impact on the circadian system by suppressing melatonin secretion, leading to a phase-delaying effect of light together with increasing cortical arousal (Rodriguez-Morilla, Madrid, Molina, & Correa, 2017). Another recommendation may be to use a particular mobile or PC application with blue light filtration properties, although their research application is sporadic (Heath et al., 2014), and it does not impact the light from LED bulbs.

Several studies have demonstrated that wearing a blue-light blocking glasses in the evening may be a promising tool to support sleep and circadian regulation (Ayaki et al., 2016). Some studies have already assessed the impact of these tools on sleep in mental disorders, such as major depressive disorder or bipolar disorder (Esaki et al., 2017; Henriksen et al., 2016). So far, two studies have focused on blue-light blocking glasses in insomnia, showing a positive effect on sleep quality (Shechter et al., 2018) and neuropsychological functioning (Zimmerman et al., 2019). This simple and potentially effective intervention may provide a promising tool for enhancing the effect of CBT-I on objective sleep parameters as well as on sleep discrepancy through decreasing arousal (van der Lely et al., 2015).

II. EXPERIMENTAL PART

The experimental part aimed to: (a) explore EEG correlates of sleep discrepancy in different insomnia patients with sleep misperception; (b) assess whether this phenomenon might be associated with psychopathology commonly observed in insomnia patients; (c) examine changes of sleep discrepancy during and after the therapy of insomnia. Four studies were conducted with the following goals:

1. To compare sleep characteristics in different insomnia subtypes with sleep discrepancy and to assess EEG correlates of sleep misperception (Janku et al., under review; Study 1)

2. To explore the association between depressive symptoms and sleep discrepancy in patients with insomnia (Study 2).

3. To examine the CBT-I effect on objective and subjective sleep discrepancy after the therapy as well as during the entire programme (Janku, Smotek, Farkova, & Koprivova, 2020b; Veldova et al., 2019; Study 3).

4. To assess the impact of CBT-I and additional chronotherapeutic intervention on sleep parameters and sleep discrepancy (Janku, Smotek, Farkova, & Koprivova, 2020a; Study 4)

4 SLEEP DISCREPANCY IN DIFFERENT INSOMNIA SUBTYPES (STUDY 1)

4.1 Aims and hypotheses

Study 1 aimed to compare groups with similar objective sleep continuity parameters according to PSG, which differed in the presence of sleep misperception (PARA vs. GS; PSY/MIS vs. PSY), and to assess sleep EEG correlates of sleep discrepancy. The observed measures included objective sleep parameters, such as sleep continuity measures, the proportion of sleep stages, relative PSA of sleep EEG, and subjective sleep parameters. The results of this study were processed into a publication that is currently under review (Janku et al., under review).

We expected to find:

1. Signs of higher cortical arousal (higher beta, lower delta EEG activity) during NREM 2 sleep in groups with sleep misperception (PSY/MIS and PARA) compared to their counterpart groups (PSY, GS) with an accurate subjective estimate of sleep (Krystal et al., 2002; Spiegelhalder et al., 2012).

2. A positive relationship between the degree of sleep discrepancy and the amount of beta EEG during NREM 2 sleep and a negative relationship between sleep misperception and the amount of delta activity during NREM 2 sleep (Krystal et al., 2002).

3. A positive correlation between sleep discrepancy and the proportion of REM sleep, which would be in line with the REM sleep instability hypothesis (Riemann et al., 2012).

4.2 Materials and methods

Participants

Study 1 involved 29 patients diagnosed with primary insomnia who underwent a PSG recording in the Prague Psychiatric Centre (now the National Institute of Mental Health; NIMH)

between 2011 and 2016, and were retrospectively selected from a database of insomnia patients. Insomnia diagnosis was established according to the 10th edition of the International Classification of Diseases (ICD-10; WHO 2004). All patients fulfilling the following inclusion criteria were included in the study: (a) subjective complaints of difficulties initiating and/or maintaining sleep, or poor sleep quality; (b) the sleep disturbance has occurred at least three times per week for at least one month; (c) preoccupation with the sleeplessness and excessive concern over its consequences at night and during the day; (d) the unsatisfactory quantity and/or quality of sleep either causes marked distress or interferes with ordinary activities in daily living. Because of the aim to compare PSA of NREM 2, NREM 3 and REM sleep stages along with the misperception of TST, subjects with sleep-maintenance insomnia were studied, rather than those with sleep onset problems. Table 5 presents the criteria for distinguishing insomnia subtypes (Perusse et al., 2015).

Group	**Objective TST**	**Sleep discrepancy**
PSY	Objective TST < 6 hours	Discrepancy between obj. and subj TST < 60 minutes
PARA	Objective TST ≥ 6 hours	Discrepancy between obj. and subj. TST ≥ 60 minutes
PSY/MIS	Objective TST < 6 hours	Discrepancy between obj. and subj. TST ≥ 60 minutes

Table 5. Criteria for distinguishing insomnia subtypes (Perusse et al., 2015). PSY: Psychophysiological insomnia, PARA: Paradoxical insomnia, PSY/MIS: Psychophysiological insomnia with sleep misperception, TST: Total sleep time.

Exclusion criteria for all patients were: (a) other comorbid sleep disorder; (b) neuropsychiatric disorder present or in the anamnesis; (c) poor-quality PSG records; (d) usage of medication known to affect sleep at least two weeks before PSG recording (e.g., benzodiazepines) and four weeks in the case of SSRI, usage of other medication known to affect sleep at a shorter time before PGS than the length of the wash-out period (4.5 x the timeout of the elimination of medication), or more than two years of regular usage.

Participants from the GS group were recruited via internet advertising. After screening the inclusion criteria, participants completed one PSG night in the sleep laboratory. Two or more

PSG nights could not be conducted due to a retrospective study design. Participants in the GS group had to be without subjective sleep complaints, as well as without objective sleep difficulties (i.e., SOL or awakenings of more than 30 minutes or a TST of less than 6 hours), with SE of 85 % or more. Exclusion criteria also included the presence of other sleep or neuropsychiatric disorders. Participants had to be without the usage of medication known to affect sleep. Table 6 summarizes the final sample characteristics.

Group	PSY/MIS	PSY	PARA	GS
n	9	9	11	9
Female/Male	4/5	4/5	4/7	3/6
Mean age	45.89 (14.44)	46.33. (11.47)	35.09 (7.68)	36.22 (15.05)
Length of insomnia (years)	2.32 (2.18)	5.33 (4.05)	3.26 (4.28)	n/a
Education (%)				
High school	44	44	46	56
University degree	56	56	54	44
Married (%)	44	67	36	56

Table 6. Sociodemographic characteristics of psychophysiological insomnia with sleep misperception (PSY/MIS), psychophysiological insomnia (PSY), paradoxical insomnia (PARA), and good sleeper control group (GS). Means (SD) are reported.

Full-night PSG

A full-night PSG was recorded from 22:00 (lights-out) to 06:00 (lights-on) by a Brainscope PSG system (Unimedis, Ltd., Czech Republic). All recordings included C3-A2, C4-A1, electrooculography (EOG), electromyography (EMG; submental, mm. tibiales ant.), electrocardiography (ECG), respiratory events, and video-monitoring. Experienced raters visually scored the records according to the AASM criteria at 30-second epochs (AASM, 2007). Sleep macrostructure variables included TST, SOL, WASO, SE (SE percentage = TST / time in bed), and a number of awakenings (NWAKE), proportion (%) of REM, NREM 1, NREM 2, and NREM 3 sleep stages.

Self-reported scales and questionnaires

All participants completed the Insomnia Severity Index (ISI; Bastien et al., 2001) to portray insomnia difficulties; the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) to assess sleep habits and sleep quality in the preceding two weeks; the Epworth Sleepiness Scale (ESS; Johns 1991) to assess daytime sleepiness; the Beck Depression Inventory-2 (BDI-II; Beck 1996); and the Beck Anxiety Inventory (BAI; Beck and Steer 1993) to assess symptoms of depression and anxiety. All subjects also evaluated their sleep after the night in the sleep laboratory, including TST, SOL, SE, and NWAKE.

Misperception Index

The misperception index (MI) was computed (MI = [objective TST – subjective TST] / objective TST) to quantify the degree of sleep discrepancy. The positive MI value reflects the underestimation of TST, while the negative value indicates an overestimation of TST, with a value of 0 for perfect estimation. MI is supposed to provide a reliable and immediate description of sleep misperception (Manconi et al., 2010).

Spectral analysis (PSA)

Spectral analysis was performed on the sleep EEG during NREM 2, NREM 3, and REM sleep. Due to a retrospective study design, we involved segments of NREM 2 and NREM 3 sleep stages only from the first sleep cycle, and REM sleep segments from the last sleep cycle. The reason for that was to enhance the probability of finding artifact-free segments as there is a higher proportion of NREM sleep during the first half of the night and enhanced REM sleep proportion at the end of the night (Kupfer, 2006; Maes et al., 2014).

Fast-Fourier transformation on 2.0-s windows with 1-s overlap (Richards et al., 2013; Welch, 1967), was applied on artifact-free data, and relative power spectra within delta (1-4 Hz), theta (4-7 Hz), alpha (7-11 Hz), sigma (12-16 Hz), beta 1 (14-20 Hz) and beta 2 (20-35 Hz) frequency bands were computed. For the relative PSA, both C3 and C4 channels were used (Perlis, Smith, Andrews, Orff, & Giles, 2001).

Statistical analysis

Statistical analysis was performed using IBM SPSS version 23. Due to non-parametric data distribution, the Mann-Whitney test was conducted to assess differences between groups in subjective sleep parameters. Although the age differences between groups analyzed by Kruskal-Wallis test were not significant, $H(3) = 6.71$, $p = .082$, due to a known effect of age on objective sleep parameters, univariate analysis of covariance (ANCOVA) was run to compare these parameters, using age as a covariate. Because of non-parametric distribution, we had transformed the data into the ranks before the ANCOVA (Quade, 1967). We did not involve a comparison of all four groups because of the large age differences (Table 1). The possible usage of multivariate analysis of covariance (MANCOVA) may not be sufficient to eliminate the effect of age. Linear regression analyses were conducted to evaluate the associations between subjective and objective sleep characteristics. Age was set as a second independent variable.

