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SCIENTIFIC INVESTIGATIONS

Underestimation of nocturnal sleep duration in central disorders of hypersomnolence: an underrecognized feature?

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Study Objectives: Sleep misperception is well-documented in insomnia but remains understudied in central disorders of hypersomnolence (CDH). This study aimed to examine (1) total sleep time (TST) misperception in CDH and healthy controls, (2) the prevalence of accurate estimators, underestimators, and overestimators, and (3) the relationship between misperception and polysomnography fragmentation variables.

Methods: We included 420 adults with CDH (38 narcolepsy type 1, 52 narcolepsy type 2, 192 idiopathic hypersomnia, 138 nonspecified hypersomnia) and 86 healthy controls tested in Montreal, Quebec, Canada. A replication cohort from a National Reference Center in France (n = 182; 79 narcolepsy type 1, 13 narcolepsy type 2, 35 idiopathic hypersomnia, 55 nonspecified hypersomnia) was also analyzed. Participants underwent full-night polysomnographies, Multiple Sleep Latency Tests, and clinical interviews. TST misperception was defined as the ratio between self-reported to objective TST. Group comparisons were performed using analyses of covariance adjusting for age and sex, and chi-square tests. Partial correlations were conducted to explore relationships between sleep fragmentation and TST misperception.

Results: In the Canadian cohort, all CDH subgroups underestimated their TST relative to controls ($P < .001$). The highest underestimation rates occurred in narcolepsy type 1 (44.7%) and idiopathic hypersomnia (26.6%), while the lowest was observed in healthy controls (11.6%). The French cohort confirmed the absence of significant differences in TST misperception between CDH subgroups. No correlations were found between polysomnography fragmentation variables and TST misperception.

Conclusions: Underestimation of nocturnal TST is common in adults with CDH and may complicate clinical assessment. These findings underscore the importance of integrating objective sleep measures when evaluating patients with hypersomnolence.

Keywords: central disorders of hypersomnolence, misperception, polysomnography, sleep fragmentation, total sleep time

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Sleep misperception, particularly the underestimation of total sleep time, is a well-known phenomenon in individuals with insomnia, often attributed to hyperarousal and sleep fragmentation. In contrast, little is known about the presence, frequency, and mechanisms of sleep misperception in central disorders of hypersomnolence (CDH), even though prolonged sleep is a defining feature, particularly in idiopathic hypersomnia. Understanding whether patients with CDH also exhibit total sleep time misperception is critical, as it could influence both diagnostic procedures and therapeutic strategies in these patients.

Study Impact: This study demonstrates that total sleep time underestimation is prevalent across CDH subgroups compared to healthy controls. Moreover, narcolepsy type 1 in the Canadian cohort and idiopathic hypersomnia in the French cohort showed mean misperception indices falling below the established cut-off, classifying them as underestimators. Notably, misperception in CDH was not linked to traditional polysomnography markers of sleep fragmentation, suggesting distinct underlying mechanisms from those observed in insomnia. These findings highlight the clinical relevance of sleep misperception in CDH and advocate for the systematic integration of objective sleep measures in the diagnostic process, such as polysomnography and actigraphy, to improve diagnostic accuracy and disease management.

INTRODUCTION

Sleep state misperception (SSM) refers to a discrepancy between objectively measured sleep parameters, such as those obtained through polysomnography (PSG), and an individual's perception of their sleep. Research has primarily focused on total sleep time (TST) underestimation in individuals with insomnia,[1,2] where mechanisms such as cortical hyperarousal

and increased fast electroencephalographic (EEG) activity have been proposed. Adults with insomnia often perceive frequent and brief awakenings detected on PSG as prolonged periods of wakefulness, resulting in a mismatch between PSG-measured TST and their perception, commonly referred to SSM in the literature.[3–6] While macrostructural PSG differences between adults with insomnia and controls remain inconsistent,[7–12] increased EEG spectral activity in alpha, beta, and sigma bands, along with reduced slow-wave activity, has been identified as neural correlates of SSM.[13–17] Although the term SSM suggests that the discrepancy originates from the individual's perception, it is important to acknowledge that part of this mismatch may also reflect inherent limitations of PSG in capturing the complexity of sleep–wake dynamics.[12] From this perspective, some cases described as SSM might equally be considered a "mismeasurement" of sleep, underscoring the need to interpret such discrepancies with caution.

More recently, some studies have proposed a broader model of SSM, including both ends of the sleep misperception spectrum, acknowledging that some individuals may overestimate their TST as well.[18,19] Trajanovic et al (2007) examined 136 individuals with various sleep disorders, including excessive daytime sleepiness (EDS) and narcolepsy, using overnight PSG, questionnaires, and the Multiple Sleep Latency Test (MSLT). They found that those who overestimated their TST experienced greater daytime sleepiness, with shorter MSLT sleep latencies (7.8 minutes) compared to those who underestimated or accurately estimated their TST (> 10.0 minutes).[18] These findings suggest that SSM extends beyond insomnia and may be present in other sleep disorders. However, the extent to which patients with central disorders of hypersomnolence (CDH) experience SSM compared to healthy controls remains underexplored.

