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REVIEW ARTICLE

Reconsidering sleep perception in insomnia: from misperception to mismeasurement

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Summary

So-called 'sleep misperception' refers to a phenomenon in which individuals have the impression of sleeping little or not at all despite normal objective measures of sleep. It is unknown whether this subjective–objective mismatch truly reflects an abnormal perception of sleep, or whether it results from the inability of standard sleep recording techniques to capture 'wake-like' brain activity patterns that could account for feeling awake during sleep. Here, we systematically reviewed studies reporting sleep macro- and microstructural, metabolic, and mental correlates of sleep (mis)perception. Our findings suggest that most individuals tend to accurately estimate their sleep duration measured with polysomnography (PSG). In good sleepers, feeling awake during sleep is the rule at sleep onset, remains frequent in the first non-rapid eye movement sleep cycle and almost never occurs in rapid eye movement (REM) sleep. In contrast, there are patients with insomnia who consistently underestimate their sleep duration, regardless of how long they sleep. Unlike good sleepers, they continue to feel awake after the first sleep cycle and importantly, during REM sleep. Their mental activity during sleep is also more thought-like. Initial studies based on standard PSG parameters largely failed to show consistent differences in sleep macrostructure between these patients and controls. However, recent studies assessing sleep with more refined techniques have revealed that these patients show metabolic and microstructural electroencephalography changes that likely reflect a shift towards greater cortical activation during sleep and correlate with feeling awake. We discuss the significance of these correlates and conclude with open questions and possible ways to address them.

KEYWORDS
correlates, insomnia, sleep misperception, subjective-objective sleep discrepancy

1 | INTRODUCTION

The diagnosis of insomnia is currently based on purely subjective complaints, including difficulties falling or staying asleep despite adequate opportunities to do so, as well as resulting daytime symptoms such as fatigue (American Academy of Sleep Medicine, 2014). Not infrequently, sleep practitioners are confronted with complaints from patients that can be very dramatic, like 'I have not slept for weeks' or even 'months', which would hardly be sustainable and compatible with any daytime functioning. Unless there are reasons to suspect

comorbid sleep disorders, sleep recordings are generally not part of the standard evaluation of insomnia. However, when they are performed, they can be surprisingly unrevealing. Even in patients who state that they hardly slept in the laboratory, standard sleep parameters can fall entirely within the normal range. Various terms have been used throughout the years to describe such a mismatch between subjective and objective sleep reported by patients with insomnia, including 'sleep state misperception' (Thorpy, 1990), 'subjective insomnia' (Howard, 1979) or 'paradoxical insomnia' (American Academy of Sleep Medicine, 2005). In the current classification of sleep disorders, 'paradoxical insomnia' is listed as a chronic insomnia subtype without further specifications, reflecting the limited understanding of its physiopathology (American Academy of Sleep Medicine, 2014; Box 1). One fundamental question that arises is whether patients with so-called 'sleep misperception' truly underestimate their sleep duration and quality, or whether the techniques that are routinely used to measure and define sleep are inadequate to account for brain activity changes that may mediate feeling awake during sleep. Thus, the phenomenon of 'sleep misperception' raises questions that go beyond insomnia: what underlies our normal perception of sleep? Are there objective markers of sleep quality? And is the way we routinely measure sleep suited to assess insomnia complaints? Polysomnography (PSG), the current 'gold standard' in sleep medicine, only incompletely predicts subjective aspects of sleep (Kaplan et al., 2017) and it has long been postulated that more subtle changes in brain activity might be responsible for the apparently paradoxical perception of wake during sleep in insomnia patients (Perlis et al., 1997). In addition, it is now well established that localised sleep and wake patterns, which are not adequately captured by standard sleep recordings (PSG) and scoring methods, can coexist in both physiological and pathological conditions, and likely determine sleep-related conscious experiences (Siclari et al., 2017; Siclari & Tononi, 2017).

BOX 1 Historical development of the concept of subjective–objective sleep discrepancy.

In 1997, the *International Classification of Sleep Disorders* (ICSD) introduced the term of 'sleep state misperception' (ICSD-1; Thorpy, 2017) to refer to patients who greatly underestimated their sleep time, although presenting normal 'objective sleep' (Figure 1). This terminology referred specifically to patients reporting poor sleep quality despite normal sleep based on PSG measures. Later, 'paradoxical insomnia' officially entered the list of insomnia phenotypes in the ICSD-2 (American Academy of Sleep Medicine, 2005). This diagnosis described patients presenting consistent and marked subjective–objective discrepancy despite normal sleep, defined this time as a total sleep time (TST) of at least 6.5 h and a sleep efficiency >85% (Edinger et al., 2004). The usefulness of this diagnosis was subsequently debated. Not only did ICSD-2 paradoxical insomnia criteria show poor inter-rater agreement (Edinger et al., 2011); but subjective–objective discrepancy was also shown to be variably present in almost all insomnia subtypes (Vanable et al., 2000); with the exception of insomnia patients with objective short sleep duration who correctly estimated or over-estimated their sleep (Vgontzas et al., 2013).

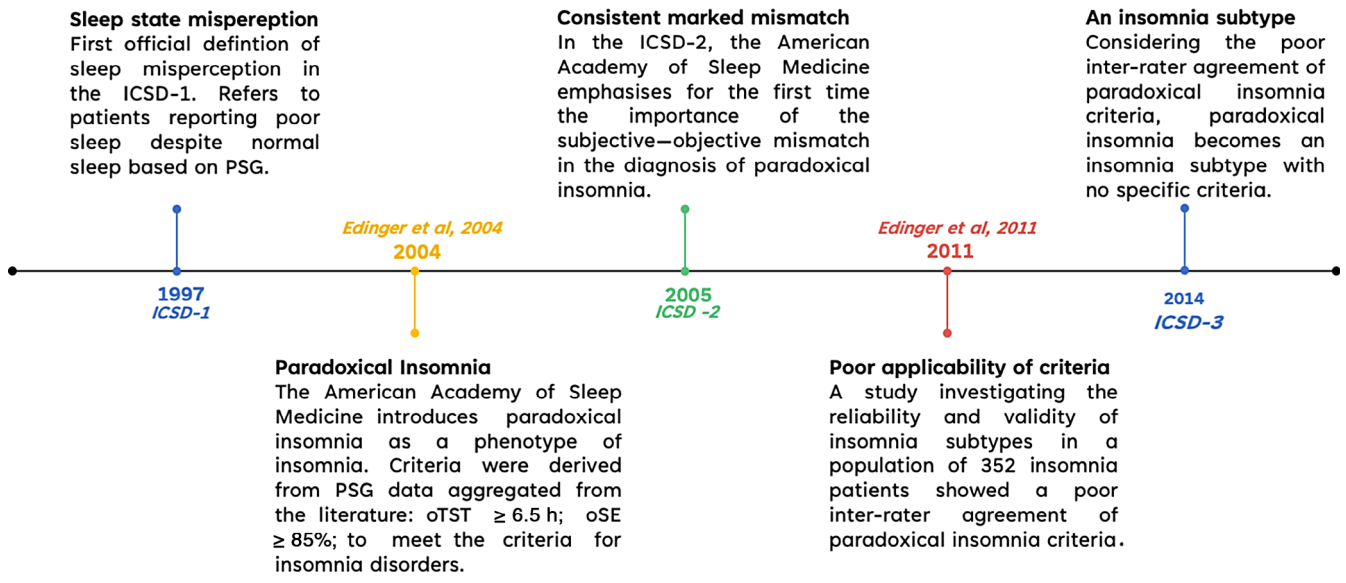

FIGURE 1 Changes in the clinical definition of sleep misperception in insomnia over time. ICSD, *International Classification of Sleep Disorders*; oSE, objective sleep efficiency; oTST, objective total sleep time; PSG, polysomnography. [Color figure can be viewed at wileyonlinelibrary.com]

With these considerations in mind, we aimed to systematically review studies reporting sleep macro- and microstructural, metabolic, and mental correlates of sleep (mis)perception. The article starts by providing an overview of how the perception of sleep is evaluated and of what constitutes 'normal' sleep perception in the general population and patients with insomnia. It then reviews the correlates of sleep perception, assesses the evidence for treatment benefits in this context, and concludes with open questions of possible ways to address them.

2 | METHODS

We conducted a comprehensive literature search using PubMed and Cochrane search engines to identify relevant publications on sleep (mis) perception and insomnia. Our search included publications with any of the following words in the title: 'insomnia', 'insomniac', 'paradoxical insomnia', 'sleep perception', 'sleep state misperception', 'subjective-objective discrepancy', 'subjective-objective sleep discrepancy', 'subjective sleep' associated to any of the following words in the title or abstract: 'dreaming', 'sleep perception', 'sleep state misperception', 'subjective-objective discrepancy', 'subjective-objective sleep discrepancy', 'underestimation of sleep'. The search resulted in 152 hits, which were screened for duplicates and relevance. The abstracts of these publications were inspected, 112 publications were excluded based on the following exclusion criteria: nocturnal subjective–objective discrepancy was not evaluated or investigated, case report, commentary paper, conference abstract. Finally, 43 additional relevant publications were found through references in other publications. Throughout the article, we use the term 'subjective–objective sleep discrepancy' (SOSD) to refer to a mismatch between subjective estimations of sleep and objective standard measurements of sleep (obtained with conventional scoring of PSG recordings).