4.3 Results

Objective and subjective sleep parameters

Tables 7 and 8 present subjective evaluations of sleep quality, daytime symptoms, and subjective and objective sleep parameters. According to the ISI scores, the PSY/MIS group evaluated the insomnia symptoms as more severe than the PSY group did. There was no significant difference in other questionnaires between these two groups. The PARA group evaluated the insomnia symptoms and sleep disturbances as significantly worse than the GS group, according to ISI and PSQI scores. This group also showed significantly lower levels of daytime sleepiness, according to the ESS. The PSY/MIS complained about a significantly longer subjective SOL, shorter subjective TST, and lower SE than the PSY group. There was no significant difference in NWAKE between these two groups. The PARA group showed a significantly longer subjective SOL, shorter subjective TST, and lower subjective SE than the GS group.

As expected, PSY and PSY/MIS did not differ in objective sleep parameters (SOL, TST, NWAKE, WASO, and SE). The same results were found comparing the PARA group with the GS group. In the case of sleep macrostructure, the only significant difference between PSY and

PSY/MIS group was found in the amount of REM sleep, which was significantly lower in PSY/MIS. The same difference was found between PARA and GS, with a decreased period of REM sleep in the PARA group. The PARA group also showed a significantly lower proportion of the NREM 3 sleep stage and a higher proportion of the NREM 1 sleep stage compared to the GS group.

	PSY/MIS	**PSY**	***p***	**PARA**	**GS**	***p***
Questionnaires						
ISI	17.44 (3.50)	13.75 (2 .57	**.024**	16.18 (4.09)	4.90 (2.39)	**.000**
PSQI	11.11 (3.35)	9.11 (2.51)	.113	11.36 (2.71)	5.00 (0.45)	**.000**
ESS	7.89 (4.28)	10.44 (5.58)	.489	7.18 (3.01)	10.90 (2.59)	**.020**
BAI	8.67 (4.08)	5.83 (3.62)	.340	6.73 (3.82)	6.20 (3.46)	.882
BDI-II	8.89 (3.11)	6.56 (4.30)	.136	5.73 (3.62)	7.90 (5.96)	.710
Subjective sleep measures						
SOL (min)	152.22 (42.11)	41.67 (32.92)	**.000**	59.00 (41.57)	26.67 (13.54)	**.000**
TST (min)	174.44 (30.23)	322.22 (36.31)	**.000**	264.55 (91.19)	376.67 (40.28)	**.010**
NWAKE	3.00 (1.33)	4.44 (2.74)	.297	5.09 (4.01)	4.33 (1.15)	.378
SE %	44.31 (9.56)	71.00 (9.02)	**.000**	54.41 (18.65)	89.67 (4.62)	**.000**
MI	0.40 (0.09)	- 0.15 (0.28)	**.000**	0.35 (0.26)	0.04 (0.09)	**.000**

Table 7. Differences in subjective sleep parameters and daytime symptoms between insomnia patients and good sleepers. Results of the Mann-Whitney test are reported. Means (SD) are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). PSY/MIS: psychophysiological insomnia with sleep misperception, PSY: psychophysiological insomnia, PARA: paradoxical insomnia, GS: and good sleepers, ISI: Insomnia Severity Index, PSQI: Pittsburgh Sleep Quality Index, ESS: Epworth Sleepiness Scale, BAI: Beck Anxiety Inventory, BDI: Beck Depression Inventory SOL: sleep onset latency, TST: total sleep time, NWAKE: number of awakenings, SE: sleep efficiency, MI: misperception index.

	PSY/MIS	PSY	*F*	*p*	PARA	GS	*F*	*p*
SOL (min)	26.97 (14.27)	26.48 (26.79)	.502	.490	12.50 (5.74)	16.13 (9.92)	.583	.456
TST (min)	289.78 (34,29)	296.22 (58.50)	.676	.424	410.50 (30.48)	398.06 (27.13)	.505	.487
NWAKE	41.78 (15.22)	37.85 (23.14)	1.873	.191	23.35 (10.04)	13.44 (7.47)	1.908	.185
WASO %	29.00 (0.09)	25.16 (0.13)	1.932	.185	8.43 (0.03)	6.23 (0.05)	1.148	.299
SE %	71.00 (0.09)	74.71 (0.13)	1.932	.185	91.57 (0.03)	93.77 (0.05)	1.148	.299
NREM1 %	15.76 (0.05)	13.24 (0.05)	1.391	.257	8.28 (0.03)	2.47 (0.01)	20.603	**.000**
NREM2 %	34.43 (0.09)	38.88 (0.14)	.444	.515	45.22 (0.07)	49.81 (0.05)	3.311	.086
NREM3 %	12.02 (0.06)	10.06 (0.05)	1.200	.291	16.80 (0.05)	24.09 (0.07)	4.739	**.044**
REM %	7.31 (0.04)	12.18 (0.05)	5.600	**.032**	15.96 (0.05)	21.73 (0.04)	5.524	**.031**

Table 8. Differences in objective sleep parameters in insomnia patients and good sleepers. Results of the ANCOVA analysis are reported. Means (SD) are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). PSY/MIS: psychophysiological insomnia with sleep misperception, PSY: psychophysiological insomnia, PARA: paradoxical insomnia, GS: and good sleepers, SOL: sleep onset latency, TST: total sleep time, NWAKE: number of awakenings, WASO: wake after sleep onset, SE: sleep efficiency, NREM: non-rapid eye movement sleep, REM: rapid eye movement sleep.

Sleep EEG activity

Table 9 shows the mean values of relative PSA at central sites in PSY/MIS, PSY, PARA, and GS groups and the results of the ANCOVA. Comparison analyses revealed no differences in spectral power during sleep between the groups. However, delta EEG activity during NREM 3 sleep tended to be lower in the PSY/MIS compared to the PSY group ($F = 4.789$; $p = .056$).

	PSY/MIS	PSY	*F*	*p*	PARA	GS	*F*	*p*
NREM 2								
Delta	0.58 (0.11)	0.63 (0.12)	.068	.800	0.61 (0.12)	0.62 (0.08)	.004	.951
Beta 1	0.04 (0.02)	0.04 (0.04)	.020	.890	0.05 (0.03)	0.05 (0.02)	.258	.618
Beta 2	0.03 (0.03)	0.01 (0.01)	1.397	.267	0.03 (0.04)	0.03 (0.03)	.571	.460
NREM 3								
Delta	0.71 (0.11)	0.75 (0.10)	4.789	.056	0.77 (0.08)	0.77 (0.07)	.011	.918
Beta 1	0.02 (0.01)	0.01 (0.01)	.016	.901	0.02 (0.01)	0.02 (0.01)	.008	.931
Beta 2	0.01 (0.01)	0.01 (0.00)	1.175	.307	0.01 (0.01)	0.01 (0.01)	1.815	.196
REM								
Delta	0.5 (0.13)	0.58 (0.12)	2.759	.131	0.53 (0.1)	0.55 (0.08)	.324	.577
Beta 1	0.05 (0.02)	0.04 (0.02)	.021	.889	0.05 (0.04)	0.05 (0.01)	.192	.667
Beta 2	0.04 (0.01)	0.03 (0.01)	.627	.449	0.06 (0.08)	0.04 (0.02)	.319	.579

Table 9. Differences in sleep EEG activity during NREM and REM sleep. Power spectral analysis: mean values (SD) of relative powers at central sites during NREM 2, NREM 3, and REM sleep stages; ANCOVA results are presented. Relative power was computed as the power within a frequency band (in μV2/Hz) divided by the power across all frequencies (1-35 Hz) (also in μV2/Hz). PSY/MIS: psychophysiological insomnia with sleep misperception, PSY: psychophysiological insomnia, PARA: paradoxical insomnia, GS: and good sleepers, NREM: non-rapid eye movement sleep, REM: rapid eye movement sleep.

Correlates of sleep discrepancy

In all four groups, the proportion of REM sleep was negatively related to MI (β = -0.36; p = .016) and positively related to subjective TST (β = 0.78; p <. 000). After the exclusion of the GS group, the negative correlation between REM proportion and MI did not remain significant (β = -0.34; p = .091). Nevertheless, the relation between REM proportion and subjective TST was still significant (β = 0.69; p < .000). The analysis did not reveal any significant correlations between PSA parameters and subjective sleep measures.

5 SLEEP DISCREPANCY IS RELATED TO DEPRESSIVE SYMPTOMS IN INSOMNIA PATIENTS (STUDY 2)

5.1 Aims and hypotheses

Study 2 aimed to answer the question of whether insomnia patients with depressive symptoms show a higher degree of sleep discrepancy compared to insomnia patients without depressive symptoms. The second aim was to explore whether the REM sleep proportion contributes to sleep discrepancy and depressive symptoms in patients with insomnia. Based on the reviewed literature, we did not distinguish PSY and PARA patients because studies using this methodology did not report differences in depression (see chapter 2.3.2. Sleep discrepancy and depression).

We expected to find:

1. A higher degree of TST discrepancy in insomnia patients with depressive symptoms.
2. A positive correlation between the degree of sleep discrepancy and the severity of depressive symptoms (Tsuchiyama et al., 2003).
3. A negative correlation between the REM sleep proportion and sleep discrepancy (based on the results of Study 1).
4. A negative correlation between the REM sleep proportion and depressive symptoms.

5.2 Materials and methods

Participants

Participants were retrospectively selected from a database of 141 insomnia patients who completed one night of PSG at the NIMH, Czech Republic, between the years 2016 and 2019. Patients were diagnosed with insomnia by physicians, according to the ICD-10 (WHO, 2004). The inclusion criteria were: (a) age 18 to 70; (b) absence of severe comorbid psychiatric, neurological or somatic disease; (c) no usage of medication affecting sleep. Exclusion criteria were (a) night shift employment; (b) age over 70 years.

Subjective scales and questionnaires

Questionnaires involved in the final analyses were BDI-II, BAI, and ESS to evaluate symptoms of depression, anxiety, and daytime sleepiness. Based on established cut-off scores (A. T. Beck, Steer, R. A., Brown, G.K., 1996), different severity of depressive symptoms was evaluated: (a) 0-13 minimal depression; (b) 14-19 mild depressive symptoms; (c) 20-28 moderate depressive symptoms; (d) 29-63 severe depressive symptoms. Anxiety severity was assessed according to the following cut-off scores (A. T. Beck & Steer, 1993): (a) 0-7 minimal anxiety; (b) 8-15 mild anxiety; (c) 16-25 moderate anxiety; (d) 26-63 severe anxiety. In the morning, all patients answered questions about the subjective sleep quality of the previous night in the sleep laboratory. The subjective sleep variables included SOL, TST, and SE. The misperception index was computed for the TST variable.