CDH encompasses several sleep disorders, such as narcolepsy type 1 (NT1), narcolepsy type 2 (NT2), and idiopathic hypersomnia (IH), all characterized by EDS that interferes with daily functioning.[20] In CDH, particularly in IH, SSM can complicate both diagnosis and treatment. Indeed, an IH diagnosis is supported by a 24-hour TST of at least 660 minutes, measured via PSG or actigraphy. However, if TST is below this threshold, IH may still be diagnosed based on clinical judgment. In such cases, self-reported reports of prolonged sleep can provide supportive information.[20] Aside from the study mentioned above, only 3 others have examined the prevalence of SSM in large cohorts of individuals with sleep-wake disorders, and none included healthy controls.[21–23] Two studies reported that most patients with hypersomnia and/or narcolepsy accurately estimated their TST, with similar proportions of under and overestimators.[21,22] The only study specifically focusing on EDS compared SSM during MSLT daytime naps in 103 patients with CDH (NT1: n = 33, NT2: n = 14, IH: n = 56) and 62 individuals with obstructive sleep apnea or insufficient sleep.[23] No significant differences in SSM prevalence were found between groups. However, because these studies lacked healthy control groups and primarily focused on daytime naps, it remains unclear whether patients with CDH tend to systematically overestimate or underestimate their TST compared to individuals without sleep disorders.

To address these gaps, the present study evaluates TST perception in a large cohort of patients with CDH (NT1, NT2, IH and nonspecified hypersomnia [NSH]) and healthy controls using full-night in-laboratory PSG. Our objectives were to: (1) identify the frequency of TST underestimators, overestimators, and normoestimators in both CDH and healthy controls, (2) examine the relationship between TST perception and PSG-derived sleep variables associated with sleep fragmentation across the CDH subgroups and healthy controls, and (3) test the robustness of these findings in an independent replication cohort.

METHODS

Participants

We retrospectively selected 420 consecutive patients with CDH aged 18–60 (71.4% females, 34.5 ± 10.9 years), who were tested between 2000 and 2019 at the Center for Advanced Research in Sleep Medicine within the Centre Intégré Universitaire de Santé et de Services Sociaux du Nord -de -l'Île-de-Montréal, Montreal, Quebec, Canada. All participants who fulfilled the inclusion criteria outlined below and for whom complete PSG and MSLT data were available were included in the study. Each patient was diagnosed with CDH by a sleep physician based on a clinical interview and a full night of in-laboratory PSG, followed by an adapted MSLT with 4 naps. Diagnoses were subsequently revised according to the current *International Classification of Sleep Disorders*.[20]

All participants with CDH reported experiencing EDS for a minimum of 3 months. Specifically, the inclusion criteria for NT1 (n = 38) were: (1) cataplexy, (2) mean sleep onset latency ≤ 8 minutes on MSLT, (3) 2 or more sleep-onset rapid eye movement (REM) periods on the MSLT-PSG procedure, (4) cerebrospinal fluid hypocretin-1 concentration, measured by immunoreactivity, is either ≤ 110 pg/mL or < 1/3 of mean values obtained in normal individuals with the same standardized assay when measured. NT2 participants (n = 52) had to meet the following criteria: (1) no cataplexy, (2) mean sleep onset latency ≤ 8 minutes on MSLT, (3) 2 or more sleep-onset REM periods on MSLT-PSG procedure, (4) cerebrospinal fluid hypocretin-1 concentration either not measured or normal. IH (n = 192) was diagnosed when patients had: (1) no cataplexy, (2) PSG-MSLT findings not consistent with a diagnosis of NT1 or NT2, (3) mean sleep onset latency ≤ 8 minutes on MSLT and/or a TST on 24 hours ≥ 660 minutes. Most patients lacked actigraphy data or did not undergo ad-libitum PSG. However, according to the sleep physician's clinical judgment and the *International Classification of Sleep Disorders*, third edition, text revision guidelines,[20] patients who reported symptoms of hypersomnolence (eg, EDS, long sleep duration, or sleep inertia) but who did not meet objective PSG or MSLT criteria for NT1, NT2, or IH were classified as having NSH (n = 138).

Exclusion criteria for all CDH subgroups were: (1) any change in sleep disorder diagnosis over time; (2) neurologic comorbidities (eg, Parkinson's disease, epilepsy, multiple sclerosis, moderate to severe traumatic brain injury, dementia, stroke); (3) use of psychostimulant medication that could not be

stopped for the PSG recording; (4) less than 6 hours of sleep during the nighttime PSG; (5) major psychiatric disorders (eg, schizophrenia, bipolar disorder); (6) apnea-hypopnea index ≥ 15 events/h; and (7) shift work, circadian rhythm disorders, and chronic sleep deprivation.

For comparison, a group of 86 healthy controls aged 18–60 years old (51.2% females, 36.2 ± 11.6 years) were selected from 2 local databanks: The Montreal Archive of Sleep Studies[24] and Carrier's Lab databank.[25–28] Healthy controls were tested at the Center for Advanced Research in Sleep Medicine between 1999 and 2013 as part of research protocols. Exclusion criteria were the same as for participants with CDH and all healthy controls were free from medications that could influence PSG recordings or the nervous system. This study was approved by the local research ethics committee (REB 2020-1905).