2.1 | The assessment of SOSD

Subjective–objective sleep discrepancy is typically evaluated by comparing subjective assessments of sleep with objectively measured sleep parameters. *Subjective estimates* are usually collected through questionnaires, diary entries, or interviews taking place either after a night of sleep, and thus reflect a retrospective evaluation of the whole night of sleep, or after serial awakenings (Siclari et al., 2013), in which case estimates of a more momentaneous perception of sleep are obtained. *Objective sleep parameters* are usually derived from PSG or actigraphy and usually include total sleep time (TST), sleep onset latency (SOL), and wake after sleep onset (WASO). The SOSD is then

TABLE 1 Measures of subjective–objective discrepancy in the literature.

Measures	Referred to as	Perfect estimation	Sleep underestimation	Unit	Used in
Absolute discrepancy					
s–oSOL	SOL absolute discrepancy	0	<0	Min	Perlis et al., 2001; Parrino et al., 2009; Crönlein et al., 2019; Hermans et al., 2021; Lovato et al., 2021; Ma et al., 2021
o–sSOL	SOL absolute discrepancy	0	>0	Min	Kay et al., 2017; Perrault et al., 2022
s–oWASO	WASO absolute discrepancy	0	<0	Min	Perlis et al., 2001
o–sWASO	WASO absolute discrepancy	0	>0	Min	Feige et al., 2021
s–oTST	TST absolute discrepancy	0	<0	Min	Perlis et al., 2001; Parrino et al., 2009; Fernandez-Mendoza et al., 2011; Bianchi et al., 2013; Crönlein et al., 2019; Dzierzewski et al., 2019; Lindert et al., 2020; Lovato et al., 2021; Ma et al., 2021
o-sTST	TST absolute discrepancy	0	>0	Min	Krystal et al., 2002; Manconi, 2010; Perrault et al., 2022
Absolute ratio					
s/oSOL%	SOL absolute ratio	100	>100	%	Lecci et al., 2020
s/oWASO%	WASO absolute ratio	100	>100	%	Lecci et al., 2020
s/oTST%	TST absolute ratio	100	<100	%	Pinto et al., 2009; Choi et al., 2016; Lecci et al., 2020; Stephan et al., 2021; Lee, 2022; Perrault et al., 2022
Normalised discrepancy					
o–s/oSOL	SOL normalised discrepancy	0	>0		Maes et al., 2014
s–o/oTST	TST normalised discrepancy	0	<0		Means et al., 2003; Li et al., 2022; Valko, 2021
o–s/oTST	TST normalised discrepancy	0	>0		Krystal et al., 2002; Manconi, 2010; Normand et al., 2016; Lindert et al., 2020; Janků et al., 2020; Castelnovo et al., 2021

Abbreviations: o, objective; s, subjective; SOL, sleep onset latency; TST, total sleep time; WASO, wake after sleep onset.

BOX 2 Can insomniacs with objectively short sleep underestimate their sleep duration?

Some studies report a normal estimation or even an overestimation of sleep in insomnia with objectively short sleep (Fernandez-Mendoza et al., 2011; Krystal et al., 2002). Nevertheless, the distribution of subjective estimations in relation to objective sleep duration indicates that sleep underestimation can also occur in patients with objectively short sleep (Åkerstedt et al., 2016; Bastien et al., 2013; Lecci et al., 2020; Lovato et al., 2021; Valko et al., 2021;Figure 3). Thus, separating patients with insomnia on the sole basis of their objective sleep duration to define them as 'misperceptors' or not, without obtaining subjective evaluations, is at best, an oversimplification, and should be avoided.

calculated by relating the subjective to the objective assessment of these parameters in one the following ways (Table 1): (i) absolute discrepancy (subtraction); (ii) absolute ratio (division); and (iii) normalised discrepancy over objective measures (subtraction and division).

Some studies in patients with insomnia exclusively use objective measures of sleep (long versus short sleep time) to define sleep misperception, without assessing subjective parameters or a mismatch between the two. The implicit assumption underlying these studies is that normal sleep in patients with insomnia is the result of a 'misperception' and that patients with short sleep do not have SOSD. As discussed in Box 2, this assumption is problematic.

2.2 | The study of subjective–objective sleep discrepancy—what is the norm?

2.2.1 | Subjective–objective sleep discrepancy in the general population and in good sleepers

When assessed in the general population, in healthy individuals or good sleepers presenting no sleep complaints, subjective evaluations of sleep parameters are usually relatively accurate with respect to standard PSG measures (Table 2). For instance, 2092 individuals who were part of a general population cohort gave a retrospective sleep duration estimate that was 99.4% (± 6%) of their TST measured by PSG (mean absolute ratio; Lecci et al., 2020). This result is consistent with studies performed in healthy individuals and good sleepers who overestimated their TST, on average, by only 12 to 24 min (Fernandez-Mendoza et al., 2011; Ma et al., 2021; Manconi et al., 2010; Silva et al., 2007), estimated their TST within 0%–+6% of objective values (Krystal et al., 2002; Li et al., 2022; Manconi et al., 2010), and perceived their TST to be between 91% and 124% of the objective TST (Choi et al., 2016; Lecci et al., 2020; Pinto et al., 2009). One notable exception is a study in which good sleepers with short sleep durations (< 6 h) presented an important overestimation of their sleep duration, with an absolute discrepancy of TST of +126 min (Fernandez-Mendoza et al., 2011). In terms of sleep latency, good sleepers (Kay et al., 2015; Kay et al., 2017; Ma et al., 2021) greatly overestimated their SOL to N1, with an average ratio of 490% of the objective measure (Lecci et al., 2020), but estimated their SOL to N2 within 0–2 min of its objective value (Kay et al., 2015; Kay et al., 2017; Ma et al., 2021). With regards to WASO, good sleepers underestimated it by 20 min (Kay et al., 2015); with a ratio between subjective and objective values of 68% on average (Lecci et al., 2020).

> *Thus, individuals who do not present complaints of insomnia usually retrospectively overestimate their sleep latency to N1, precisely estimate their sleep duration and underestimate the time they spent awake after sleep onset with respect to standard PSG measures.*

Studies awakening and interrogating good sleepers in different stages of sleep and at different times of the night have shown that the feeling of being asleep sets in progressively and often after the onset of objective sleep. At the beginning of the night, when entering the first relatively stable period of N1 sleep, i.e., three consecutive 30-s epochs—as many as five out of 12 awakenings (42%) were followed by reports of feeling awake (Hsiao et al., 2018). At the onset of stable N2 sleep (i.e., three consecutive 30-s epochs of N2 (Hsiao et al., 2018; Yang et al., 2010) or 2 min after first sleep spindle (Bonnet & Moore, 1982), feeling awake was reported in nine out of 20 awakenings (31%) in a functional magnetic resonance imaging (fMRI) study (Hsiao et al., 2018); in 11 out of 20 (55%) awakenings in a PSG nap study (Yang et al., 2010), and in 64% of awakenings in a electroencephalography (EEG) study (Bonnet & Moore, 1982). At 5 min after the onset of stable N2 sleep, 20%–50% still reported feeling awake (Bonnet & Moore, 1982; Borkovec et al., 1981; Yang et al., 2010). The proportion of 'feeling awake' instances in the sleep onset period was shown to decrease steadily with time in one study, with 5% of individuals still experiencing it 16 min after the first sleep spindle, and none after half an hour (Bonnet & Moore, 1982). In studies assessing sleep perception across the whole night of sleep, good sleepers reported feeling awake much more frequently in non-rapid eye movement (NREM) than in rapid eye movement (REM) sleep (12%–19% [Feige et al., 2018; Stephan et al., 2021] versus 0%–8% to 3% [Feige et al., 2018; Stephan et al., 2021] of instances, respectively), irrespective of arousal thresholds (Feige et al., 2018). Surprisingly, in this study feeling awake occurred just as frequently in N3 sleep, traditionally called 'deep' sleep, as in N2 sleep (Stephan et al., 2021). Within NREM sleep, feeling awake was less frequent at >2 h of sleep

(30% in the first 2 h after lights off and < 10% the rest of the night (Stephan et al., 2021)).

> *In summary, good sleepers display a consistently delayed perception of sleep onset with regards to objective sleep parameters, with a remarkable number of individuals still feeling awake in the first NREM sleep cycle, before progressively feeling more asleep across the rest of the night. In REM sleep, on the other hand, good sleepers consistently feel deeply asleep.*

2.2.2 | Subjective–objective sleep discrepancy in insomnia

The reported extent of SOSD in patients with insomnia is more variable between studies than for good sleepers. It ranges from an underestimation of TST by 187 min or 34% of objective values (representing a ratio of 61% to 81% of the objective TST) to an overestimation of TST by 10 min or 7% (Bianchi et al., 2013; Choi et al., 2016; Dzierzewski et al., 2019; Fernandez-Mendoza et al., 2011; Lecci et al., 2020; Lee et al., 2021; Lovato et al., 2021; Ma et al., 2021; Manconi et al., 2010; Pinto et al., 2009; Valko et al., 2021). While most studies report an underestimation of TST in patients with insomnia, three publications report an overestimation (Fernandez-Mendoza et al., 2011; Krystal et al., 2002; Ma et al., 2021). In two out of three studies, only patients with short sleep duration (< 6 h [Fernandez-Mendoza et al., 2011] or < 6.5 h [Krystal et al., 2002]) overestimated their TST and were not different from the control group (Krystal et al., 2002) (Table 2). Among the studies that have directly compared SOSD in patients with insomnia to good sleepers, most have shown a greater underestimation of TST in the patients with insomnia compared to good sleepers (Fernandez-Mendoza et al., 2011; Lecci et al., 2020; Li et al., 2022; Manconi et al., 2010); while one publication has shown a comparable absence of underestimation in both populations (Ma et al., 2021). Across studies, patients with insomnia overestimated the time it took them to fall asleep between 7 and 95 min (Kay et al., 2015; Kay et al., 2017; Lee et al., 2021; Lovato et al., 2021; Ma et al., 2021; Valko et al., 2021), exceeding their objective SOL by 163% on average (Valko et al., 2021) and representing up to 416% of their objective SOL (Lecci et al., 2020). This overestimation of SOL was greater in patients with insomnia than in good sleepers in two studies (Kay et al., 2015; Kay et al., 2017) and comparable to good sleepers in two others (Lecci et al., 2020; Ma et al., 2021). Perceived WASO in insomnia has been reported to exceed objective WASO by up to +95 min (Kay et al., 2015; Lovato et al., 2021; Valko et al., 2021) or +38% (Valko et al., 2021) and to represent ~362% of their objective WASO (Lecci et al., 2020). In the only two publications comparing them to a control group, overestimation of WASO was greater in patients with insomnia (Kay et al., 2015; Lecci et al., 2020).