Polysomnography

A whole-night PSG was recorded. All recordings included EEG according to the 10/20 standard system, EOG, EMG (three submental electrodes), ECG, and video monitoring. Data were recorded using Brainscope polysomnography system (M&I spol. s.r.o., Czech Republic). The records were visually scored by experienced clinicians, according to the American Academy of Sleep Medicine criteria at 30-second epochs (AASM, 2007). Sleep macrostructure variables included TST, SOL, WASO, SE, proportion (%) of REM, NREM 1, NREM 2, and NREM 3 sleep stages and the REM sleep onset latency.

Statistical analysis

Statistical data analysis was performed using IBM SPSS version 23. Chi-squared test was used to compare group differences in sex composition and proportion of participants with different severity of depression (according to the BDI-II score) and anxiety (according to the BAI score). Independent t-tests and Mann-Whitney U test was used to assess differences between the groups. Based on the data distribution, Pearson's r or Spearman's r_s was computed to assess the correlation between the MI, BDI, and REM sleep proportion.

5.3 Results

Sample characteristics

Eighty-eight patients with insomnia fulfilling the inclusion criteria were included in the final analysis. A total number of 53 patients had to be excluded: 30 patients were using medication affecting sleep, ten patients were over 70 years old, five patients had severe comorbidities, one person worked in nigh-shifts, and six patients did not fill in the BDI-II. To compare the two groups differing in depressive symptoms, we further divided the sample into two groups according to the established cut-off score (A. T. Beck, Steer, R. A., Brown, G.K., 1996), distinguishing minimal and mild depressive symptoms: (1) insomnia without depressive symptoms (BDI-II ≤ 13; INS); (2) insomnia with depressive symptoms (BDI-II > 13; INS-D). In the INS-D group, 25 patients reported mild depression, ten patients reported moderate depression, and seven participants reported severe depression.

The total sample and the two subgroups characteristics are presented in Table 10. Apart from a significant difference in the BDI-II score, the two groups also significantly differed in the BAI score, which showed higher anxiety symptoms in the INS-D group. The INS-D group involved a significantly lower number of participants reporting minimal anxiety and a higher number of participants with moderate anxiety compared to the INS group. There was no significant difference in the ESS score between the two groups.

	Total sample	INS	INS-D	*t / χ2/ U*	P-value
N	88	46	42		
Age †	40.29 (14.09)	44 (14.23)	38.78 (12.68)	1.808	.074
Sex, female (%)	47.4	39.13	54.76	2.156	.142
BDI-II ¥	13.57 (9.92)	6.28 (4.26)	21.57 (7.99)	0.000	**.000**
BAI ¥	10.72 (9.71)	6.84 (9.71)	14.97 (7.81)	337	**.000**
ESS †	8.75 (5.4)	7.73 (4.93)	9.75 (5.74)	-1.765	.081
Anxiety severity based on BAI (n):					
Minimal anxiety	35	28	7	17.908	**.000**
Mild anxiety	15	17	14	0.587	.443
Moderate anxiety	13	0	13	16.706	**.000**
Severe anxiety	6	1	5	3.272	.070

Table 10. Clinical and sleep characteristics of the total sample, INS, and INS-D group. Results of independent t-tests, chi-squared test, and Mann-Whitney U test are presented. Means (SD) are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). INS: insomnia patients without depressive symptoms, INS-D: insomnia patients with depressive symptoms, BDI-II: Beck Depression Inventory, BAI: Beck Anxiety Inventory, ESS: Epworth Sleepiness Scale.
† Results of independent t-tests are presented.
¥ Results of the Mann-Whitney U test are presented.

Subjective and objective sleep characteristics

As summarized in Table 11, only a difference in subjective TST was close to the significance threshold (p = .055), indicating shorter subjective TST in the INS-D group. No significant difference in any other subjective and objective sleep continuity parameters (SOL, TST, WASO, or SE) was reported, nor in sleep architecture (REM, NREM 1, NREM 2, NREM 3 and REM sleep onset latency). A significantly higher MI TST was observed in the INS-D compared to the INS group.

	Total sample	INS	INS-D	*t / U*	P-value
Subjective sleep parameters					
SOL (min.) ¥	57.59 (48.39)	57.06 (55.16)	60.33 (42)	641.5	.307
TST (min.)	305.65 (125.63)	323.97 (112.67)	272.19 (132.61)	1.944	.055
SE (%)	63.36 (30.69)	65.18 (28.51)	57.2 (32.42)	1.164	.248
Polysomnography					
SOL (min.) ¥	28.32 (35.02)	28.437 (31.07)	29.43 (38.6)	957	.805
TST (min.) ¥	352.91 (88.52)	351.88 (76.03)	344.31 (102.29)	973	.908
WASO (%) ¥	18.98 (15.83)	17.64 (10.41)	21.32 (20.97)	970	.889
SE (%) ¥	80.86 (15.3)	81.24 (12.71)	79.53 (18.67)	956.5	.802
REM (%)	16.09 (6.79)	16.86 (6.5)	15.45 (7.25)	0.968	.336
REM sleep latency (min.)	114.93 (62.41)	124.79 (69.73)	103.09 (54.25)	1.593	.115
NREM 1 (%) ¥	5.94 (3.25)	6.27 (3.37)	5.58 (3.31)	893	.44
NREM 2 (%)	42.99 (10.92)	43.66 (10.16)	41.94 (12.34)	0.72	.473
NREM 3 (%)	17.45 (6.52)	16.93 (6.17)	17.32 (6.48)	-0.285	.775
MI TST	0.14 (0.31)	0.07 (0.31)	0.23 (0.3)	-2.389	**.019**

Table 11. Objective and subjective sleep measures in the total sample, INS, and INS-D groups. Results of independent t-tests. Means (SD) are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). INS: insomnia patients without depressive symptoms, INS-D: insomnia patients with depressive symptoms, SOL: sleep onset latency, TST: total sleep time, WASO: wake after sleep onset, SE: sleep efficiency, REM: rapid eye movement, NREM: non-rapid eye movement, MI: Misperception Index.
¥ Results of the Mann-Whitney U test are presented.

Correlations

A significant positive correlation was found between the BDI-II score and MI TST, r_s = .267, p = .013. Because of the higher degree of anxiety symptoms in the INS-D group compared to INS, we also explored whether the BAI score is related to the MI TST. No significant relationship was observed, r_s = .175, p = .109. There was a significant negative correlation between REM sleep proportion and MI TST, r = -.346, p = .001. The BDI-II score was not significantly related to REM sleep proportion, r_s = -.12, p = .265.

6 SLEEP DISCREPANCY DURING AND AFTER CBT-I (STUDY 3)

6.1 Aims and hypotheses

The aim of Study 3 was to explore changes in the subjective and objective discrepancy of SOL, TST, and WASO after CBT-I in adults with insomnia. Moreover, we aimed to assess changes in TST discrepancy during the entire therapeutic programme. Since not all insomnia patients underestimate their sleep quantity and the correction of sleep misperception cannot explain the treatment effect in all patients with insomnia, another objective was to assess the effect of CBT-I in two different insomnia subgroups defined by the presence or absence of negative sleep discrepancy. The results of Study 3 has been published in a peer-reviewed impacted journal. The details of the study can be found in Janku, Smotek, Farkova, and Koprivova (2020b).

We expected to find:

1. Reduced TST, SOL, and WASO discrepancy after CBT-I in patients who show negative sleep discrepancy (underestimation of TST) at the beginning of treatment (Kay et al., 2017; Lund et al., 2013).

2. A first significant change of TST discrepancy in the second week of treatment due to a sleep restriction implementation and continuation of this change throughout the weeks of therapy because of other CBT-I interventions.

3. No changes in sleep discrepancy measures in a subgroup of patients without a tendency to underestimate sleep duration.

6.2 Materials and methods

Participants

Fifty patients diagnosed with insomnia were recruited at the Department of Sleep Medicine of the NIMH, Czech Republic, and enrolled in the CBT-I group programme. Insomnia

diagnosis was established according to the ICD-10 (WHO 2004). Inclusion criteria involved: (a) a minimum age of 18; (b) absence of severe comorbid psychiatric, neurological or somatic disease; (c) motivation to complete the CBT-I programme; (d) no or stable use of medication affecting sleep. Exclusion criteria were: (a) discontinued CBT-I programme; (b) previous experience with CBT-I without effect; (c) night shift employment.

Baseline measures

At the beginning of the CBT-I programme, all patients completed a battery of self-reported questionnaires: ISI; ESS; the Hyperarousal Scale (HAS), empirically designed to measure daytime alertness, reflecting the enhanced arousal with the cut-off score > 40 (Regestein, Dambrosia, Hallett, Murawski, & Paine, 1993); and a modified version of the brief World Health Organization Quality of Life questionnaire (QOL; Harper et al., 1998) to measure patients' quality of life. Participants filled in the same battery at the end of CBT-I.

Sleep diary

Patients were asked to complete a sleep diary every day during the six weeks of therapy. The questions included information about daily lights-out time, waking and rising times, self-reported estimates of SOL, a number of nocturnal awakenings, and WASO. Items also recorded sleep medication and rated sleep quality and daytime tiredness. The primary outcomes were average SOL, TST, WASO, and SE for every week. TST was calculated by subtracting the total time spent awake (SOL, WASO, and time spent awake before getting out of bed) from the total time in bed. The leading therapist analyzed diaries weekly.

Actigraphy

Objective sleep measurement was performed by actigraphic monitoring using a wristwatch-like device. Actigraphy provides the detection and quantification of a person's movement to assess sleep patterns objectively. The agreement with PSG is about 88 % (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992). For the current study, the MotionWatch 8 (CamNtech Ltd., Cambridge, UK) actigraphic watch was used. Patients received the devices at the beginning of CBT-I and wore them on their non-dominant wrist continuously for six consecutive weeks. Patients were asked to press the event marker every time they got in or out of bed. The data were downloaded and analysed using MotionWare 1.4 software. In the analyses of records, the

recommended algorithm for sleep scoring every 60 s epoch was used (CamNtech, 2012). Event markers determined the length of time spent in bed. In the case of missing event markers, sleep diary data were used. The extracted outcomes were the same as for sleep diaries.