Clinical interviews and questionnaires

At the sleep clinic, patients underwent a clinical interview conducted by a sleep medicine expert, who collected demographic and clinical data characterizing CDH. This included information on the presence of refreshing naps, self-reported experiences of prolonged sleep, current and past medical conditions, medication usage, experience of cataplexy or hallucinations, family history, etc. Patients completed questionnaires either on the night before or the morning after the PSG recording to assess sleepiness, sleep, and mood symptoms. Depression symptoms were evaluated using the Beck Depression Inventory,[29] with the severity of symptoms being categorized as minimal (0–13), mild (14–19), moderate (20–28), or severe (29–63). Anxiety symptoms were assessed using the Beck Anxiety Inventory,[30] with severity classified as minimal (0–7), mild (8–15), moderate (16–25), or severe (30–63). Self-reported sleepiness was measured using the Epworth Sleepiness Scale,[31] with scores ranging from 0–24, where higher scores indicate greater daytime sleepiness.

Sleep assessment and MSLT

Psychoactive medication was stopped at least 5 half-lives before the PSG recording. In the laboratory, bedtime and wake time were established in accordance with the participant's typical schedule; however, bedtime was not set earlier than 10:00 PM and wake time was not set later than 7:00 AM. Additionally, participants were instructed to avoid using their phones/laptops for 30 minutes before going to bed. Participants were assessed with an all-night in-laboratory video-PSG (Harmonie Stellate Systems, Montreal, Quebec, Canada), which included at least a 4-channel EEG montage (C3, C4, O1, O2). For recordings after 2013, additional electrodes (F3, F4, C3, C4, O1, O2) were used, all referred to earlobes with 10-kΩ resistance. The setup also included electrooculograms, an electrocardiogram, and surface electromyograms (submental and right and left anterior tibialis muscles). An oronasal cannula, an oronasal thermistor, and a thoracoabdominal strain gauge were used in addition to a transcutaneous finger pulse oximeter to measure respiratory variables and oxygen saturation. For patients with CDH, an adapted version of the MSLT was conducted the next day with 4 naps (9:00 AM, 11:00 AM, 1:00 PM, and 3:00 PM), and only the EEG, electrooculogram, and submental electromyogram were retained.[32] All PSG and MSLT recordings were rescored according to the 2023 American Academy of Sleep Medicine criteria by experienced sleep technologists, who were partially blinded to participants' clinical diagnosis and self-reported sleep estimate.[33] Sleep-onset REM periods were identified when REM sleep occurred within 15 minutes of sleep onset during both the nightly PSG and MSLT. The average sleep latency for each participant was measured during the MSLT.

For each sleep recording, we measured the following variables: TST, sleep onset latency, REM sleep onset latency, sleep efficiency, wake after sleep onset, duration and percentage of each stage (wake, stage 1 non-REM sleep, stage 2 non-REM sleep, stage 3 non-REM sleep, total non-REM sleep, and REM sleep), periodic legs movements during sleep index and apnea-hypopnea index. Apnea was defined as a reduction of airflow of ≥ 90% for at least 10 seconds, and hypopnea was defined as a reduction of airflow of ≥ 30% for at least 10 seconds associated with an oxygen desaturation ≥ 3% and/or an arousal.[33]

SSM measure

Measures of SSM were extracted by comparing the degree of mismatch between objective PSG and self-reported measures from the question "How long do you think your total sleep time was?" included in a morning questionnaire. We computed the Sleep State Misperception Index (SSMi), which is the ratio between self-reported and objective TST in percentage ([self-reported TST/objective TST] * 100).[16] A SSMi of 100% indicated perfect accuracy between self-reported and PSG-measured TST, while values less than 100% indicated underestimation and values greater than 100% indicated overestimation. Based on a previous population-based study that provided normative values, participants with a SSMi value < 88.31% were defined as underestimators, and the individuals with an SSMi > 110.43% were considered overestimators.[16] Of note, in this article, 1,252 individuals were tested, and 1,147 (95% of the sample) were identified as accurate estimators, 52 were underestimators (< 2.5th percentile) and 53 were overestimators (> 97.5th percentile).

Methods for independent cohort

A total of 182 patients with CDH (58% females; mean age 34.0 ± 15.3 years), diagnosed according to the *International Classification of Sleep Disorders*, third edition, text revision criteria (diagnoses subsequently revised), were tested from 2022–2024 at the National Reference Center for Rare Hypersomnia in Montpellier (France). No control group was available in this replication cohort and therefore, this cohort was mainly used to replicate group comparisons among CDH subgroups.

All participants underwent the same standardized evaluation protocol, which included a medical interview by a sleep expert, a full night in-laboratory video-PSG followed by a MSLT (5 naps), performed according to standard guidelines.[32,33] Patients with IH additionally underwent a 32-hour bed-rest PSG recording under controlled conditions. The cohort consisted of patients with a final diagnosis of NT1 (n = 79), NT2 (n = 13), IH (defined by mean sleep onset latency ≤ 8 minutes on MSLT and/or TST at bedrest > 19 hours/32; n = 35), and

NSH (n = 55). Similar to the Montreal cohort, patients with NSH were identified based on persistent complaints of hypersomnolence without meeting objective diagnostic criteria for other CDH subtypes. Of note, a recent study by the Montpellier team investigated sleep misperception in patients with CDH using an ad libitum sleep protocol.[34] Although conceptually related, the present analysis differs in that it examines TST misperception following a single standardized PSG night. A comparison of datasets indicated that 11 participants were included in both studies (2 NT1, 7 IH, and 2 NSH). Exclusion criteria were consistent with those applied to the cohort tested in Montreal, and none of the participants were using psychostimulant and/or psychoactive medication. Similar to the primary cohort, PSG recordings followed American Academy of Sleep Medicine-standard lead placements, including EEG (central C3, C4; occipital O2, referenced to the contralateral mastoid), left and right electrooculograms, chin electromyogram, annular/pressure transducer system, mouth thermistor, chest and abdominal bands, pulse oximeter, electromyogram electrodes on both anterior tibialis muscles, and electrocardiogram.[32] Sleep scoring was performed by expert sleep physicians, who were blinded to the clinical information and final diagnosis. The main features of the participants in the Montpellier cohort are detailed in **Table S1** in the supplemental material.