Although patients with insomnia can present with an important mismatch between objective measures and subjective estimations of their sleep, it might come as a surprise that both measures correlate significantly nonetheless (Bianchi et al., 2013; Bonnet & Moore, 1982; Lecci et al., 2020; Valko et al., 2021). Strong correlations have been shown for TST in a cohort of 2092 individuals (Lecci et al., 2020), in a mixed population of 92 patients with insomnia and 66 patients with sleep apnea (Bianchi et al., 2013) and in 2738 patients with diverse sleep disorders (Valko et al., 2021). In this last population, subjective–objective correlation was also observed for WASO and SOL. This correlation indicates that subjective sleep perception is not arbitrary but instead has a meaningful and reliable relationship with objective measures of sleep.

> *Thus, most studies report that individuals with insomnia consistently underestimate the time they slept and overestimated the time they spent awake, with the exception of two studies in patients with short objective sleep who tended to correctly or over-estimate their sleep duration* (Fernandez-Mendoza et al., 2011; Krystal et al., 2002) (Box 2/Figure 2).

Studies using serial awakening paradigms (Bianchi et al., 2013; Bonnet & Moore, 1982; Lecci et al., 2020; Valko et al., 2021) showed that both the 'delayed' perception of sleep onset and the propensity to feel awake during the rest of the night seen in good sleepers was accentuated in patients with insomnia (Borkovec et al., 1981; Rechtschaffen & Monroe, 1969). When awakened 5 min after N2 onset, between 88% and 96% of patients with insomnia felt awake during the night and 68% during a nap; all proportions were significantly greater than in the good sleeper control group (Borkovec et al., 1981; Yang et al., 2010). Similarly to good sleepers, the proportion of instances in which patients felt awake was strongest in the first 2 h of the night (Stephan et al., 2021). However, it was much higher than in good sleepers, reaching up to 75% of reports (Stephan et al., 2021). Furthermore, feeling awake during sleep remained high across the whole night, with 15%–45% of instances perceived as wake beyond the first 2 h of the night (Stephan et al., 2021). Patients reported feeling awake more frequently than good sleepers in all stages of sleep that were investigated: in 23%–50% of NREM (N2 and N3) awakenings (Feige et al., 2018; Mendelson, 1993; Mercer et al., 2002; Stephan et al., 2021) and 10%–53% of REM awakenings (Feige et al., 2018; Mendelson, 1993; Stephan et al., 2021). Importantly, the increased proportion of feeling awake in patients was not associated to a decreased arousal threshold compared to controls (Feige et al., 2018).

Unlike good sleepers, who typically exhibit a narrow distribution around an accurate estimation of their sleep, several large sample studies have shown a widespread distribution of SOSD among insomniacs, with a peak occurring at moderate to severe underestimation of sleep (Edinger & Fins, 1995; Lindert et al., 2020; Manconi et al., 2010) (Figure 3). Another noteworthy difference between patients with insomnia and good sleepers is the degree to which SOSD presents

TABLE 2 Misperception values in the different populations with different measures. [Color table can be viewed at wileyonlinelibrary.com]

Reference	Inclusion criteria (*N*)	Measure	HV (*N*)	INS	SSM (*N*)	Group comparison
SOL						
Kay et al., 2015	HV: GS (51) INS: primary (63)	s-oSOL, min	+2 min	+19 min		INS > HV
Kay et al., 2017	HV: GS (30) INS: primary (32)	s-oSOL N2, min	0 min	+9 min		INS > HV
Lee et al., 2021	INS: primary (110)	o-sSOL N2, min		+41 min		
Lovato et al., 2021	INS: sleep maintenance (91)	s-oSOL N2, min		+13 to +25 min		
Ma et al., 2021	HV: GS (126) INS: primary (73)	s-oSOL, min	0.6 min	+7 min		INS = HV
Valko et al., 2021	INS: primary (294)	s-oSOL, min		+38 min		
Lecci et al., 2020	HV: GS (24) SSM: sleep misperception (10)	s/oSOL N1, %	490 (84)		416 (104)	SSM = HV
Valko et al., 2021	INS: primary (294)	s-o/oSOL		+163%		
WASO						
Kay et al., 2015	HV: GS (51) INS: primary (63)	s-oWASO, min	−20 min	+18 min		INS > HV
Lovato et al., 2021	INS: sleep maintenance (91)	s-oWASO, min		+70 to +95 min		
Valko et al., 2021	INS: primary (294)	s-oWASO, min		−3 min		
Lecci et al., 2020	HV: GS (24) SSM: sleep misperception (10)	s/oWT, %	68%		362%	SSM > HV
Valko et al., 2021	INS: primary (294)	s-o/oWASO		+38%		
TST						
Bianchi et al., 2013	INS: primary (92)	s-oTST, min		−66 min		
Dzierzewski et al., 2019	INS: chronic (159)	s-oTST, min		−59 min		
Fernandez-Mendoza et al., 2011	HV: GS (724) INS: chronic (142)	s-oTST, min	+12/+126[a]	−60/+72 min		INS < HV
Lee et al., 2021	INS: primary (110)	s-oTST, min		−81 min		
Lovato et al., 2021	INS: sleep maintenance (91)	s-oTST, min		−96 to −60 min		
Ma et al., 2021	HV: GS (126) INS: primary (73)	s-oTST, min	+24 min	+10 min		INS = HV
Valko et al., 2021	INS: primary (294)	s-oTST, min		−48 min		
Manconi et al., 2010	HV: healthy controls (288) INS: chronic (159)	s-oTST, min	+20 min	−187 min		INS < HV
Lecci et al., 2020	HV: GS (24) SSM: sleep misperception (10)	s/oTST, %	97 %		61%	SSM < HV
Pinto et al., 2009	HV: GS (28) INS: primary (36)	s/oTST, %	124%	77%		
Choi et al., 2016	HV: GS (80) INS: chronic (69)	s/oTST, %	91 %	80 %		
Krystal et al., 2002	HV: GS (20) INS: sleep maintenance (18) SSM: objective sleep (12)	s-o/oTST, %	+4 %	+7.5%	−15 %	SSM < INS and GS INS = GS
Manconi et al., 2010	HV: healthy controls (288) INS: chronic (159)	s-o/oTST, %	+6 %		−35%/−99%	SSM < HV
Li et al., 2022	HV: GS (31) INS: chronic (33)	s-o/oTST, %	0 %		−14%	SSM < HV
Valko et al., 2021	INS: primary (294)	s-o/oTST, %		−12%		

Note: Reported average subjective–objective discrepancy values in populations of healthy controls, good sleepers, patients with insomnia with sleep misperception or insomniacs without misperception. The first column reports the type of insomnia and the size of the population in the study. Good sleepers are either individuals with no sleep complaint or with normal objective sleep. Healthy controls are individuals with no major pathologies whose sleep has not been assessed. The last column report group differences assessed in the publication. Red indicates publications in which individuals with insomnia had a lower value compared to healthy controls or insomniacs with subjective–objective sleep discrepancy had a lower value than insomniacs without or healthy controls; green indicates when they had a higher value and blue a statistically comparable value.

[a]First value comes from a group of normal objective sleep and second from a group of short objective sleep.

Abbreviations: GS, good sleepers; INS, insomnia without sleep misperception; o, objective; SSM, insomnia with sleep misperception; HV, healthy controls; s, subjective; SOL, sleep onset latency; TST, total sleep time; WASO, wake after sleep onset.