MI was calculated to quantify the objective and subjective discrepancy of TST. SOL and WASO discrepancy were obtained by computing the difference between self-reported SOL/WASO and objective SOL/WASO (Herbert et al., 2017). Negative values reflect self-reported underestimation compared to objective measures, whereas positive values represent overestimation compared to objective findings.

Cognitive behavioural therapy for insomnia

Two psychologists trained in CBT-I led the therapeutic programme. The CBT-I lasted for six weeks and consisted of one 2-hour session per week, with a maximum of eight patients per group. Each session had a specific structure according to the recommendations of the clinical manual for insomnia treatment (Morin and Espie, 2003; see Table 11). The first week of the therapy occurred without intervening in patients' sleep schedules and thus served as a baseline. Sleep restriction was implemented in Week 2. Patients were allowed to spend the same amount of time in bed as their average TST during the previous week. The minimum length was set at 5 h. The sleep window was titrated every week, according to average SE. If the SE was 85% or higher, then the time in bed was prolonged by 15 min. Otherwise, time remained the same for another week.

Psychoeducation was provided at the beginning of each session. Stimulus control therapy was set up in Week 3. The recommendations involved the following: (a) leaving the bed if one cannot fall asleep within 20 min, performing a pleasant and relaxing activity in a different room, and coming back to bed when feeling sleepy; (b) avoiding naps; (c) only using the bed and bedroom for sleep and sex. The last three sessions mainly focused on cognitive therapy (identification and reduction of dysfunctional beliefs about sleep, insomnia, and its consequences).

1. session	Introduction of CBT-I, education about development and maintenance of insomnia
2. session	Setting the goals of therapy, education about circadian and homeostatic regulation of sleep, sleep restriction
3. session	Education about sleep architecture, hyperarousal, relaxation, stimulus control
4. session	Education about the vicious cycle of insomnia and dysfunctional beliefs about sleep
5. session	Cognitive restructuring
6. session	Cognitive restructuring, relapse prevention, individualized recommendations

Table 11. Structure of CBT-I sessions. CBT-I: cognitive behavioural therapy for insomnia

Statistical analysis

Statistical analyses were performed using IBM SPSS (IBM Corporation, USA). Independent t-tests were used to assess differences in baseline sociodemographic, clinical, and sleep parameters as well as in a number of CBT-I sessions completed. Chi-squared test was used to compare group differences in the sex composition and proportion of participants with different types of insomnia complaints, insomnia severity (according to ISI score), significant daytime sleepiness (based on ESS score), and hyperarousal (based on HAS score), marital status and education. Based on data distribution, paired-samples t-tests or Wilcoxon signed-rank tests were used to assess differences before and after CBT-I in the total sample. Cohen's *d* was computed for effect size (Lakens, 2013).

Two-way mixed analysis of variance (ANOVA) was used to assess changes before and after the treatment within each group. For each dependent variable, Group (underestimating and accurate/overestimating) was set as a between-subject factor, and Time as a within-subject factor (pre- and post-treatment). The main effects for Group, Time, and the interaction of Group and Time were assessed. Partial eta square (η^2) was used for effect size (Vacha-Haase & Thompson, 2004). To assess changes of MI during the therapy (from Week 1 to Week 6), one-way repeated measure ANOVA was used. Multiple comparisons were corrected using the Sidak test (Sidak, 1967).

6.3 Results

Sample characteristics

Since there are no validated quantitative criteria for sleep misperception (Castelnovo et al., 2019) and the degree of sleep discrepancy varies across patients (Edinger & Fins, 1995; Tang & Harvey, 2006), we have divided the sample into two groups based on their MI to distinguish patients who underestimated sleep quantity (Manconi et al., 2010). The first group consisted of patients who underestimated their TST (MI > 0; i.e., the underestimating [UN] group). The second group comprised patients with accurate sleep perception or with a tendency to overestimate TST (MI ≤ 0; accurate/overestimating [A/O] group).

Data from 36 participants were included in the final analyses. Sociodemographic and clinical characteristics of the total sample are summarized in Table 12. Baseline differences in sociodemographic, clinical, and sleep characteristics between the two groups are presented in Tables 12 and 13. A significant difference was observed in the number of patients showing severe insomnia based on the ISI score, which was higher in the UN group. No difference was found in any other questionnaire score between the groups.

As shown in Table 13, the UN group subjectively estimated sleep parameters as significantly worse than the A/O group (shorter TST, lower SE, and longer WASO), whereas the groups did not differ significantly in most of the objective sleep parameters. The only difference was found in objective SE, which was higher in the UN group. A significant difference was observed in all sleep discrepancy parameters (SOL, TST, and WASO) between groups.

	Total sample (n = 36)	UN group (n = 16)	A/O group (n = 20)	$t / \chi 2$	p
Sex, % female	61	75	50	2.338	.126
Age (mean, SD) †	46.7 (13.9)	46.2 (11.8)	47.1 (15.8)	-0.181	.857
Length of insomnia (mean years, SD) †	5.92 (5.34)	6.39 (5.21)	5.38 (5.63)	0.513	.612
Insomnia symptoms (n):					
Sleep onset problems	7	2	5	1.036	.309
Sleep continuity problems	15	7	8	0.010	.922
Waking up earlier than desired	1	1	0	1.222	.269
Combination	12	6	6	0.135	.713
Insomnia severity according to ISI (n):					
Mild insomnia	10	4	6	0.283	.595
Moderate insomnia	22	8	14	0.971	.324
Severe insomnia	4	4	0	5.100	**.024**
Significant daytime sleepiness based on ESS (n)	5	2	3	0.117	.732
Significant hyperarousal based on HAS (n)	14	6	8	0.169	.681
Comorbidities (n):					
Depressive symptoms	7	5	2	0.009	.925
Anxiety symptoms	2	2	0	1.694	.193
Tinnitus	2	2	0	0.655	.418
Hypertension	5	4	1	2.973	.085
Thyroid disease	3	2	1	0.655	.418
Married (%)	50	50	45	0.330	.566
Education (%):					
Secondary school	39	44	35		
University degree	61	56	65	0.139	.710

Table 12. Sociodemographic and clinical characteristics of the total sample and the two subgroups. Results of independent-samples t-tests and Chi-squared test are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). UN Group: Underestimating group, A/O Group: Accurate/overestimating group, ISI: Insomnia Severity Index, ESS: Epworth Sleepiness Scale, HAS: Hyperarousal Scale.
† Results of independent t-test.

	UN group	*A/O group*	*t*	P-value
Questionnaires				
ISI	18.44 (4.97)	15.61 (3.13)	2.010	**.053**
ESS	6.09 (4.66)	6.92 (4.17)	-0.544	.590
HAS	39.19 (11.89)	39.89 (8.35)	-0.201	.842
QOL	48.31 (11.9)	49.79 (8.55)	-0.426	.673
Sleep Diary				
SOL (min.)	48.47 (32.68)	33.11 (20.63)	1.701	.098
TST (min.)	330.56 (58.38)	388.44 (46.74)	-3.306	**.003**
SE (%)	67.08 (7.86)	80.01 (6.27)	-5.419	**.000**
WASO (min.)	61.00 (46.60)	28.27 (22.08)	2.882	**.007**
Actigraphy				
SOL (min.)	11.58 (8.84)	17.82 (15.07)	-1.463	.153
TST (min.)	372.73 (33.51)	347.73 (43.40)	1.808	.080
SE (%)	77.26 (4.68)	71.96 (5.99)	2.897	**.007**
WASO (min.)	97.57 (26.07)	108.68 (35.24)	-0.994	.328
Sleep discrepancy				
SOL (min.)	34.75 (34.52)	15.29 (20.58)	2.100	**.043**
WASO (min.)	-41.35 (48.92)	-80.71 (38.95	2.574	**.015**
TST (MI)	0.13 (0.08)	-0.13 (0.09)	8.681	**.000**

Table 13. Baseline differences between the two insomnia subgroups. Results of independent-samples t-tests are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). UN Group: Underestimating group, A/O Group: Accurate/overestimating group, ISI: Insomnia Severity Index, ESS: Epworth Sleepiness Scale, HAS: Hyperarousal Scale, QOL: Quality of Life scale, SOL: sleep onset latency, TST: total sleep time, SE: sleep efficiency, WASO: wake after sleep onset, MI: misperception index, ES: Cohen´s *d* for paired t-test.

Treatment attendance

Three patients missed one session focused on the introduction to cognitive therapy (Session 4). In the total sample, the average number of completed sessions was 5.92 (SD = 0.28), without a significant difference between the UN group (M = 5.88, SD = 0.34) and A/O group (M = 5.95, SD = 0.22), $t(34) = -0.79$, $p = .433$. Adherence to treatment was encouraged by completing daily sleep diaries and was controlled by therapists every session.

Changes in sleep and daytime symptoms in the total sample after CBT-I

After CBT-I, there was a significant reduction of ISI score, a significant reduction of HAS score, and increased QOL score (Table 14). These results reflect a decrease of insomnia severity from moderate to mild insomnia (Bastien et al., 2001), decreased subjective hyperarousal, and increased quality of life after the treatment. There was no change in daytime sleepiness measured by the ESS score. This score was not clinically significant at baseline (Johns, 1991). CBT-I was associated with a significant reduction of subjective SOL and WASO, and a higher subjective SE, with no change in self-reported TST. In the case of objective parameters, both SOL and TST were significantly reduced after the therapy. There was no change in objective SE and WASO.

Sleep discrepancy during CBT-I in the total sample

The MI of TST was found to vary across the weeks of treatment, $F(5, 170) = 11.79$, $p < .001$. No significant change was observed between Week 1 (-0.01, SD = 0.16) and Week 2 (-0.07, SD = 0.16, $p = .13$) in the total sample. Visual presentation of MI TST changes in the total sample throughout the treatment is shown in Figure 6.