Statistical analyses

We used analyses of covariance to compare the 4 CDH subgroups (NT1, NT2, IH, NSH) and healthy controls on SSMi, adjusted for sex and age, and post hoc tests to determine where the differences were if the analysis of covariance was statistically significant. Moreover, we performed the Benjamini-Hochberg correction for multiple comparisons. We used a chi-squared test to analyze the Group differences for the proportion of under, over, and normoestimators. We also performed these analyses in a second independent cohort from Montpellier.

Only in the Montreal cohort, Partial Spearman's correlations corrected for sex and age were used to test the association between SSMi and PSG variables of sleep fragmentation (wake after sleep onset, awakening index, number of awakenings, sleep efficiency, and N1 and N3 sleep percentage) in the CDH subgroups and healthy controls.

Depressive and anxiety symptom scores were not included in the main models due to potential multicollinearity and conceptual overlap with hypersomnolence symptoms. Additionally, Beck Depression Inventory data were largely unavailable for healthy controls (n = 5).

All statistical analyses were performed using JASP, Version 0.19.2 (JASP Team, University of Amsterdam, Amsterdam, the Netherlands) and IBM SPSS Statistics, Version 29.0.2.0 (IBM Corp., Armonk, NY, USA). Significance levels were set at $P < .05$.

RESULTS

Sample clinical features

Table 1 presents participants demographic, clinical, PSG, and MSLT data and statistics. There was a higher percentage of females in the NSH compared to the narcolepsy groups and the healthy control group. Healthy controls had a lower BMI compared to the NT1 and both IH and NSH groups. Participants with NT1 reported more severe EDS (as measured by the Epworth Sleepiness Scale total score) compared to other CDH groups, whereas the healthy controls had fewer anxiety symptoms than all CDH groups. As expected, during the MSLT protocol, participants with NSH had longer sleep onset latency compared to the other CDH groups.

During the PSG, participants with NT1 experienced more sleep fragmentation (as indicated by WASO and the number of awakenings) and reduced TST, sleep efficiency, and REM latency compared to IH, NSH and healthy controls. In contrast, participants with IH showed longer TST and higher sleep efficiency than the other groups.

SSM index in CDH groups and the control group

We found a significant Group effect on the SSMi ($P < .001$), where all CDH subgroups underestimated their TST when compared to healthy controls (mean SSMi: 103.71 ± 15.03%): NT1 (86.83 ± 22.97%, $P < .001$, Benjamini-Hochberg adjusted P value: $P = .002$), NT2 (94.21 ± 20.39%, $P = .039$, Benjamini-Hochberg adjusted P value: $P = .039$), IH (94.95 ± 14.51%, $P < .001$, Benjamini-Hochberg adjusted P value: $P = .002$), NSH (94.89 ± 20.08%, $P = .002$, Benjamini-Hochberg adjusted P value: $P = .0026$) (**Figure 1**). No significant differences were observed between the CDH subgroups.

Frequency of under, over, and normoestimators in CDH groups and the control group

The chi-squared test revealed a significant group effect between CDH subgroups and healthy controls ($\chi^2 = 23.954$, $P = .002$). Specifically, the NT1 group had a significantly higher proportion of underestimators (44.7%) compared to healthy controls (11.6%). Furthermore, the IH group showed a significantly lower percentage of overestimators (12%) relative to healthy controls, who demonstrated a higher prevalence of overestimation (26.7%), followed by the NT2 group (13.5%). In contrast, the proportion of normoestimators was comparable across healthy controls, NT2, IH, and NSH groups, ranging from 60.9–63.5%. Detailed group distributions are presented in **Figure 2**.

Partial correlation analyses between sleep fragmentation variables and SSMi

In line with our third objective, we conducted partial correlation analyses, corrected for age and sex, between the SSMi and several PSG variables in each group. We found a negative correlation between SSMi and the number of awakenings ($r = -.272$, $P = .012$), as well as between SSMi and N1 sleep percentage ($r = -.228$, $P = .037$) in the healthy controls group only (**Figure 3**). No other statistically significant associations were found.