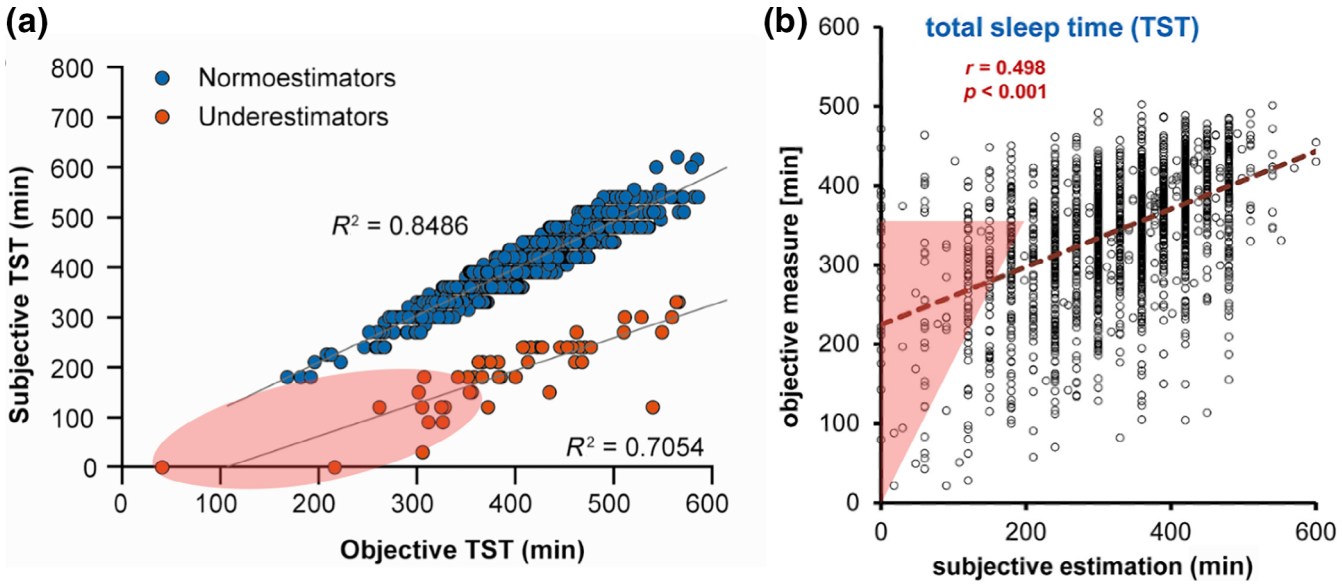

FIGURE 2 The presence of underestimation of sleep duration across a wide range of objective total sleep time (TST). Scatter plot representation of the relationship between subjective estimations and objective measures of TST in individuals accurately perceiving their sleep (blue, left panel), individuals underestimating their sleep (red, left panel) and a mixed population of individuals with sleep disorders (black, right panel). This figure highlights that sleep underestimation can be dissociated from sleep duration. As can be seen in the areas shaded in red, even in individuals with the shortest sleep durations (that would be considered 'objectively short' sleepers)—some underestimate their sleep duration. (adapted from Valko et al., 2021 and Lecci et al., 2020). [Color figure can be viewed at wileyonlinelibrary.com]

consistently across nights. Individuals with chronic insomnia experience SOSD more consistently across nights than good sleepers. For instance, a significant correlation between the proportion of instances perceived as wake in different recording nights was found only for patients with SOSD, and not for healthy controls in a serial awakening study (Stephan et al., 2021). Other studies seem to confirm that SOSD is consistently high in patients with insomnia and correlates with reported sleep difficulty in previous months (Lecci et al., 2020; Means et al., 2003). A notable exception was a study showing a subgroup of insomniacs presenting SOSD during home recordings but not during laboratory recordings (Edinger & Krystal, 2003).

> *In summary, patients with insomnia present an exaggerated and consistent delay in subjective sleep onset, more frequently feel awake while asleep beyond the first 2 h of sleep and particularly during REM sleep—a sleep stage in which good sleepers almost never feel awake.*

2.3 | The neural correlates of subjective–objective sleep discrepancy

To investigate the correlates of SOSD, three main methodologies have been used. Macro- or microstructural sleep parameters were either: (i) compared between a group of individuals with sleep state misperception to a group without (between group); (ii) correlated to SOSD derived from morning subjective reports obtained after whole night recordings (within groups); (iii) correlated with moment-to-moment perception of sleep obtained after serial awakenings (within individual).

2.3.1 | Between group comparisons: individuals with versus without subjective–objective sleep discrepancy

Sleep macrostructure

Studies comparing sleep macrostructure based on PSG between insomniacs with SOSD, insomniacs without SOSD, and good sleepers reported highly variable results (Table 3) (Andrillon et al., 2020; Bastien et al., 2013; Chouvarda et al., 2013; Feige et al., 2018; Krystal et al., 2002; Lecci et al., 2020; Parrino et al., 2009; Salin-pascual et al., 1992).

Studies comparing patients with SOSD to good sleepers did not reveal consistent differences in PSG parameters. Although half of the studies reported an increase in N1 and/or N2 (Andrillon et al., 2020; Lecci et al., 2020; Parrino et al., 2009; Salin-pascual et al., 1992) and a decrease in N3 sleep proportion in SOSD compared to good sleepers (Lecci et al., 2020; Parrino et al., 2009; Ren et al., 2023; Salin-pascual et al., 1992), the other half reported no differences in N1 (Andrillon et al., 2020; Bastien et al., 2013; Feige et al., 2018; Lecci et al., 2020), N2 (Bastien et al., 2013; Feige et al., 2018;Lecci et al., 2020; St-Jean et al., 2013), and/or N3 proportion (Feige et al., 2018; St-Jean et al., 2013). However, the two studies using the most severe cut-offs—a TST absolute discrepancy of 2 h (Parrino et al., 2009; Ren et al., 2023) and a TST absolute ratio under 60% in 2092 participants (Lecci et al., 2020)—reported consistent results, including a higher proportion of stage N1 and N2 sleep and/or a lower proportion of stage N3 in those with sleep misperception compared to good sleepers (Lecci et al., 2020; Parrino et al., 2009; Ren et al., 2023); as well as

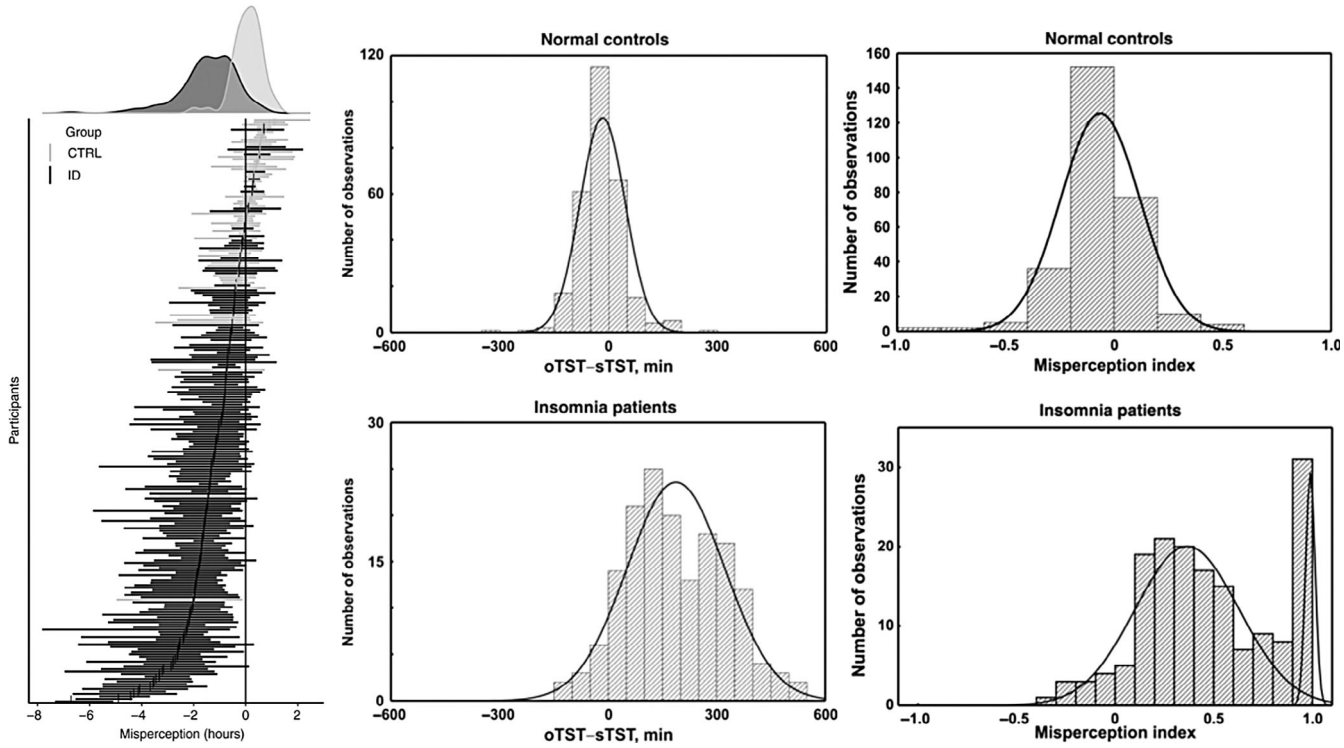

FIGURE 3 Distribution of subjective–objective sleep discrepancy (SOSD) measures in patients with insomnia and healthy controls. The distribution of absolute and normalised SOSD in total sleep time sharply peaks around perfect estimation in healthy controls (grey in left panel, top row in right panel); and is more widespread in patients with insomnia, ranging from accurate estimation to underestimation of 6–10 h in extreme cases (black in left panel, bottom row in right panel). (from Lindert et al., 2020; Manconi et al., 2010).

more wake time after lights off (Lecci et al., 2020) and more awakenings (Parrino et al., 2009). Such differences were not found when considering the studies investigating PSG measures within patients with insomnia (i.e., with versus without SOSD), which did not show consistent differences between the groups (Table 4).