Total sample (n = 36)					
	Pre-treatment	**Post-treatment**	***t / T***	***P-value***	***ES***
Questionnaires					
ISI	16.9 (4.3)	9.8 (3.7)	9.5	**.000**	**1.68**
ESS±	6.5 (4.4)	6.4 (4.1)	161	.712	0.00
HAS±	39.6 (10)	36.2 (10.1)	13.6	**.039**	**0.39**
QOL±	49.1 (10.1)	54.1 (10.6)	102.5	**.003**	**-0.38**
Sleep Diary					
SOL (min.)±	39.7 (27.1)	19.5 (7.3)	48	**.000**	**0.81**
TST (min.)	362.7 (59.1)	369.4 (48.5)	-1.1	.297	-0.18
SE (%)	75.3 (9.1)	86.3 (7.6)	-6.3	**.000**	**-1.09**
WASO (min.)	41.16 (38.24)	24.60 (18.41)	2.9	**.005**	**0.52**
Actigraphy					
SOL (min.) ±	15 (12.9)	8.9 (9.5)	57	**.000**	**0.49**
TST (min.)	358 (41)	323.9 (34.7)	5	**.000**	**0.86**
SE (%)	74.3 (6)	75.3 (5.3)	-1.1	.269	-0.19
WASO (min.)	103.96 (31.71)	90.04 (21.86)	2.5	**.017**	**0.44**
Sleep discrepancy					
SOL (min.) ±	23.9 (28.9)	11.4 (12.2)	199.5	**.036**	**0.47**
WASO (min.)	-65.3 (47.23)	-69.88 (25.47)	0.6	.514	0.12
TST (MI)	-0.01 (0.16)	-0.16 (0.14)	6.9	**.000**	**1.16**

Table 14. Pre- to post-treatment differences in the total sample, mean (SD).. Results of paired-sample t-tests, Wilcoxon Signed Ranks Test, and Cohen´s *d* and *r* for effect size are presented. Bold indicates a statistically significant result *(*$p \leq 0.05$). ISI: Insomnia Severity Index, ESS: Epworth Sleepiness Scale, HAS: Hyperarousal Scale, QOL: Quality of Life scale, SOL: sleep onset latency, TST: total sleep time, SE: sleep efficiency, WASO: wake after sleep onset, MI: misperception index, ES: Cohen´s *d* for paired t-test, *r* for Wilcoxon test.

± Wilcoxon Signed Ranks Test, *r* for effect size is presented

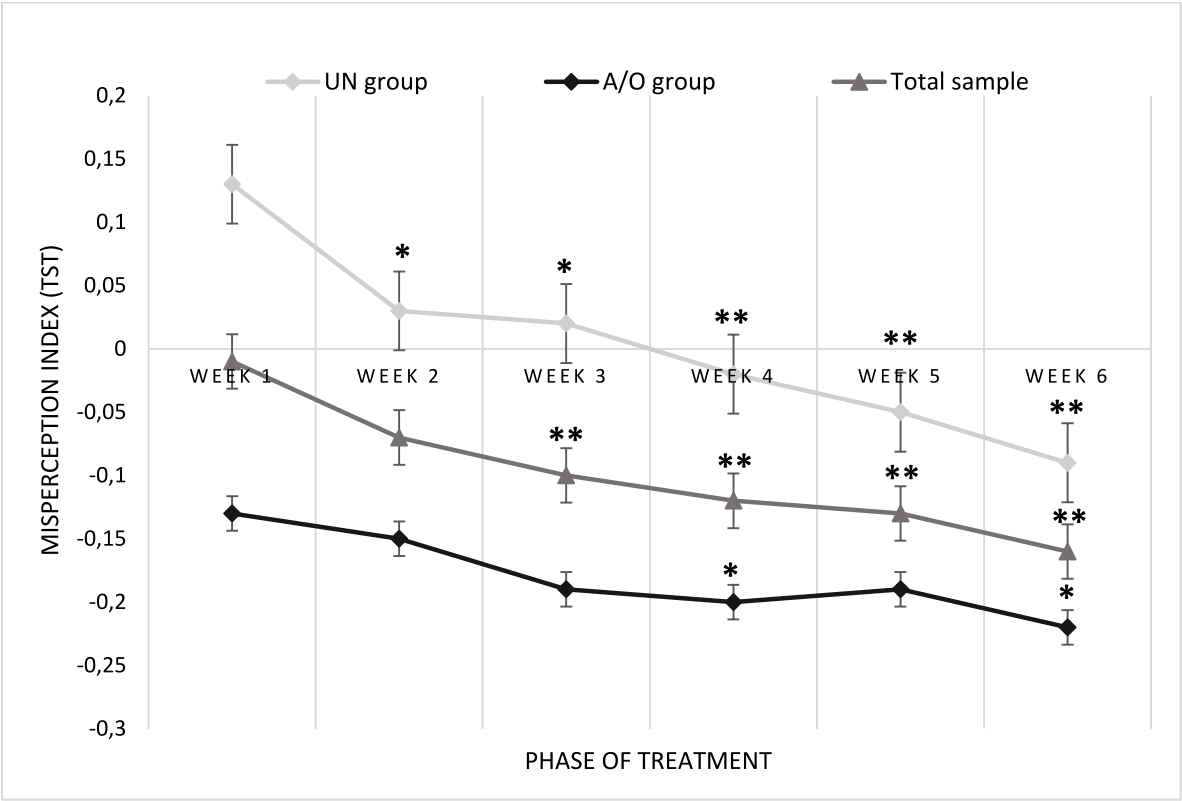

Figure 6. Changes of MI TST (mean) during the CBT-I in the total sample, UN group, and A/O group. TST: total sleep time, UN group: Underestimating group, A/O group: Accurate/overestimating group $^{*}p \leq 0.05$, $^{**}p \leq 0.01$ for comparison with Week 1.

Effect of CBT-I in the UN and A/O group

No significant Time x Group interaction was found in any of the questionnaires (Table 15). Participants in both groups reported significantly lower insomnia severity as well as lower self-reported hyperarousal and significantly higher quality of life after the treatment, without a significant change in daytime sleepiness.

Condition	*Pre-treatment*	*Post-treatment*	*Main effects*	*df*	*F(1, 34)*	*p*	*ES (η^2)*
ISI							
UN group	18.4 (4.9)	10.3 (3.7)	Time	1, 30	91.902	**.000**	**.754**
A/O group	15.6 (3.1)	9.4 (3.7)	Group		2.301	.140	.071
			Time X Group		1.210	.280	.039
ESS							
UN group	6.1 (4.7)	6.7 (4.8)	Time	1, 31	0.019	.891	.001
A/O group	6.9 (4.2)	6.1 (3.5)	Group		0.041	.841	.001
			Time X Group		2.292	.140	.069
HAS							
UN group	39.2 (11.9)	35.1 (10.6)	Time	1, 31	5.504	**.032**	**.140**
A/O group	39.9 (8.4)	37.2 (9.9)	Group		0.182	.672	.006
			Time X Group		0.183	.672	.006
QOL							
UN group	48.3 (11.9)	54.2 (12.1)	Time	1, 31	6.447	**.016**	**.172**
A/O group	49.8 (8.6)	54.1 (9.6)	Group		0.067	.789	.002
			Time X Group		0.200	.658	.006

Table 15. Pre- to post-treatment differences in subjective scales and questionnaires in the UN and A/O group. Results of two-way mixed ANOVA and partial eta square (η^2) for effect size are presented, means (SD). Bold indicates a statistically significant result ($p \leq 0.05$). UN group: Underestimating group, A/O group: Accurate/overestimating group. ISI: Insomnia Severity Index, ESS: Epworth Sleepiness Scale, HAS: Hyperarousal Scale, QOL: Quality of Life scale, ES: effect size.

A significant Time x Group interaction was found in subjective SE, WASO, objective TST, and SOL discrepancy (Table 16). Only the UN group showed significantly higher self-reported SE and lowered self-reported WASO after the therapy, as well as shorter objective TST and lower SOL discrepancy. Both groups reported significant reductions of self-reported SOL, objective SOL, and objective WASO with no change in self-reported TST. No difference in objective SE was found in either group. Both groups reported a significant change of TST discrepancy after CBT-I and no change in WASO discrepancy (Table 17).

Condition	*Pre-treatment*	*Post-treatment*	*Main effects*	*df*	*F*	*p*	*ES (η^2)*
Sleep diary							
SOL (min.)							
UN group	51.00 (34.53)	23.15 (5.33)	Time	1, 31	21.953	**.000**	**.415**
A/O group	33.11 (20.62)	16.80 (7.38)	Group		5.550	**.025**	**.152**
			Time X Group		1.497	.230	.046
TST (min.)							
UN group	330.56 (58.38)	334.7 (37.2)	Time	1, 34	0.999	.325	.029
A/O group	388.40 (46.70)	397.30 (37.60)	Group		19.072	**.000**	**.359**
			Time X Group		0.134	.726	.004
SE (%)							
UN group	67.1 (7.9)	83.5 (7.9)	Time	1, 31	47.766	**.000**	**.606**
A/O group	80 (6.3)	88.2 (6.7)	Group		20.268	**.000**	**.395**
			Time X Group		4.362	**.045**	**.123**
WASO (min.)							
UN group	61.00 (49.25)	30.69 (23.04)	Time	1, 31	12.446	**.001**	**.286**
A/O group	28.27 (22.08)	20.65 (13.91)	Group		6.510	**.016**	**.174**
			Time X Group		4.453	**.043**	**.126**
Actigraphy							
SOL (min.)							
UN group	11.6 (8.8)	8.5 (11.8)	Time	1, 34	7.681	**.009**	**.184**
A/O group	17.8 (15.1)	9.1 (7.4)	Group		1.202	.281	.034
			Time X Group		1.754	.194	.049
TST (min.)							
UN group	372.7 (33.5)	316.3 (36.8)	Time	1, 32	39.220	**.000**	**.551**
A/O group	347.7 (43.4)	330.3 (35.8)	Group		0.168	.685	.005
			Time X Group		11.370	**.002**	**.262**
SE (%)							
UN group	77.3 (4.7)	77.8 (4.8)	Time	1, 34	1.119	.297	.032
A/O group	71.9 (5.9)	73.2 (4.8)	Group		11.034	**.002**	**.245**
			Time X Group		0.158	.694	.005
WASO (min.)							
UN group	97.57 (26.07)	72.60 (11.27)	Time	1, 31	8.069	**.008**	**.207**
A/O group	108.68 (35.24)	102.88 (18.64)	Group		8.751	**.006**	**.220**
			Time X Group		3.134	.087	.092

Table 16. Pre- to post-treatment differences in objective and subjective sleep parameters in the UN and A/O group. Results of two-way mixed ANOVA and partial eta square (η^2) for effect size are presented, means (SD). Bold indicates a statistically significant result ($p \leq 0.05$). UN group: Underestimating group, A/O group: Accurate/overestimating group. SOL: sleep onset latency, TST: total sleep time, SE: sleep efficiency, WASO: wake after sleep onset, MI: misperception index, ES: effect size.