Results for independent cohort

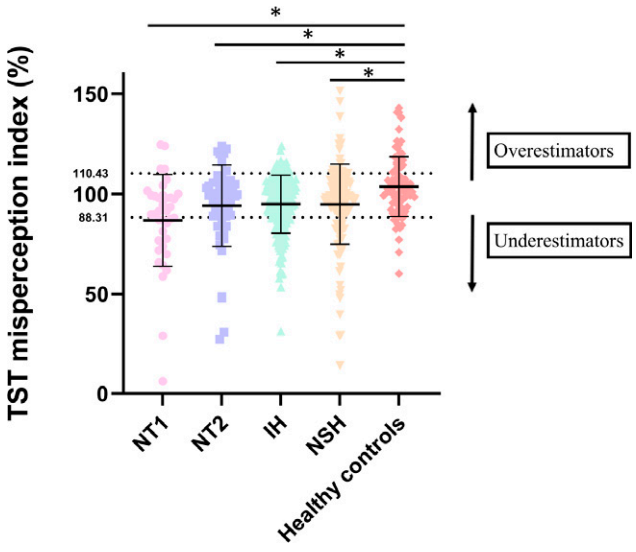
When comparing CDH subgroups in a second independent cohort, we found no significant differences on the SSMi between CDH subgroups (NT1: 94.42 ± 24.23%; NT2: 102.51 ± 11.31%; IH: 87.90 ± 22.85%; NSH: 94.05 ± 25.87%; $P = .319$). However,

Table 1—Demographic, clinical, polysomnography and MSLT characteristics of participants.

	NT1, A (n = 38)	NT2, B (n = 52)	IH, C (n = 192)	NSH, D (n = 138)	Healthy Controls, E (n = 86)	*P*, Effect Size or Cramer's V	Group Differences
Demographic/Clinical Data							
Females (n; %)	24, 63.2%	31, 59.6%	135, 70.3%	110, 79.7%	44, 51.2%	< .001, .211	D > A, B, E C > E
Age (years)	33.6 ± 10.4	31.3 ± 11.5	35.8 ± 10.8	34.2 ± 10.7	36.2 ± 11.7	.051, .019	
BMI (kg/m^2)	27.8 ± 6.5	25.1 ± 5.00	26.2 ± 5.1	25.5 ± 6.5	23.2 ± 3.6	< .001, .051	E < A, C, D
ESS score	18.7 ± 2.8	16.0 ± 4.2	16.5 ± 4.1	16.2 ± 3.7	NA	.005, .032	A > B, C, D
Beck Depression Inventory score	12.5 ± 8.5	12.9 ± 9.4	14.4 ± 10.8	11.6 ± 8.1	NA	.147, .018	
Beck Anxiety Inventory score	10.0 ± 9.4	9.3 ± 9.2	9.7 ± 7.9	9.4 ± 8.2	1.2 ± 1.9	< .001, .180	E < A, B, C, D
MSLT Data							
Mean sleep onset latency (minutes)	2.8 ± 2.1	3.9 ± 2.8	4.8 ± 1.9	11.5 ± 2.8	NA	< .001, .676	A < C, D > A, B, C
Polysomnographic Data							
Total sleep time (minutes)	434.1 ± 42.6	450.9 ± 46.2	460.7 ± 37.3	446.1 ± 43.4	406.9 ± 46.8	< .001, .168	A < C, E < A, B, C, D C > D
Sleep onset latency (minutes)	6.2 ± 8.4	10.5 ± 23.7	9.1 ± 7.5	14.9 ± 11.4	12.0 ± 9.6	< .001, .053	A, C < D
REM sleep latency (minutes)	58.7 ± 82.5	68.6 ± 48.8	108.3 ± 62.1	110.4 ± 59.3	99.1 ± 55.2	< .001, .073	A, B < C, D, E
WASO (minutes)	58.1 ± 40.8	42.5 ± 30.4	32.7 ± 20.9	41.2 ± 29.0	50.7 ± 39.3	< .001, .069	A > C, D C < E
Number of awakenings	32.0 ± 15.5	27.5 ± 14.2	23.4 ± 9.9	23.8 ± 11.2	25.8 ± 10.8	< .001, .043	A > C, D
Sleep efficiency (%)	88.1 ± 8.2	91.3 ± 6.1	93.1 ± 4.8	91.4 ± 6.1	89.1 ± 8.2	< .001, .070	A < C, D C > E
N1 (%)	14.5 ± 7.2	11.4 ± 6.1	10.4 ± 5.7	10.1 ± 5.7	9.8 ± 4.6	< .001, .042	A > C, D, E
N2 (%)	53.3 ± 8.8	54.3 ± 8.2	55.3 ± 8.8	54.9 ± 8.8	58.2 ± 7.1	.011, .025	A < E
N3 (%)	12.2 ± 9.3	13.3 ± 9.4	14.7 ± 8.6	15.0 ± 9.3	11.9 ± 8.4	.041, .020	
REM sleep (%)	20.4 ± 6.9	21.0 ± 6.6	19.5 ± 6.9	19.9 ± 6.5	20.1 ± 4.7	.683, .005	
Sleep stage transition index*	25.5 ± 8.7	23.9 ± 7.8	23.7 ± 7.1	22.7 ± 7.3	22.9 ± 6.4	.262, .010	
Microarousal index (events/h of sleep)	11.7 ± 4.9	11.0 ± 7.2	10.1 ± 6.1	9.4 ± 6.5	9.3 ± 4.8	.140, .014	
AHI (events/h of sleep)	3.6 ± 3.1	2.5 ± 2.7	2.7 ± 3.2	1.8 ± 2.7	0.9 ± 1.8	< .001, .064	A > D, E < A, B, C,
PLMS index (events/h of sleep)	18.7 ± 17.2	11.1 ± 11.5	13.0 ± 18.4	10.7 ± 15.3	5.4 ± 9.8	< .001, .048	E < A, C,

The results are expressed as the mean ± standard deviation for continuous variables, and as the number of participants and percentage for categorical variables. *The sleep stage transition index was calculated as the number of sleep stage transitions per hour of sleep. AHI = apnea-hypopnea index, BMI = body mass index, ESS = Epworth Sleepiness Scale, IH = idiopathic hypersomnia, MSLT = Mean Sleep Latency Test, n = number, N1 = stage 1 non-REM sleep, N2 = stage 2 non-REM sleep, N3 = stage 3 non-REM sleep, NSH = nonspecified hypersomnia, NT1 = narcolepsy type 1, NT2 = narcolepsy type 2, PLMS = periodic leg movements in sleep, REM = rapid eye movement, WASO = wake after sleep onset.