> *Thus, consistent PSG correlates of SOSD were only found when comparing individuals with severe sleep misperception to good sleepers, and consisted of a higher proportion of lighter NREM sleep stages and a lower proportion of slow-wave sleep in patients with SOSD. Studies comparing PSG parameters within insomnia groups (with and without sleep misperception) showed inconsistent differences.*

Sleep microstructure

Studies comparing EEG power spectral density (the strength of the EEG activity in different frequency bands) between patients with insomnia with SOSD and good sleepers have consistently shown that compared to good sleepers, patients with SOSD present indices of a higher 'cortical activation' in NREM sleep, consisting in either lower delta power (Andrillon et al., 2020; Krystal et al., 2002; Lecci et al., 2020), increased power from the alpha band upwards, including sigma (Andrillon et al., 2020; Krystal et al., 2002), beta (Andrillon et al., 2020; Krystal et al., 2002; Lecci et al., 2020) and/or gamma power (Lecci et al., 2020; St-Jean et al., 2013) (Table 4) or a flatter 1/f slope (i.e., lower slow and higher fast EEG activity [Andrillon et al., 2020]). However, similarly to insomniacs with SOSD, insomniacs without SOSD also displayed indices of higher EEG activation compared to good sleepers, including decreased relative delta (Andrillon et al., 2020; Krystal et al., 2002); increased relative alpha, sigma and beta EEG power, and a decreased 1/f slope in stage N2 (Andrillon et al., 2020). However, some studies reported power spectral density differences that nonetheless distinguished the two populations of insomniacs (with and without SOSD), including lower relative delta (Krystal et al., 2002), theta (Andrillon et al., 2020; St-Jean et al., 2013), and alpha power (Andrillon et al., 2020), higher sigma and beta power (Krystal et al., 2002) and lower 1/f slope (Andrillon et al., 2020) in SOSD compared to insomniacs without SOSD (Table 4). Only two studies performed group comparisons in REM sleep (Lecci et al., 2020; St-Jean et al., 2013), only one observed the tendency for low-frequency power (delta and theta) to be lower and high-frequency power to be higher (beta) in patients with SOSD compared to good sleepers (St-Jean et al., 2013), similar to what other studies found in NREM sleep (St-Jean et al., 2013).

Other studies have investigated specific sleep elements including slow waves, spindles and microarousals. In one study, patients with insomnia with SOSD (here: with normal objective sleep measures)—presented fewer, slower and flatter EEG slow waves in N2 and N3 sleep, as well as more numerous, faster and smaller spindles compared to good sleepers, but comparable values to patients with insomnia without SOSD (Andrillon et al., 2020). Another study reported shorter

TABLE 3 Group comparison of standard polysomnography measures. [Color table can be viewed at wileyonlinelibrary.com]

Reference	Criteria (*N*)	SE	WASO	Lat. to 1st N1	Lat. to 1st N2	Lat. to 1st REM	N1, %	N1, min	N2, %	N2, min	N3, %	N3, min	REM, %	REM, min	AI, /h	Awk, *n*
SSM vs HV																
Andrillon et al., 2020	HV: good sleepers (89) SSM: objective sleep (59)	↑	↓		↓		=		↑		↑		↑		=	
Bastien et al., 2013	HV: good sleepers (30) SSM: sleep misperception (28)	↓	↑		↑		=		=		=		=			
Chouvarda et al., 2013	HV: good sleepers (10) SSM: N/A (10)									↑		=				
Feige et al., 2018	HV: good sleepers (42) SSM: sleep misperception (41)	↓	=		↑		=		=		=		=			
Krystal et al., 2002	HV: good sleepers (20) SSM: objective sleep (12)	↓	=	=				=		=		=		↑		
Lecci et al., 2020	HV: general pop (1147) SSM: sleep misperception (52)	↓	↑	=			↑		=		↓		=		↑	
Lecci et al., 2020	HV: good sleepers (24) SSM: sleep misperception (10)	=	=	=			=		=		↓		=		=	=
Parrino et al., 2009	HV: good sleepers (20) SSM: sleep misperception (20)			=			↑		↑		↓		=		↑	↑
Ren et al., 2023	HV: good sleepers (41) SSM: sleep misperception (89)	=	=			↓	=		=		↓		↑			
Salin-pascual et al., 1992	HV: good sleepers (7) SSM: sleep misperception (7)		=	=			↑		↑		↓		↓			
SSM vs INS																
Bastien et al., 2013	INS: psychophysiological (30) SSM: sleep misperception (28)	=	=		=		=		=		=		=			
Chouvarda et al., 2013	INS: psychophysiological (10) SSM: N/A (10)									=		=				
Krystal et al., 2002	INS: sleep maintenance (18) SSM: objective sleep (12)	↑	↓	=				=		↑		=		↑		
Lee et al., 2021	INS: PSQI >5 (33) SSM: sleep misperception (65)	↑	↓		↑		↓		=		=		=			
Liang et al, 2022	INS: chronic (80) SSM: sleep misperception (156)	↑	↓		↑		=		=		=		=			
Salin-pascual et al., 1992	INS: chronic (7) SSM: sleep misperception (7)		↓	↓			=		↓		↑		=			

Note: Top section indicates the contrast between patients with insomnia with sleep misperception (SSM) versus healthy controls (HV). Bottom section indicate the contrasts between patients with insomnia with (SSM) and without sleep misperception (INS). In both sections, the up arrow (green cells) indicates a higher value in SSM compared to the other group and a down arrow (red cells) indicates a lower value in SSM compared to the other group. Good sleepers are either individuals with no sleep complaint or with normal objective sleep.

Abbreviations: AI, Arousal Index; Awk, awakenings; HV, healthy controls; INS, insomnia without sleep misperception; N/A, not available; PSQI, Pittsburgh Sleep Quality Index; REM, rapid eye movement; SE, sleep efficiency; SSM, insomnia with sleep misperception; WASO, wake after sleep onset.

TABLE 4 Relative power differences between populations with and without sleep misperception and between moment-to-moment perception of wakefulness versus sleep. [Color table can be viewed at wileyonlinelibrary.com]

	Publication	Criteria (*N*)	Delta	Theta	Alpha	Sigma	Beta	Gamma	1/f
NREM sleep relative power									
SSM versus HV	Krystal et al., 2002	HV: good sleepers (20) SSM: objective sleep (12)	SSM < HV	SSM = HV	SSM > HV	SSM > HV	SSM > HV	SSM = HV	
	Lecci et al., 2020	HV: good sleepers (24) SSM: sleep misperception (10)	SSM < HV	SSM = HV	SSM = HV	SSM = HV	SSM > HV	SSM > HV	
	Andrillon et al., 2020	HV: good sleepers (89) SSM: objective sleep (59)	SSM < HV	SSM = HV	SSM > HV	SSM > HV	SSM > HV	SSM > HV	SSM < HC (American Academy of Sleep Medicine, 2014)
	St-Jean et al., 2013	HV: good sleepers (21) SSM: sleep misperception (20)	SSM > HV	SSM < HV	SSM < HV	SSM < HV	SSM = HV	SSM = HV	
SSM versus INS	Krystal et al., 2002	INS: sleep maintenance (18) SSM: objective sleep (12)	SSM < INS	SSM = HV	SSM = HV	SSM > INS	SSM > INS	SSM = HV	
	Andrillon et al., 2020	INS: chronic (288) SSM: objective sleep (59)	SSM = INS	SSM < INS	SSM < INS	SSM = INS	SSM = NS		SSM < INS (Thorpy, 1990)
	St-Jean et al., 2013	INS: psychophysiological (26) SSM: sleep misperception (20)	SSM = INS	SSM < INS	SSM = INS	SSM = INS	SSM = INS	SSM = INS	
FAW versus FAS	Hsiao et al., 2018	HV: good sleepers (36)	FAW = FAS	FAW < FAS	FAW > FAS	FAW = FAS	FAW > FAS	FAW > FAS	
REM sleep relative power									
SSM versus HV	Lecci et al., 2020	HV: good sleepers (24) SSM: sleep misperception (10)	SSM = HV	SSM = HV	SSM = HV	SSM = HV	SSM = HV	SSM = HV	
	St-Jean et al., 2013	HV: good sleepers (21) SSM: sleep misperception (20)	SSM < HV	SSM < HV	SSM < HV	SSM < HV	SSM = HV	SSM = HV	
SSM versus INS	St-Jean et al., 2013	INS: psychophysiological (26) SSM: sleep misperception (20)	SSM = INS	SSM < INS	SSM < INS	SSM < INS	SSM = INS	SSM = INS	
FAW versus FAS	Benz et al., 2020	HV: good sleepers (23) SSM: sleep misperception (22)	FAW < FAS	FAW < FAS	FAW < FAS	FAW = FAS	FAW = FAS	FAW = FAS	

Note: Top section reports results in NREM sleep and bottom section results in REM sleep. Red indicates cells where either patients with SSM or instances when individuals report FAW is inferior to the other group or state, and green indicate when SSM or FAW are superior to the other group or state. Good sleepers are either individuals with no sleep complaint or with normal objective sleep, American Academy of Sleep Medicine, 2014 *in N2 sleep only*; Thorpy, 1990 *in N3 sleep only*.