Condition	*Pre-treatment*	*Post-treatment*	*Main effects*	*df*	*F*	*p*	*ES (η^2)*
SOL discrepancy (min.)							
UN group	34.75 (34.52)	15.96 (13.38)	Time	1, 34	68.775	**.000**	**.669**
A/O group	15.29 (20.58)	7.67 (10.12)	Group		6.253	**.017**	**.155**
			Time X Group		11.904	**.002**	**.259**
WASO discrepancy (min.)							
UN group	-41.35 (48.92)	-49.64 (17.63)	Time	1, 30	0.475	.496	.016
A/O group	-80.71 (38.95	-83.73 (20.29)	Group		13.242	**.001**	**.306**
			Time X Group		0.072	.790	.002
TST discrepancy (MI)							
UN group	0.13 (0.1)	-0.1 (0.1)	Time	1, 34	8.816	**.005**	**.206**
A/O group	-0.13 (0.1)	-0.2 (0.1)	Group		37.122	**.000**	**.522**
			Time X Group		1.572	.219	.044

Table 17. Pre- to post-treatment differences in objective and subjective sleep discrepancy parameters in the UN and A/O group. Results of two-way mixed ANOVA and partial eta square (η^2) for effect size are presented, means (SD). Bold indicates a statistically significant result ($p \leq 0.05$). UN group: Underestimating group, A/O group: Accurate/overestimating group. SOL: sleep onset latency, TST: total sleep time, WASO: wake after sleep onset, MI: misperception index, ES: effect size.

Sleep discrepancy during CBT-I in the UN and A/O group

The MI of TST varied across the weeks of treatment in the UN group, $F(5, 70) = 12.44$, $p < .001$, with a significant decrease from Week 1 (+0.14, SD = 0.08) to Week 2 (+0.02, SD = 0.15, $p = .048$). In the A/O group, MI TST also varied across the therapy, $F(5, 95) = 2.93$, $p < .05$, without a significant change of MI TST from Week 1 (-0.13, SD = 0.09) to Week 2 (-0.15, SD = 0.13, $p = .99$). Changes of MI TST during the CBT-I are visualized in Figure 6.

7 EFFECT OF CBT-I AND BLUE LIGHT-BLOCKING GLASSES ON SLEEP DISCREPANCY (STUDY 4)

7.1 Aims and hypotheses

Following recent research (Shechter et al., 2018), the aim of this randomized controlled trial was to for the first time to assess the effect of CBT-I in combination with blue-light blocking glasses to explore whether the additional chronotherapeutic intervention could enhance the impact of CBT-I on sleep parameters. A CBT-I group with active (blue-light blocking) glasses was compared with a CBT-I group wearing clear placebo glasses. Results of Study 4 have been published in a peer-reviewed impacted journal. The details can be found in Janku et al. (2020a).

We expected to find:

1. Improvement in sleep parameters in both active and placebo CBT-I groups.
2. Larger effect on sleep parameters in a group receiving CBT-I and blue-light blocking glasses.
3. Larger effect on sleep discrepancy parameters (TST, SOL, and WASO) in a group receiving CBT-I and blue-light blocking glasses.

7.2 Materials and methods

Participants

Forty-five patients with insomnia who were enrolled in the CBT-I group programme were invited to participate in the present study. Insomnia diagnosis was established on the ICD-10 (WHO, 2004). Inclusion and exclusion criteria were the same as for Study 3.

Subjective sleep measures

Patients were asked to complete a sleep diary every day during the CBT-I programme. The primary outcomes were average SOL, TST, WASO, and SE for every week. The diary was analysed weekly by the leading therapist. Patients also completed a battery of self-reported

questionnaires to assess sleep complaints and daytime symptoms at the beginning and the end of CBT-I. The battery included PSQI (Buysse et al., 1989); ISI; and HAS.

Actigraphy

Actigraphy recording was based on the same methodology as in Study 3. The extracted outcomes were the same as for sleep diaries. The MI was computed to evaluate the effect of CBT-I and blue-light blocking glasses on TST discrepancy. SOL and WASO discrepancy parameters were obtained based on the same formula as in Study 3 (Herbert et al., 2017).

Interventions

The CBT-I group programme had the same structure as in Study 3. For the active condition, the UVEX S1933X (U.S. certification ANSI Z87+ and CSA Z94.3) orange glasses were given to patients. Based on the used spectrum control technology, they were supposed to reduce up to 98 % of lights of blue wavelength. As the placebo condition, the UVEX S1900 (U.S. certification ANSI Z87+ and CSA Z94.3) clear glasses with no ability to filtrate blue light were used. For illustration, see Figure 6. Patients of both groups were instructed to wear the glasses 90 minutes before scheduled bedtime from week 2 till the end of the programme. A separate item was added to the sleep diary for the patients to report the usage of glasses every evening to enhance their compliance. The patients reported no adverse effects.

Figure 7. Orange (active) glasses and clear (placebo) glasses.

Procedure

At the first CBT-I session, participants were asked to fill in the questionnaires and received actigraphs. The first week served for baseline measurement. In the second session, patients received either active glasses or placebo glasses. Patients in one group had the same type. They were told that the study is focused on several types of glasses with different filtration properties. The instruction was to wear the glasses every day of the treatment at least 90 minutes before bedtime. To increase compliance, patients were repeatedly educated about light hygiene and were asked to report usage of glasses in sleep diaries every day.

Statistical analyses

IBM SPSS Statistics software (v 23.0) was used for the analyses. A sample size calculation was performed before the study began using a large effect size (d = 0.90) with α = 0.05. To detect significant differences in subjective sleep parameters (SOL, SE, TST) before and after the therapy, the suggested sample size was n = 6 – 9 in each group (Cervena et al., 2004; Koffel et al., 2015). For the detection of differences in objective sleep parameters measured by actigraphy, at least 8 patients were suggested (Vallieres & Morin, 2003). As such, we aimed to include 30 patients in total, 15 patients in each group.

The independent-samples t-tests were used to compare both groups at baseline. The General Linear Model (GLM) was used to compare differential values (pre- to post-treatment change) between both groups with age, gender, and leading therapist set as covariates. Paired t-tests were used to assess changes for each variable separately within each group..

7.3 Results

Sample characteristics

A detailed flow of the enrolment and allocation of participants can be seen in Janku et al. (2020a). A final sample of 30 participants was involved in the analyses. Basic characteristics are summarized in Table 18. A difference in age between the two groups reached the threshold of statistical significance [$t(28) = -2.052$, p = 0.050], and was used as a covariate together with gender and assigned therapist. Baseline measures in each group are presented in Table 19.

	Total sample	Active group	Placebo group
n	30	15	15
Female / Male	15 / 15	6 / 9	9 / 6
Age	48.1 (16.1)	42.4 (14.8)	53.9 (15.8)
Length of insomnia (years)	5.32 (5.07)	6.33 (6.62)	4.38 (2.68)
Married (%)	50 %	46 %	53 %
Education (%):			
High school	26 %	15 %	36 %
University degree	74 %	85 %	64 %

Table 18. Socio-demographic characteristics of participants in "active" (blue-light blocking glasses) and "placebo" (clear glasses) group. Mean (SD) years for age and length of insomnia are presented.

	Baseline Values			
	Active (n = 15)	***Placebo (n = 15)***	***t***	***p***
ISI	17.26 (5.42)	17.20 (3.23)	0.041	.968
PSQI	12.60 (4.36)	13.33 (3.46)	-0.511	.614
HAS	41.60 (8.40)	37.07 (10.42)	1.312	.200
Sleep diary				
SOL (min.)	36.79 (25.94)	56.51 (61.35)	-1.146	.261
TST (min.)	369.84 (47.74)	393.69 (79.11)	-1.000	.326
WASO (min.)	42.23 (40.82)	43.57 (29.53)	-0.103	.919
SE (%)	75.10 (12.03)	75.41 (11.26)	-0.073	.943
Actigraphy				
SOL (min.)	12.47 (15.11)	18.12 (12.78)	-1.059	.299
TST (min.)	359.97 (52.17)	378.79 (49.45)	-0.975	.339
WASO (min.)	100.78 (26.54)	112.45 (33.24)	-1.033	.311
SE (%)	74.52 (6.21)	73.67 (5.46)	0.381	.706
Sleep discrepancy				
MI TST	-0.54 (0.17)	-0.05 (0.23)	0.191	.850
SOL (min.)	24.33 (23.88)	25.98 (40.65)	-0.133	.895
WASO (min.)	-58.54 (49.65)	-73.66 (45.01)	0.838	.409

Table 19. Baseline characteristics of Active and Placebo group. Mean (SD) scores at the baseline for both "active" (blue-light blocking glasses) and "placebo" (clear glasses) groups. ISI: Insomnia Severity Index, PSQI: Pittsburgh Sleep Quality Index, ESS: Epworth Sleepiness Scale, HAS: Hyperarousal Scale,

SOL: Sleep Onset Latency, TST: Total Sleep Time, WASO: Wake After Sleep Onset, SE: Sleep Effectivity.

Effect of therapy in the Active and Placebo group

Changes between pre- and post-treatment scores in each group are presented in Table 20. For both groups, a significant difference was found in the ISI, PSQI, and self-reported WASO and SE. In the active group only, the HAS score was significantly reduced, and subjective TST was prolonged. In the placebo group, a significant reduction of objective TST was observed. A significant change of MI TST and a reduction of SOL discrepancy was present only in the active group, while the WASO discrepancy did not change in any of the groups.