Figure 1—Distribution of individual scores on TST misperception index in patients with CDH and healthy participants.



The y-axis represents the TST misperception index (%) for each CDH subgroup and healthy controls (on the x-axis). A TST misperception index of 100% indicates a perfect self-reported estimation of the TST with respect to the TST obtained with the objective measures. Values below 100% indicate an underestimation, and values above 100% represent an overestimation. The dotted line above 100 represents the cut-off above which participants are considered overestimators (110.43%); conversely, the dotted line below 100 indicates the cut-off below which patients are considered underestimators (88.31%) based on a study that provided normative values.[16] CDH = central disorders of hypersomnolence, IH = idiopathic hypersomnia, NSH = nonspecified hypersomnia, NT1 = narcolepsy type 1, NT2 = narcolepsy type 2, TST = total sleep time.

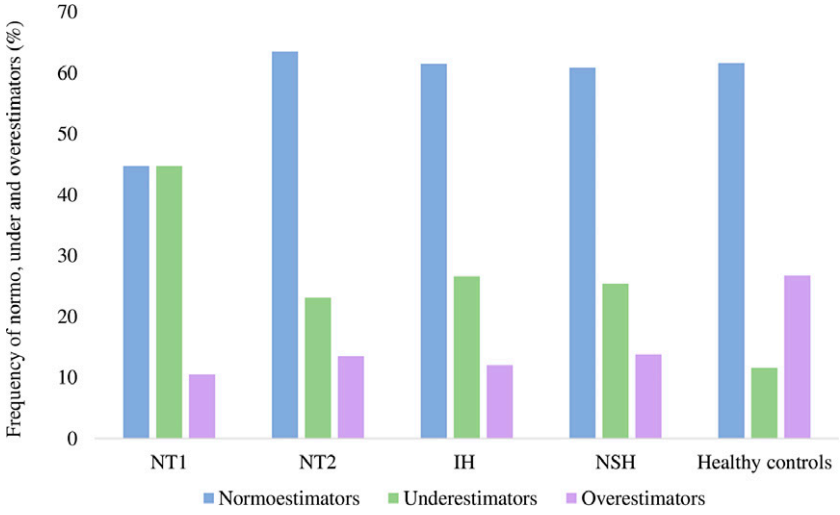
the IH group exhibited a mean SSMi value consistent with the "underestimator" category. Individual results are presented in Figure 4.

Figure 5 presents the frequency of TST underestimation, overestimation, and normoestimation across CDH subgroups in the France cohort. The chi-squared test revealed no significant group effect among the CDH subgroups ($\chi^2 = 6.302$, $P = .390$). Qualitatively, the IH group exhibited the highest proportion of underestimators (42.9%), followed by the NT1 group (38.0%). In contrast, individuals with NT2 exhibited a higher proportion of both normoestimators (69.2%) and overestimators (23.1%).

Sensitivity analyses

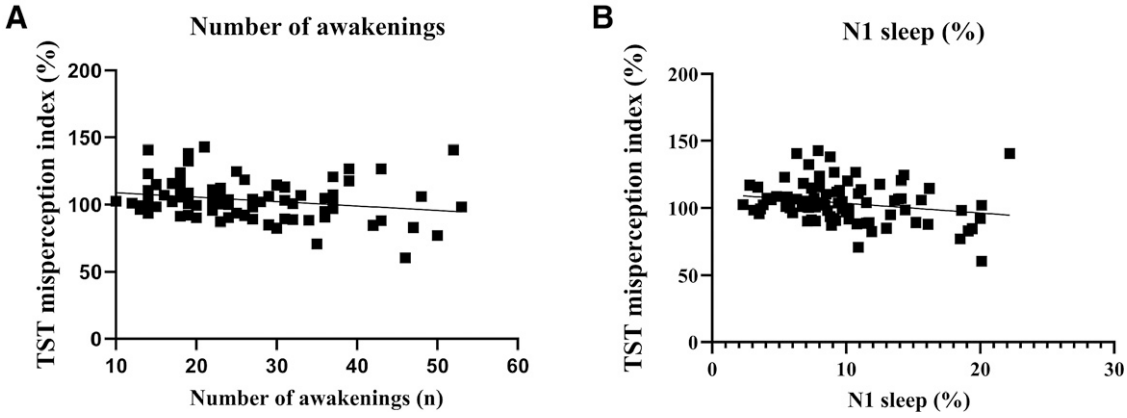
Although psychoactive medication was stopped at least 5 half-lives before the PSG recording, some CDH participants in the Montreal cohort were taking medications that could potentially influence sleep architecture (eg, antidepressants, anxiolytics, hypnotics, opioids). We conducted a sensitivity analysis to verify the robustness of our findings. After excluding all patients who reported the use of such medications during the PSG night, an analysis was performed on a subsample of 24 NT1, 48 NT2, 121 IH, and 113 patients with NSH, along with 86 healthy controls. We observed a significant Group effect on the SSMi ($P < .001$), with all CDH subgroups showing a marked underestimation of their TST compared to healthy controls (mean SSMi: 103.71 ± 15.03%). Specifically, SSMi was significantly lower in NT1 (84.59 ± 22.49%, $P < .001$; Benjamini-Hochberg adjusted $P = .004$), NT2 (93.60 ± 21.08%, $P = .019$; adjusted $P = .0019$), IH (94.91 ± 14.82%, $P = .006$; adjusted $P = .008$), and NSH (94.54 ± 20.37%, $P = .004$; adjusted $P = .008$) relative to controls. No significant differences emerged among the CDH subgroups.