Abbreviations: FAS, instances when individuals reported feeling asleep; FAW, feeling awake in serial awakening paradigms; HV, healthy controls; INS, insomnia without sleep misperception; (N)REM, (non-) rapid eye movement; SSM, insomnia with sleep misperception.

sleep spindles in SOSD compared to good sleepers, but with comparable density, amplitude, and frequencies (Normand et al., 2016) (Table 5). The pattern of smaller and fewer sleep slow waves and faster sleep spindles likely reflects a state of relatively higher cortical/sub-cortical activation (Andrillon et al., 2011). The rate of cyclic alternating pattern (CAP) in NREM sleep (the alternation between states of intermittent EEG activation and deactivations [Parrino et al., 2012]) has been shown to be increased in N1 and N2 sleep (Chouvarda et al., 2013; Parrino et al., 2009). A recent study showed an increase in REM but not NREM microarousals in patients with insomnia with SOSD compared to good sleepers (Ren et al., 2023). However, in this study both CAP rate and microarousals in REM and NREM were shown to be comparable to patients with insomnia without SOSD (Andrillon et al., 2020; Chouvarda et al., 2013).

> *In summary, while macrostructural sleep parameters (PSG) do not appear to differ consistently between insomnia patients with SOSD and good sleepers, several studies have reported microstructural changes (spectral power density, slow waves and spindle parameters, CAP rate) that appear to reflect a higher degree of 'EEG activation' in insomnia patients with SOSD. These microstructural changes have less consistently been reported when comparing these patients to insomnia patients without SOSD, likely reflecting the highly variable definitions of SOSD across studies (for a review on this topic see* Castelnovo et al., 2019*)*

2.3.2 | Within-Group correlation

The few correlational studies investigating EEG power spectral density and SOSD tend to confirm that the EEG activation correlates with feeling awake during sleep. Overestimating SOL to N2 correlated with higher relative EEG activation (lower delta/beta EEG power) in patients with primary insomnia and good sleepers (Maes et al., 2014). Additionally, lower delta power in NREM sleep was shown to correlate with higher underestimation of TST across patients and good sleepers, while no correlations were found for other frequency bands (Krystal et al., 2002). Similarly, a third study found that underestimation of TST was associated with increased beta EEG power (Perlis et al., 2001). Only one study investigated the direct relationship between SOSD and microstructural sleep elements, and found no correlation between spindle parameters (number, density, duration, frequency, and amplitude) and TST normalised discrepancy; even when separating good sleepers, and patients with psychophysiological and paradoxical insomnia into over estimators and under estimators (Normand et al., 2016). The P2 component of auditory evoked responses, generally thought to reflect attentive processes, has been shown to positively correlate with sleep misperception in phasic REM sleep (Feige et al., 2021) and to be enlarged in patients with paradoxical insomnia with respect to patients with insomnia without SOSD and healthy controls (Bastien et al., 2013). Metabolic correlates of SOSD have been documented for SOL (Kay et al., 2017) and TST (Li et al., 2022). More specifically, SOSD in patients with insomnia positively correlated with glucose metabolism in the right anterior insula and middle/posterior cingulate cortex (Kay et al., 2017) in early NREM sleep and with connectivity between the right anterior insula and the right putamen and thalamus in wakefulness (Li et al., 2022), consistent with a higher metabolic activity in salience and arousal-related regions. Surprisingly, an opposite relation was found by both studies in good sleepers, which is not entirely understood.

2.3.3 | Within-subject contrast and correlations (serial awakening paradigms)

Serial awakening paradigms offer the opportunity to investigate the correlates of SOSD, rather than insomnia, while also providing greater statistical power by collecting multiple samples each night. In one of such rare studies, a group of 36 good sleepers had to indicate if they were awake or asleep in the moment preceding their interview, which was either in N1 or N2 sleep (Hsiao et al., 2018). Erroneously perceiving sleep as wake, regardless of sleep stage, was associated with lower relative theta and higher alpha, beta, and gamma power in frontal regions (Hsiao et al., 2018). In another study, comprising 787 serial awakenings, the degree of feeling awake during sleep in good sleepers and patients with insomnia correlated with increased high-frequency power (sigma and beta power) in NREM sleep (Stephan et al., 2021). The correlation was maximal in frontal and central brain regions, and declined in the anterior to posterior direction, being lowest or absent in inferior temporal and occipital regions (Stephan et al., 2021). This fast-frequency EEG correlate of feeling awake was more spatially diffuse in patients compared to good sleepers (Stephan et al., 2021). Such spatially widespread correlations between high-frequency power and feeling awake were found to persist in REM sleep only in patients with insomnia (Stephan et al., 2021). In another study, feeling awake in REM sleep was associated with lower low-frequency power (delta, theta, alpha) rather than higher high-frequency power (sigma, beta, gamma) compared to feeling asleep (Benz et al., 2020). This 'correlate' of feeling awake while asleep was comparable in both groups of individuals. Very few studies investigated sleep microstructural elements in moment-to-moment SOSD. One study reported that the density and amplitude of spindles in the 2 min preceding an awakening correlated negatively with perceived sleep depth in good sleepers and patients with insomnia with SOSD (Stephan et al., 2021). Another study reported that arousal density in REM sleep preceding an induced awakening was similar between instances of feeling asleep and feeling awake (Benz et al., 2020).

> *Taken together, studies evaluating the momentaneous perception of sleep through serial awakenings have shown that feeling awake during sleep is associated with a relative shift from lower to higher EEG frequencies, likely reflecting 'EEG activation'. Beyond these microstructural changes, techniques with a high spatial resolution*

TABLE 5 Differences in sleep microstructure between populations with and without sleep misperception. [Color table can be viewed at wileyonlinelibrary.com]

	Publication	Criteria (*N*)	SW quantity	SW speed	SW slope	SP density	SP speed	SP amplitude	SP frequency	SP duration	CAP
SSM versus HV	Andrillon et al., 2020	HV: good sleepers (89) SSM: objective sleep (59)	SSM < HV	SSM < HV	SSM < HV	SSM > HV	SSM > HV	SSM < HV			
	Chouvarda et al., 2013	HV: good sleepers (10) SSM: N/A (10)									SSM > HV
	Normand et al., 2016	HV: good sleepers (29) SSM: sleep misperception (17)				SSM = HV		SSM = HV	SSM = HV	SSM < HV	
	Parrino et al., 2009	HV: good sleepers SSM: sleep misperception									SSM > HV
SSM versus INS	Andrillon et al., 2020	INS: chronic (288) SSM: objective sleep (59)	SSM = INS	SSM = INS	SSM = INS	SSM = INS	SSM = INS	SSM = INS			
	Chouvarda et al., 2013	HV: good sleepers (10) SSM: N/A (10)									SSM = INS
	Normand et al., 2016	INS: chronic psychophysiological (24) SSM: sleep misperception (17)									

Note: Top section indicates the comparisons between patients with SSM and HV and the bottom section compares between patients with SSM and INS. Good sleepers are either individuals with no sleep complaint or with normal objective sleep. Red indicates cases where patients with SSM had an inferior value to the other group and green indicate when SSM had a superior value to the other group or state. Good sleepers are either individuals with no sleep complaint or with normal objective sleep.

Abbreviations: CAP, cyclic alternating pattern; HV, healthy controls; INS, insomnia without sleep misperception; N/A, not available; (N)REM, (non-) rapid eye movement; SP, sleep spindles; SSM, insomnia with sleep misperception; SW, slow wave.

(high-density EEG, nuclear imaging and fMRI), have revealed regional correlates of feeling awake during sleep in patients with insomnia, including increased metabolic activity and blood flow in the insula and cingulate cortex and high-frequency EEG activity in widespread anterior and central cortical areas.

2.3.4 | Cognitive and psychological factors influencing subjective–objective sleep discrepancy

Patients with insomnia with SOSD experience intrusive mental activity (rumination, worry) while attempting to fall asleep (Harvey, 2002); and rumination propensity (as evaluated with the rehearsal scale of the Emotional Control Questionnaire) was shown to correlate with lower sleep quality (assessed with the Pittsburgh Sleep Quality Index [PSQI] score) in a longitudinal study in 126 young adults (Thomsen et al., 2003). Moreover, cognitive arousal in the moments preceding sleep—such as the quantity of negative pre-sleep cognition about sleep and beliefs about the uncontrollable nature of sleep-onset—has been shown to correlate with overestimation of SOL in patients with sleep onset insomnia (van Egeren et al., 1983). In individuals without sleep disorders, sleep onset overestimation has also been shown to correlate with pre-sleep cognitive arousal and perceived somatic arousal—such as reported increased heart rate (Takano et al., 2016); while underestimating TST correlated with pre-sleep cognitive but not somatic arousal (Takano et al., 2016). In fact, inducing anxious or neutral cognitive arousal in good sleepers prior to a nap increased their TST underestimation compared to a control group (Tang & Harvey, 2004). Thus, pre-sleep emotional state might participate in SOSD (Perlis et al., 1997). In patients with insomnia, another interest is the nature of their nocturnal mentation. In studies using serial awakening paradigms, the only aspect of nocturnal mentation distinguishing patients with insomnia with SOSD from good sleepers was the more thought-like type of mentation in this group (Stephan et al., 2021; Wassing et al., 2016). This thought-like sleep mentation related to not being able to fall asleep in about one out of six awakenings in patients with SOSD, while such preoccupations were never mentioned by good sleepers (Stephan et al., 2021). This form of nocturnal mentation has been shown to increase with arousal density in REM sleep but not NREM sleep, to be correlated to self-reported hyperarousal and to be more present in patients presenting emotional distress lasting overnight, which correlated with insomnia severity (Wassing et al., 2016). Surprisingly, only two studies have directly investigated the relationship between dreams and SOSD. One study demonstrated that *high misperception* compared to *moderate misperception* (Castelnovo et al., 2021) was associated with reduced morning dream recall rate in patients with insomnia. Another study in lucid dreamers showed no correlation between self-reported dream frequency and SOSD (Ribeiro et al., 2020).