	Active (n = 15)						Placebo (n = 15)				
Sleep parameter	***Pre-treatment***	***Post-treatment***	***t***	***P-value***	***ES***	***Sleep parameter***	***Pre-treatment***	***Post-treatment***	***t***	***P-value***	***ES***
Questionnaires						**Questionnaires**					
ISI	17.27 (5.42)	10.53 (3.36)	6.29	**.000**	**1.60**	**ISI**	16.83 (2.98)	10.92 (3.42)	4.75	**.001**	**1.37**
PSQI	12.60 (4.36)	8.40 (12.39)	4.18	**.001**	**1.08**	**PSQI**	13.08 (3.42)	8.92 (3.20)	5.00	**.000**	**1.44**
HAS	41.60 (8.40)	36.93 (10.02)	2.90	**.012**	**0.75**	**HAS**	35.5 (10.13)	32.08 (11.28)	1.26	.233	0.36
Sleep diaries						**Sleep diaries**					
SOL (min.)	36.80 (27.01)	18.41 (6.15)	2.65	**.021**	**0.73**	**SOL (min.)**	59.77 (62.30)	25.48 (23.53)	2.08	.058	0.56
TST (min.)	369.14 (48.93)	406.02 (50.16)	-2.73	**.018**	**-0.76**	**TST (min.)**	382.73 (69.27)	375.69 (49.32)	0.56	.588	0.15
WASO (min.)	43.95 (41.94)	20.63 (11.81)	2.17	**.049**	**0.61**	**WASO (min.)**	43.78 (30.64)	30.83 (22.92)	2.37	**.034**	**0.63**
SE (%)	74.59 (12.63)	90.09 (4.28)	-4.35	**.001**	**-1.21**	**SE (%)**	74.67 (11.30)	85.09 (9.07)	-3.80	**.002**	**-1.02**
Actigraphy						**Actigraphy**					
SOL (min.)	12.47 (15.12)	8.83 (9.83)	1.67	.117	0.43	**SOL (min.)**	18.12 (12.31)	12.31 (11.74)	1.69	.115	0.47
TST (min.)	359.97 (52.18)	350.22 (47.50)	1.01	.329	0.26	**TST (min.)**	378.79 (49.46)	352.13 (37.17)	2.58	**.024**	**0.72**
WASO (min.)	100.78 (26.54)	94.33 (29.35)	0.99	.339	0.34	**WASO (min.)**	112.45 (33.24)	102.46 (31.60)	1.58	.140	0.44
SE (%)	74.53 (6.21)	75.74 (5.67)	-1.19	.254	-0.31	**SE (%)**	73.68 (5.47)	74.95 (5.99)	-1.49	.161	-0.41
Sleep discrepancy						**Sleep discrepancy**					
MI TST	-0.05 (0.17)	-0.16 (0.06)	2.74	**.018**	**0.76**	**MI TST**	-0.05 (0.23)	-0.09 (0.13)	1.32	.212	0.38
SOL (min.)	24.33 (23.88)	8.5 (10.97)	2.34	**.037**	**0.65**	**SOL (min.)**	25.98 (40.65)	17.42 (24.62)	1.32	.213	0.38
WASO (min.)	-58.54 (49.65)	-75.11 (33.44)	1.4	.186	0.38	**WASO (min.)**	-73.66 (45.01)	-70.93 (33.23)	0.17	.863	0.05

Table 20. Effect of intervention within each group. Results of the paired-samples t-tests are presented for a group with "active" filtering glasses and "placebo" glasses. *t*-values, statistical significance, and effect sizes (Cohen's *d*) are provided. Bold indicates a statistically significant result ($p \leq 0.05$). ISI: Insomnia Severity Index, PSQI: Pittsburgh Sleep Quality Index, HAS: Hyperarousal Scale, SOL: Sleep Onset Latency, TST: Total Sleep Time, WASO: Wake After Sleep Onset, SE: Sleep Effectivity.

8 DISCUSSION

Our results highlight the role of REM sleep in the subjective evaluation of sleep duration and its underestimation in insomnia patients. Both of the studies conducted to explore the objective correlates of sleep discrepancy showed a significant association between a reduction of REM sleep proportion and a degree of sleep misperception. A lower proportion of REM sleep was also the only common feature found in both sleep misperception groups (PSY/MIS, PARA) compared to their control groups. This result was neither affected by similarities in objective SE and TST in misperception groups and controls. An increased NREM 1 and decreased NREM 3 sleep stage found in PARA compared to GS confirmed the alteration in different sleep parameters than in conventional sleep continuity measures, such as SOL, TST, WASO, or SE (Bastien et al., 2014; Krystal et al., 2002; Salin-Pascual, Roehrs, Merlotti, Zorick, & Roth, 1992) in insomnia patients underestimating their sleep quantity. The only difference in spectral sleep characteristics was found in PSY/MIS patients who showed a trend towards reduced delta EEG activity during NREM 3 sleep compared to PSY. Although the result was not significant, this trend could potentially reflect higher cortical activation during the NREM 3 sleep in the PSY/MIS group, as hypothesized. Our analysis did not confirm the results of previous studies showing an increased high-frequency EEG activity during sleep in PARA patients compared to GS (Krystal et al., 2002; Spiegelhalder et al., 2012). However, because of the absence of an adaptation night, GS group might have been aroused as the PARA group, due to a first-night effect (Byun, Kim, Moon, Motamedi, & Cho, 2019). Also, the small sample size may have an impact on these results.

The result of a negative correlation between REM sleep and sleep discrepancy is in line with a study by Perusse et al. (2015). These authors showed that the more time insomnia patients spent in REM sleep, the better was their subjective evaluation of sleep quality and quantity. REM sleep could contribute to more accurate estimates of sleep duration because of vivid and emotional dreams, which are easier to remember (Perusse et al., 2015). Thus, one could better distinguish between sleep and a state of wakefulness. This assumption was supported by a study where patients with insomnia were awakened from either REM or NREM sleep stage. Results showed that patients perceived their sleep better when they were awakened from REM sleep than from the NREM sleep period (Mercer & Lack, 2003).

In Study 2, a greater sleep discrepancy was connected with more severe depressive symptoms, which is in line with previous research (Fernandez-Mendoza et al., 2011; Herbert et al., 2017; Tsuchiyama et al., 2003; Williams et al., 2013). It has been documented in both patients with depression (Mathews & Bradley, 1983) and a non-clinical sample with induced depressive mood (Matt, Vazquez, & Campbell, 1992) that a current mood state may influence an individual's memory recalls or information processing. Patients with depressive mood might be, therefore, more prone to evaluate their sleep as worse than it is according to the objective measures in order to be congruent with their current mood. Studies have also demonstrated a memory bias congruent with the current mood in symptoms reporting (Larsen, 1992). The opposite direction of this relationship may be possible too. A more significant discrepancy between subjective and objective TST may worsen a next-day mood. An association between sleep quality and mood upon awakening has also been documented (Pemberton & Fuller Tyszkiewicz, 2016).

Another possible explanation is related to the assumption that sleep discrepancy may be associated with alterations in specific brain regions and sleep stages that may cause a mood disruption and may be common for insomnia as well as for depression. Both insomnia and depression have been linked to increased cortical arousal during sleep (Hein et al., 2017) and altered REM sleep (Riemann et al., 2012). The REM sleep stage is crucial for emotion regulation processes, which are suggested to be impaired in insomnia and especially in depression. As such, the emotion dysregulation has been proposed as another factor that may underlie the link between these two syndromes (C. Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010). Our analyses did not reveal a difference in REM sleep proportion between the groups differing in depressive symptoms, nor did we find the relation between the REM sleep proportion and depression severity. However, other alterations of the REM sleep stage could be involved, such as REM sleep disruption by arousals, which may disturb overnight adaptation of amygdala activity and lead to emotional dysregulation (Wassing et al., 2019). Since we did not include a control group of healthy participants, we cannot conclude whether both of the insomnia subgroups showed a significant reduction of REM sleep compared to good sleepers. Given the association between REM sleep and sleep discrepancy, a reduction of REM sleep proportion and its contribution to sleep misperception experience (Perusse et al., 2015) might have had an impact on the next-day mood subsequently (Pemberton & Fuller Tyszkiewicz, 2016). Future

studies may explore whether REM sleep reduction, which leads to sleep misperception, could be a transitional state to further REM sleep alterations and rebounds connected with more severe objectively diagnosed depression.

Studies on CBT-I revealed a significant reduction of sleep quantity underestimation after the therapy. In Study 3, objective TST was significantly reduced with no change in subjective TST, leading to an overestimation of objective TST. The reduction of objective TST after CBT-I has been reported in previous studies (Edinger, Wohlgemuth, Radtke, Coffman, & Carney, 2007) as a possible consequence of sleep restriction (Kyle et al., 2014). In line with our hypotheses, the UN group reported a significant decrease in TST discrepancy as well as in SOL discrepancy, which is in accordance with previous research (Cronlein et al., 2019; Kay et al., 2015; Lund et al., 2013). The UN group also reported increased subjective SE and decreased subjective WASO, which was not observed in the A/O group. Baseline differences in self-reported sleep parameters between groups could contribute to different magnitudes of change in these sleep measures. The UN group showed significantly lower sleep quality based on sleep diary parameters, more significant sleep discrepancies, and involved more patients with severe insomnia compared to the A/O group, which could increase the probability of more considerable change (Jin, 1992).

Sleep discrepancies could have been affected by several CBT-I components. In the UN group, the change of TST misperception could have been caused by a significant shortening of objective sleep duration after CBT-I due to the sleep restriction (Kyle et al., 2014). This assumption was further supported by a significant change of TST discrepancy from Week 1 to Week 2 when the sleep restriction was implemented. Due to the subjective underestimation of sleep duration in the UN group at the beginning of treatment, this group could have a stricter sleep window, leading to an increased sleep pressure, more consolidated sleep (Kyle et al., 2014), and the subsequent correction of sleep misperception. This effect of sleep restriction would be in line with Maric et al. (2019), who showed that underestimation of TST shifted towards overestimation during sleep restriction in healthy volunteers (Maric, Burgi, Werth, Baumann, & Poryazova, 2019). However, since this cannot fully explain the shift from accurate estimation to overestimation of TST in the A/O group, other CBT-I components should be taken into account.