Figure 2—Proportion of normoestimators, underestimators, and overestimators in patients with CDH and healthy participants.



The x-axis displays the classification of estimation types, normoestimators, underestimators, and overestimators, within each participant subgroup: NT1, NT2, IH, NSH, and healthy controls. The y-axis represents the proportion of participants in each category, expressed as a percentage. CDH = central disorders of hypersomnolence, IH = idiopathic hypersomnia, NSH = nonspecified hypersomnia, NT1 = narcolepsy type 1, NT2 = narcolepsy type 2.

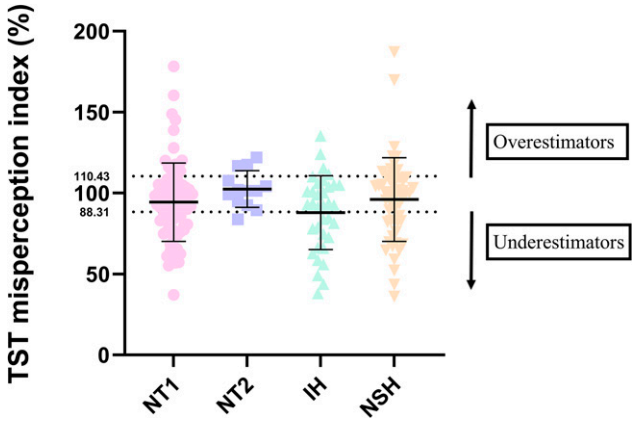
Figure 3—Association between total sleep time misperception index and number of awakenings and proportion of N1 sleep in healthy controls.



Association between total sleep time misperception index and number of awakenings **(A)** and proportion of N1 sleep **(B)** in healthy controls. Partial correlations with 95% confidence intervals between the TST misperception index and sleep fragmentation variables, adjusted for age and sex, in healthy controls. Scatterplots display individual data points. **(A)** A higher TST misperception index was associated with fewer number of awakenings (count). **(B)** A higher TST misperception index was associated with a lower percentage of N1 sleep. N1 = stage 1 non-REM sleep, TST = total sleep time.

We also conducted sensitivity analyses to further assess our findings regarding the TST misperception index using an alternative method. Specifically, we calculated the relative mismatch using the following formula: (TST self-reported – TST objective)/TST objective * 100,[22,35] and compared the Group based on this alternative SSMi. In addition, following previous research,[22] we categorized the relative mismatch as follows: values between −10% and 10% were considered accurate estimations; −10% to −30% as mild underestimation; −30% to −50% as moderate underestimation; −50% to −100% as severe underestimation; 10–30% as mild overestimation; 30–50% as moderate overestimation; and values above 50% as severe overestimation. Group differences in the distribution of estimation categories were assessed using a chi-squared test (see supplemental materials for detailed results).

Figure 4—Distribution of individual scores on TST misperception index in patients with CDH.