In summary, patients with insomnia struggle with intrusive thoughts (ruminations) while falling asleep. Their sleep mentation is more thought-like compared to controls and often relates to sleep difficulties. Some studies have shown these aspects to correlate to the extent to which patients present SOSD.

2.3.5 | The effect of treatment on subjective–objective sleep discrepancy

Cognitive behavioural therapy for insomnia (CBT-I) is a promising solution to SOSD as not only does it use sleep restriction, thus affecting macro- and microstructure of sleep (and potentially the EEG activation associated to SOSD), but it also targets cognition about one's own sleep and pre-sleep state of anxiety. CBT-I has been shown to significantly reduce sleep onset discrepancy (by −11 to −32 min and −169% of the absolute ratio) in patients with sleep onset insomnia, sleep maintenance insomnia and mixed insomnia (Janků et al., 2020; Kay et al., 2015;Lund et al., 2013; Perrault et al., 2022), effectively suppressing their SOSD (Lund et al., 2013; Perrault et al., 2022); while patients on a waiting list and patients receiving placebo stress management interventions had no significant changes SOSD (Lund et al., 2013; Perrault et al., 2022). The effect of CBT-I on sleep onset discrepancy was shown to be still significant 3 months after intervention (Perrault et al., 2022). With regards to TST, CBT-I has been shown to re-normalise SOSD absolute ratio (Perrault et al., 2022), reducing significantly absolute discrepancies by 20–60 min up to 12 months after intervention (Crönlein et al., 2019; Kay et al., 2015) and reducing relative discrepancy by 10% on the second week and by 22% on the sixth week of treatment (Janků et al., 2020) to values within the norms) (Perrault et al., 2022). This effect was not present in patients submitted to 'placebo' stress management intervention (Lund et al., 2013) or patients on a waiting list (Perrault et al., 2022). Finally, most publications also show a reduction in subjective objective WASO discrepancy by 22–60 min (Dzierzewski et al., 2019; Kay et al., 2015) up to 12 months after intervention (Dzierzewski et al., 2019). The change in WASO discrepancy after CBT-I therapy was shown to be predicted by the change in Insomnia Severity Index (ISI) score in older adults with insomnia (Kay et al., 2015). It remains unclear whether CBT-I effect on SOSD relates to its associated sleep restriction. In patients with psychophysiological insomnia, CBT-I was reported to increase delta power and decrease sigma and beta activity (Cervena et al., 2004), thus 'compensating' the previously described higher EEG activation associated to sleep misperception. Finally, a few studies investigated the effect of pharmacological treatment on sleep misperception. Triazolam (benzodiazepine), lorazepam (benzodiazepine) and zolpidem (non-benzodiazepine hypnotic) have been shown to reduce the likelihood of reports of wakefulness during serial awakening nights in patients with insomnia (Mendelson, 1993; Mendelson et al., 1988). Zoplicone (non-benzodiazepine hypnotic) has also been shown to decrease the subjective–objective SOL and TST discrepancy (Hermans et al., 2021). Surprisingly, both benzodiazepine and non-benzodiazepine hypnotics increase EEG activity in the sigma and beta range (Bastien

et al., 2003; Borbely et al., 1985; Brunner et al., 1991; Feinberg et al., 2000), which seems to be in contradiction with the correlation between EEG activity in these frequency bands and feeling awake during sleep (Bonnet et al., 1978; Johnson et al., 1976; Johnson et al., 1979; Johnson & Spinweber, 1983; Mendelson et al., 1988; Tan et al., 2003). This apparent paradox calls for further exploration of the neurochemical mechanism underlying feeling awake while asleep in insomnia (Box 3).

BOX 3 Open questions and research agenda.

Should we still talk about sleep misperception?

Studies using refined techniques to record and analyse brain activity have increasingly shown that feeling awake during sleep correlates with measurable shifts towards more 'wake-like' brain activity patterns. A legitimate question is therefore whether 'sleep misperception' as a concept still holds, or whether it reflects an inability of standard recording techniques to capture such wake-like activity intrusions into sleep. While there are still no definitive answers to the nature of this phenomenon, there are sufficient arguments to conceptualise it as a 'mis-measurement' rather than a 'misperception'.

How should sleep misperception be defined?

The large variability in the definition of sleep misperception across studies makes it difficult to conclude on whether sleep misperception represents a distinct insomnia entity, or whether it is present to various degrees in all forms of insomnia. Cut-offs separating patient populations should therefore be well motivated (as outlined in Castelnovo et al., 2019) and always be based on subjective evaluations of sleep durations given by patients instead of only objective criteria (Box 2). A way to circumvent the problem of definition altogether is to assess sleep perception systematically in large populations of patients with insomnia and use data-driven clustering analyses to determining whether subgroups with respect to sleep perception emerge (Benjamins et al., 2017).

Can we find markers of subjective sleep quality and understand their neurobiological basis?

Developing algorithms that automatically identify brain activity correlating with feeling awake during sleep (Hsiao et al., 2018; Kay et al., 2017; Stephan et al., 2021) would allow for the validation of these markers in larger populations. Such algorithms could also be adapted for use in the clinical setting. Animal studies have recently provided interesting EEG readouts of arousal system activity (for instance of the locus coeruleus), which appear to hold true for humans (Kjaerby et al., 2022; Lecci et al., 2017; Osorio-Forero et al., 2023). Translational studies of this type will be crucial to better understand which networks and neuro-modulatory systems are involved in sleep perception and to plan studies evaluating the effect of specific pharmacological manipulations on subjective sleep quality. Understanding how different arousal systems affect EEG activity will hopefully also allow to resolve some of the current perplexities regarding EEG markers of sleep perception (see discussion).

Which processes co-vary with subjective sleep perception?

Which sleep-related processes contribute to the feeling of being deeply asleep? Conceptually, one could imagine several processes that are either closely related or determine the feeling of having been deeply asleep, including for instance a profound sensory disconnection from the environment, a shift from thought-like to dream-like conscious experiences (Stephan et al., 2021), varying degrees of sleep inertia upon awakening or different degrees of amnesia for wake episodes (Perlis et al., 1997). To better understand these dimensions, and whether there are different forms of sleep perception, studies should be complemented with various behavioural measures like arousal thresholds (Feige et al., 2018), sensory stimulations (Feige et al., 2018) and reports of mental activity (Stephan et al., 2021).

What are the consequences of subjectively impaired sleep?

Insomnia has been conceptualised as a 24-h disorder, with measurable alterations in brain activity and function beyond the sleep period (Colombo et al., 2016). Previous studies have shown increased long-term health risks in patients with insomnia with objective short sleep duration compared to those with normal sleep duration (Bathgate et al., 2016; Fernandez-Mendoza, 2017), although poor sleep quality might be more predictive of future health issues than sleep duration (Tang et al., 2017). However, little is known about how low subjective sleep quality contributes to daytime functioning.

Which role does altered-REM sleep play in insomnia and sleep perception?

One of the most conspicuous differences that has emerged between good sleepers and patients with insomnia is the high degree of feeling awake specifically in REM sleep, a stage in which good sleepers consistently feel asleep (Feige et al., 2021; Stephan et al., 2021) and the duration of which predicts subjective perception of wake in insomnia (Feige et al., 2008). It has been postulated that REM sleep instability in patients could account for increased cortical and cognitive arousal (Feige et al., 2008; Riemann et al., 2012). Thus, focusing on REM sleep as a window to study the mechanisms underlying SOSD and insomnia appears particularly promising (Feige et al., 2008; Riemann et al., 2012; Van Someren, 2020; Wassing et al., 2016).