Psychoeducation and cognitive therapy have been shown to reduce dysfunctional beliefs about sleep, and these changes have been associated with improvements in objective and subjective sleep quality (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). Thus hypothetically, cognitive therapy could have contributed to the overestimation of TST in the A/O group after the therapy in the present study. However, any conclusions cannot be made since we did not involve the measurement of cognition. Moreover, a sparse amount of studies examining the association between cognitive changes and sleep discrepancy suggests that typical cognitive therapy within CBT-I has no significant effect on sleep misperception (Lund et al., 2013). Other specific interventions focusing on sleep discrepancy might be beneficial for patients with sleep misperception, e.g., education about sleep discrepancy (Cronlein et al., 2019), verbal feedback, or behavioural experiments focused on the discrepancy between subjective and objective sleep measures (Tang & Harvey, 2006).

Study 4 showed that the additional chronotherapeutic tool might enhance the effect of CBT-I on subjective sleep quality and sleep discrepancy. Specifically, the combination of CBT-I and blue-light blocking glasses was associated with significantly prolonged subjective TST and decreased subjective SOL, with no such an effect in the placebo group, which is in line with a previous study (Shechter et al., 2018). Only the group with active glasses showed a significant change in TST discrepancy, which shifted towards overestimation at the end of the therapy, and a significant reduction in SOL discrepancy. In addition, this therapeutic combination was associated with no change in objective TST, which was reduced in the placebo group, similarly as in Study 3. Hypothetically, the blue-light blocking glasses could help to maintain the objective TST and alleviate this effect of sleep restriction. More research is needed to confirm this assumption.

Studies have shown that evening exposure to blue-light usually emitted from electronic devices may cause a suppressed secretion of melatonin (Brainard et al., 2015), prolonged SOL, and decreased overall subjective sleep quality (Gronli et al., 2016). The blue-light blocking glasses may have, therefore, weakened the negative effect of blue-light exposure on sleep parameters. Decreased subjective SOL and increased subjective TST could be related to an earlier dim light melatonin onset and promoted circadian regulation. However, since we did not involve more sensitive objective measures of circadian markers (e.g., melatonin secretion), we cannot make any definitive conclusion.

The blue-light blocking glasses might have also reduced the effect of light on arousal, which could consequently improve sleep quality. A short-wavelength spectrum of light at night has been related to enhanced cortical arousal (Gaggioni, Maquet, Schmidt, Dijk, & Vandewalle, 2014; Smotek, Vlcek, Saifutdinova, & Koprivova, 2019). In line with this assumption, subjectively evaluated hyperarousal was significantly reduced only in the group with blue-light blocking glasses in our study, which could have reflected a reduction in cognitive arousal, as shown by van der Lely et al. (2015). Decreased arousal might have led to a decreased subjective SOL and reduction in SOL discrepancy as well as prolonged subjective TST and the shift towards overestimation of sleep duration in the active group.

The results of the present thesis should be interpreted with caution in light of its limitations. Because of the retrospective study design of Study 1 and Study 2, we could not implement an adaptation PSG night. This might have had an impact on sleep parameters, especially on the PSA results in the GS group. Another limitation is a small number of participants in Study 1. This was caused by the fact that we only included patients who underwent PSG, which is not usually indicated for insomnia patients in clinical practice, as well as because we aimed to involve patients who did not take any medication, which might influence their sleep EEG. Another limitation of Study 1 was that we did not compare all four groups because of the age differences. Also, the comparison of PARA and GS could be biased by the fact that GS did not complain about poor sleep and did not have a diagnosis of insomnia. However, we were able to detect a similar change of REM sleep in both PSY/MIS (compared to PSY) and PARA (compared to GS), further supporting the suggestion of an important role of REM sleep in subjective sleep evaluation. Moreover, it seems that sleep misperception may not be a phenomenon exclusively present only in insomnia patients, as some studies have already described its presence in healthy subjects (Bianchi et al., 2012). The limitation of Study 2 was related to the measurement of depressive symptoms only by a self-reported questionnaire and not by an objective assessment by a psychiatrist. Therefore, it might not reflect the real severity of reported symptoms.

One of the limitations of Study 3 and Study 4 was the absence of a control group of patients undergoing a different type of treatment or remaining untreated. Therefore, we cannot conclude with certainty that the observed effect was associated specifically with CBT-I. In Study 3, further distinguishing of patients in the A/O group (i.e., patients with accurate estimates

of sleep and patients who overestimate sleep quantity) might yield more accuracy when studying perceptions of their sleep. Study 4 would greatly benefit from controlling daytime light exposure of patients, which could have mediated the effect of the evening light exposure (Rangtell et al., 2016). Additionally, the potential role of chronotype or light hygiene before the CBT-I treatment was not explored. Future studies may also use more sensitive objective measures, such as PSG, to explore more subtle changes of sleep on different levels (sleep architecture, sleep microstructure), possibly underlying the change of subjective sleep parameters (Cervena et al., 2004). This could help to explain the shift of accurate subjective TST estimation at baseline towards overestimation after the treatment observed only in the group with blue-light blocking glasses and also in the A/O group in Study 3.

9 SUMMARY AND CONCLUSION

The present thesis aimed to explore further the phenomenon of subjective and objective sleep discrepancy often seen in patients with insomnia disorder. Specifically, the aim was to assess its objective correlates on sleep macrostructure and microstructure level, its association to depression, and its changes during and after the insomnia treatment.

In summary, our findings further confirm that insomnia patients with the objective and subjective sleep discrepancy show specific alterations of their sleep regardless of different sleep continuity parameters. A significant decrease in the REM sleep stage was the only common feature found in different groups of patients with sleep misperception, as well as the only correlate of sleep discrepancy in our studies. Our findings also point out the relation between sleep discrepancy and depressive symptoms and confirm the effectiveness of the CBT-I in correcting negative sleep discrepancies. The therapeutic combination of CBT-I with the usage of blue-light blocking glasses showed promising results by enhancing the effect of CBT-I on sleep quality. The results raise a question of whether more subtle objective sleep changes might have contributed to the overestimation of sleep quantity after the CBT-I in patients with accurate sleep estimates at baseline, which was observed in both CBT-I studies of the present thesis.

Future studies should further explore the role of sleep discrepancy in common pathophysiology of insomnia and depression. It seems that the association between sleep discrepancy, REM sleep alterations, and subsequent emotional dysregulation may be a part of the common pathway. One of the interesting questions for future research would be whether the sleep discrepancy may be one of the risk factors or predictors of depression development. Future studies should also use more sensitive measures of brain activity, e.g., high-density EEG to assess regional spectral power changes during sleep in insomnia patients who underestimate as well as in patients who overestimate their sleep quantity. Together with involving patients with psychiatric comorbidities, this may extend the understanding of insomnia pathophysiology and its association with neuropsychiatric disorders.

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PUBLICATIONS OF THE GRADUATE

9.1 Publications underlying the thesis

a) Publications in English

Janků, K., Šmotek, M., Fárková, E., Kopřivová, J. (2020). Subjective–objective sleep discrepancy in patients with insomnia during and after cognitive behavioral therapy: An actigraphy study. *Journal of Sleep Research*. https://doi.org/10.1111/jsr.13064. ***IF: 3.432***

Janků, K., Šmotek, M., Fárková, E., Kopřivová, J. (2020). Block the light and sleep well: Evening blue light filtration as a part of cognitive behavioural therapy for insomnia. *Chronobiology International*. 37(2), 248-259. https://doi.org/10.1080/07420528.2019.1692859. ***IF: 2.562***

b) Publications in Czech

Veldová, K., Bušková, J., Kopřivová, J. (2019). [Cognitive behavioural therapy for insomnia: changing sleep or its perception?]. *Psychiatrie*, 23(3):115-120.

Veldová, K., Šóš, P., Kopřivová, J. (2015). [Paradoxical insomnia and its causes]. *Psychiatrie*, 19(3):129-135.

9.2 Other publications

a) Publications in English

Anýž, J., Bakštein, E., Dudysová, D., **Veldová, K.**, Lišková, M., Fárková, E., Kopřivová, J., Španiel F. (2019). Politics: no wink of sleep. The effects of global impact events on populations' sleep characteristics. *Social Science & Medicine*. ***IF: 3.007***

b) Publications in Czech

Veldová, K., Lišková, M., Kopřivová, J. (2017). Mezioborový přístup u poruch spánku v lékařské praxi. *Zdravotnictví a medicína*. Online: https://zdravi.euro.cz/clanek/mezioborovy-pristup-u-poruch-spanku-v-lekarske-praxi-485173?seo_name=mlada-fronta-noviny-zdravi-euro-cz

Veldová, K., Šóš, P. (2016). Nový směr léčby nespavosti – elektrická stimulace mozku. *Vesmír*. Online: https://vesmir.cz/cz/on-line-clanky/2016/05/novy-smer-lecby-nespavosti-elektricka-stimulace-mozku.html

Veldová, K., Procházka, R. (2014). [Conscious strategies of coping with tinnitus]. *Československá psychologie*, 59(2):162-173. ***IF: 0.239***

c) Book chapters

Janečková, D., Weissová, K., Fárková, E., **Veldová, K.**, Lišková, M., Dudysová, D., Šmotek, M., Kopřivová, J., Bendová, Z. [Early bird catches the worm…but what about owls?] In: Horáček, J., Kesner, L., Höschl, C., Španiel F. *Mozek a jeho člověk, mysl a její nemoc*. Praha: Galén, 2016, s.146-152,OSBN:978-80-7492-283-1.

APPENDIXES - Original papers related to the thesis

APPENDIX 1

Janků, K., Šmotek, M., Fárková, E., Kopřivová, J. (2020). Subjective–objective sleep discrepancy in patients with insomnia during and after cognitive behavioral therapy: An actigraphy study. *Journal of Sleep Research*. https://doi.org/10.1111/jsr.13064

APPENDIX 2

Janků, K., Šmotek, M., Fárková, E., Kopřivová, J. (2020). Block the light and sleep well: Evening blue light filtration as a part of cognitive behavioural therapy for insomnia. *Chronobiology International*. 37(2), 248-259.

APPENDIX 3

Veldová, K., Bušková, J., Kopřivová, J. (2019). [Cognitive behavioural therapy for insomnia: changing sleep or its perception?]. *Psychiatrie*, 23(3):115-120.