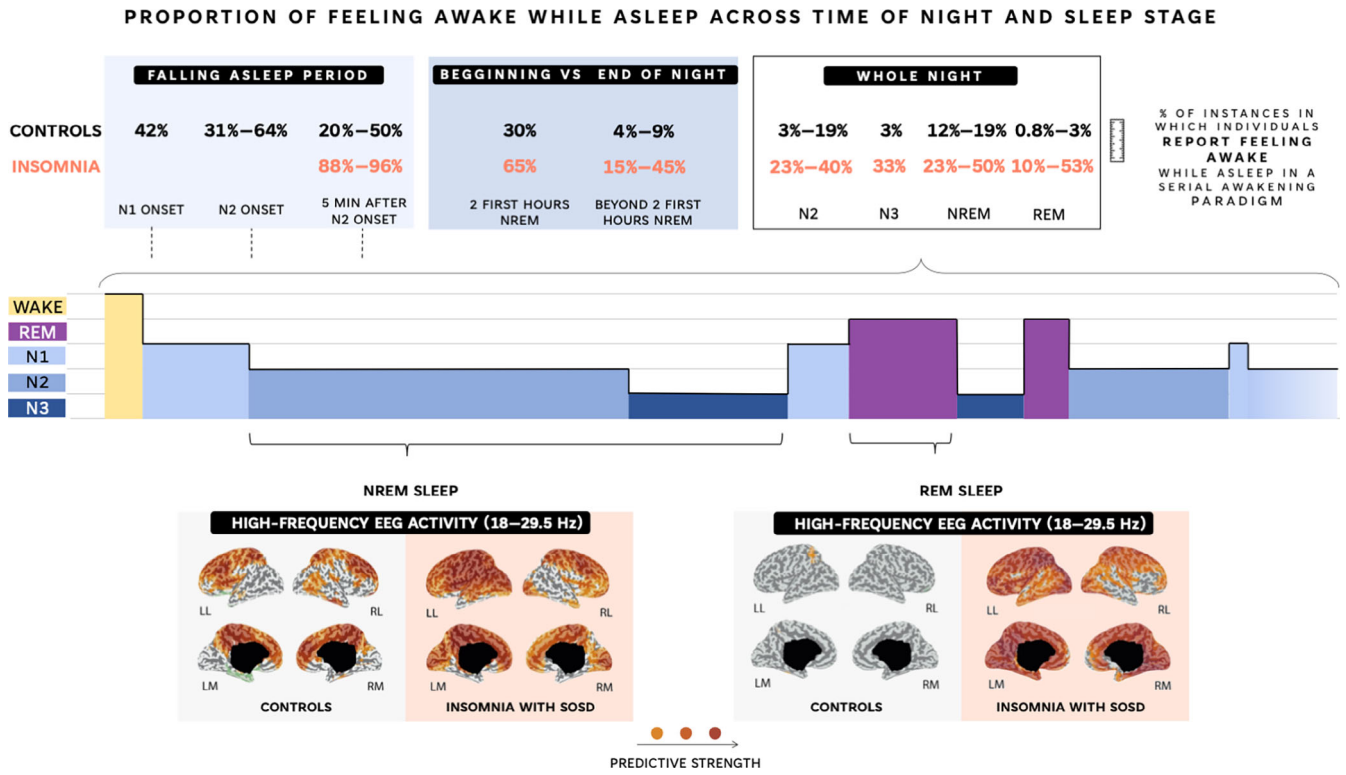

FIGURE 4 Feeling awake while asleep in patients with insomnia and good sleepers (controls with no sleep complaints). Top section: proportion of reports of feeling awake while asleep obtained by serial awakening studies in healthy controls (black) and patients with insomnia (orange). Bottom section: source localisation of beta EEG activity (18–29.5 Hz) predicting the degree of perceived sleep depth (red indicates that beta EEG activity predicts feeling relatively awake as opposed to asleep). EEG, electroencephalography; (N)REM, (non-) rapid eye movement; SOSD, subjective–objective sleep discrepancy. Adapted from Stephan et al., 2021). [Color figure can be viewed at wileyonlinelibrary.com]

Cognitive behavioural therapy for insomnia shows a promising effect on SOSD, significantly improving discrepancies in SOL, TST and WASO in various insomnia types, its effects persisting up to a year after treatment. The effect of pharmacological treatments on SOSD remains limited; however, benzodiazepines and non-benzodiazepine hypnotics have been shown to decrease wakefulness reports during sleep but paradoxically increase EEG activity, prompting further study into the neurochemical basis of SOSD.

3 | DISCUSSION

Our systematic review of the literature suggests that most individuals tend to accurately estimate their sleep duration measured with PSG. In good sleepers, instances of feeling awake during sleep are the rule at sleep onset, remain relatively frequent in the first NREM sleep cycle, including in deep (slow-wave) sleep, then decrease across the night and almost never occur in REM sleep. In contrast, patients with insomnia exhibit a more variable perception of their sleep. There are subgroups of patients who consistently underestimate their sleep duration, regardless of how long they actually sleep. Unlike good sleepers, these individuals continue to feel awake even after the first sleep cycle and, importantly, during REM sleep (Figure 4). Their mental activity tends to be more thought-like during sleep and to focus on not being able to sleep during both sleep and wakefulness. Initial studies based on standard PSG parameters largely failed to show significant or consistent objective differences in sleep macrostructure between these patients and controls. This absence of consistent abnormalities in sleep structure has posed a challenge for researchers and clinicians, raising doubts about the organic basis of the condition, and complicating the classification as an insomnia disorder (Edinger & Krystal, 2003). This inconsistency is hardly surprising, given the highly variable cut-offs in SOSD that have been used to classify patients as sleep 'misperceptors' (Castelnovo et al., 2019), calling for a consensus on the definition (Box 3). On the other hand, the absence of detectable alterations in sleep macrostructure in these patients may also result from the 'oversimplification' of sleep that results from current sleep recording and scoring techniques. Conventional PSG recordings, considered asthe 'gold standard' in clinical sleep medicine, typically comprise only few electrodes and standard sleep scoring averages sleep over long epochs (30 s). It has long been postulated that microstructural EEG changes, like increased beta activity in insomnia, might account for the patient's perception of being awake during their sleep (Perlis et al., 1997). In addition, it is now well established that sleep occurs and is regulated locally (Vantomme et al., 2019), and that regional variations of sleep patterns that account for subjective experiences during sleep (Siclari et al., 2017) are also not sufficiently

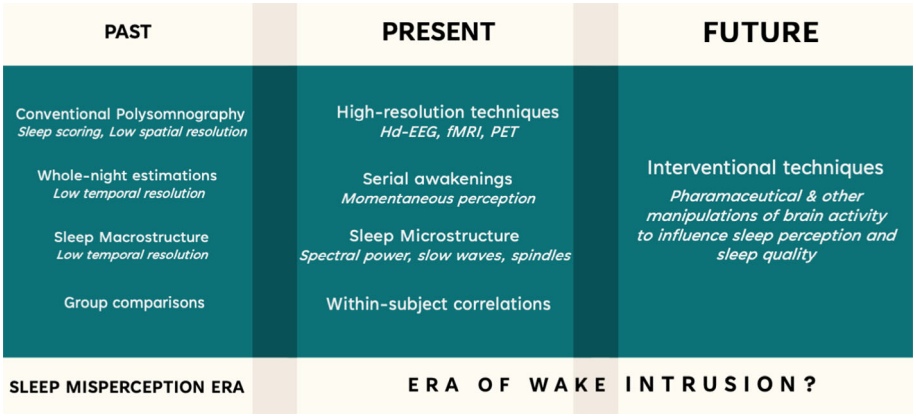

FIGURE 5 Current state of research and future directions. fMRI, functional magnetic resonance imaging; hd-EEG, high-density electroencephalography; PET, positron emission tomography. [Color figure can be viewed at wileyonlinelibrary.com]

captured by standard sleep measurements. Indeed, more recent studies, assessing microstructural instead of macrostructural sleep parameters found that patients with insomnia, including those who underestimate their actual sleep duration, show brain activity changes reflecting a relative shift towards greater EEG 'activation' compared to controls. These parameters most consistently include changes in power spectral density (i.e., a reduction of slow EEG and/or increases in fast EEG frequencies), fewer slow waves and faster spindles, suggesting a potential subthreshold activation of arousal systems. Some of these EEG parameters also directly correlate to the degree of 'feeling awake' during sleep (Benz et al., 2020; Hsiao et al., 2018; Stephan et al., 2021). Beyond these microstructural changes, techniques with a high spatial resolution (high-density EEG, nuclear imaging and fMRI), have revealed *regional* correlates of feeling awake during sleep in patients with insomnia, including increased metabolic activity and blood flow in the insula and cingulate cortex (Kay et al., 2017; Li et al., 2022; Stephan et al., 2021), and high-frequency EEG activity in widespread anterior and central cortical areas (Figure 4). However, the precise meaning of these correlates is not completely elucidated. For instance, REM sleep is characterised by an activated EEG but is associated with the lowest probability of feeling awake in good sleepers. In addition, some hypnotic treatments, which reduce the probability to feel awake during sleep, increase EEG activity in the beta range, which has been found to correlate with feeling awake (see Box 3 for future directions). Finally, CBT has shown some effectiveness on sleep misperception, as well as certain pharmacological treatments (like benzodiazepines or derivatives), although few have been specifically tested with respect to sleep perception.

In conclusion, while initial studies based on PSG suggested that sleep misperception in insomnia did not have an objective correlate, more recent studies evaluating sleep microstructure have revealed regional brain activity patterns that more consistently differ between patients with sleep misperception and controls, and correlate with feeling awake during sleep (Figure 5). By tying together this knowledge with the occurrence of feeling awake across the sleep stages and cycles, it now becomes possible to make hypotheses on which specific neuromodulatory systems may be dysregulated in insomnia and may account for sleep misperception (Stephan et al., 2021; Van Someren, 2020). In particular, noradrenergic hyperactivity in REM sleep has been discussed as a potential mechanism mediating abnormally feeling awake during this stage (Stephan et al., 2021) and emotional memory dysregulation in patients with insomnia (Van Someren, 2020). Future studies (Box 3) should therefore directly test the effect of specific pharmacological manipulations on the subjective perception of sleep. Finally, the existence of objective brain changes correlating with sleep misperception implies that sleep 'misperceptors' do not truly 'misperceive' their sleep but may in fact perceive subtle shifts towards wake-like brain activity during sleep remarkably well. As these shifts are not captured by standard scoring methods, the development and validation of EEG markers of subjective sleep quality should be a future research priority (Box 3).

AUTHOR CONTRIBUTIONS

Aurélie M. Stephan: Writing – review and editing; writing – original draft; data curation. **Francesca Siclari:** Writing – review and editing; conceptualization; supervision; writing – original draft.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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