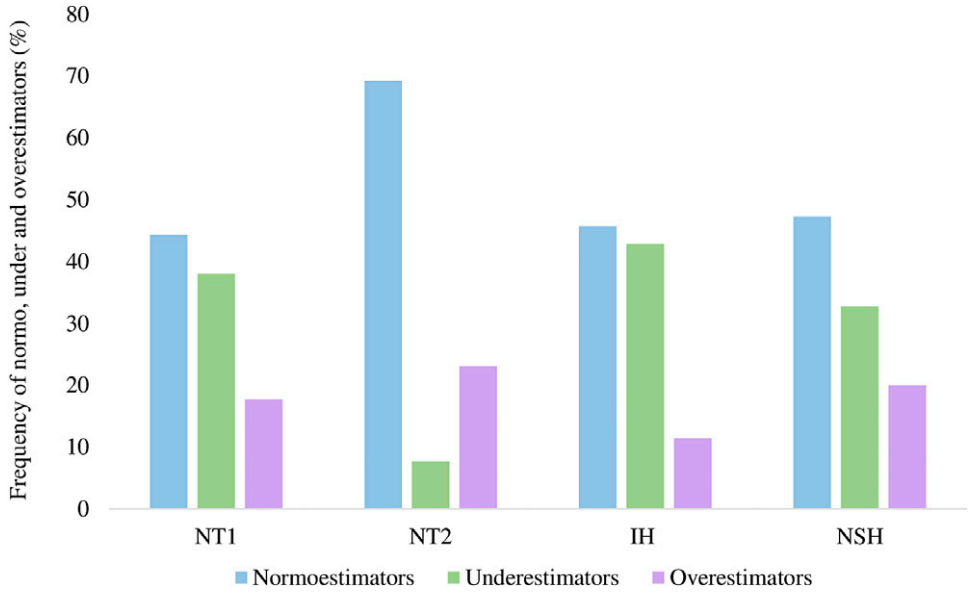


The y-axis represents the TST misperception index (%) for each CDH subgroup (on the x-axis). A TST misperception index of 100% indicates a perfect self-reported estimation of the TST with respect to the TST obtained with the objective measures. Values below 100% indicate an underestimation, and values above 100% represent an overestimation. The dotted line above 100 represents the cut-off above which participants are considered overestimators (110.43%); conversely, the dotted line below 100 indicates the cut-off below which patients are considered underestimators (88.31%) based on a study that provided normative values.[16] CDH = central disorders of hypersomnolence, IH = idiopathic hypersomnia, NSH = nonspecified hypersomnia, NT1 = narcolepsy type 1, NT2 = narcolepsy type 2, TST = total sleep time.

DISCUSSION

This study provides a detailed comparison of TST perception between adults with CDH and healthy controls. In the Canadian cohort we observed a significant group effect on the TST misperception index. Post hoc analyses revealed that all CDH subgroups (NT1, NT2, IH, NSH) significantly underestimated their TST compared to healthy controls. However, no significant differences emerged among the CDH subgroups themselves. Of note, the NT1 group had a mean SSMi of 86.83 ± 22.97%, which falls below the Lecci et al cut-off of 88.31%, indicating that NT1 participants can be classified as underestimators according to this criterion. In the French cohort, there was no healthy control group, and, similar to the Canadian sample, no statistically significant differences were observed between the CDH subgroups. Qualitatively, the IH group had a mean SSMi of 87.90 ± 22.85%, also below the Lecci et al cut-off, suggesting that IH participants can be considered underestimators according to this classification. Although less frequent, overestimation was also observed, particularly among NT2 participants in the replication cohort. Unlike healthy controls, no correlation was found between sleep fragmentation and the

Figure 5—Proportion of normoestimators, underestimators, and overestimators in patients with CDH.



The x-axis displays the classification of estimation types, normoestimators, underestimators, and overestimators, within each participant subgroup: NT1, NT2, IH, and NSH. The y-axis represents the proportion of participants in each category, expressed as a percentage. CDH = central disorders of hypersomnolence, IH = idiopathic hypersomnia, NSH = nonspecified hypersomnia, NT1 = narcolepsy type 1, NT2 = narcolepsy type 2.

misperception index in CDH groups. These findings underscore the clinical relevance of sleep misperception in hypersomnia related disorders, suggesting the need for a more nuanced diagnostic and therapeutic approach to CDH. Moreover, they indicate that different mechanisms may underlie sleep misperception in insomnia and CDH, warranting further investigation.

TST underestimation in CDH

Previous studies focused on a single sleep disorder or lacked control groups.[21–23] The present study represents a step forward by including a group of healthy sleepers, providing a more comprehensive understanding of TST misperception. The inclusion of this control group highlights that while TST underestimation is prevalent among individuals with EDS, healthy individuals tend to accurately estimate or slightly overestimate their TST.[36,37] This observation raises important questions about how self-reported sleep perception is shaped. We can question whether healthy individuals may benefit from more adaptive cognitive-emotional processes, such as lower preoccupation with sleep, more stable mood regulation, or reduced attentional focus on bodily sensations, which could support a more accurate or even optimistic estimation of sleep.[6,12] These mechanisms could contribute to their greater alignment between perceived and objective sleep, despite shorter actual sleep duration compared to patients with CDH, but this hypothesis needs to be tested.

A stringent cutoff, an absolute TST ratio under 88.3% in 1,252 participants,[16] was employed to classify normo, under, and overestimators across subgroups. The robustness of the findings is further strengthened by the large sample size, the inclusion of 4 distinct CDH subgroups.

The highest frequency of TST underestimation was observed in the NT1 subgroup in the Canadian cohort, where over 40% of patients reported sleeping less than objectively measured. This aligns with the cortical hyperarousal hypothesis,[6] which posits that individuals with more fragmented sleep are prone to sleep misperception. NT1 specific symptoms, including REM sleep fragmentation, lucid dreaming, disrupted nighttime sleep, sleep-related hallucinations, and inappropriate wake-sleep transitions due to hypocretin deficiency,[38–41] may contribute to this underestimation.

In the French cohort, patients with IH were classified as underestimators based on their mean SSMi. This finding is particularly notable, as IH is characterized by consolidated sleep,[42] which challenges predictions derived from the cortical hyperarousal hypothesis. This discrepancy suggests that alternative mechanisms may contribute to TST underestimation in IH. One possibility is that distinct IH phenotypes, such as individuals reporting unrefreshing naps, prolonged TST, and marked sleep inertia,[43–46] may exhibit dissociation between sleep and wake states. Recent evidence from extended bedrest protocols indicates that sleep misperception in hypersomnia is not limited to underestimation: in IH without long sleep time and in NSH, overestimation of TST can be frequent and severe, particularly during daytime periods, and is associated with greater daytime sleepiness and a higher number of sleep bouts.[34] This apparent discrepancy with the present findings may reflect both methodological differences (single-night PSG with circadian cues vs prolonged bedrest without temporal cues) and clinical heterogeneity between subtypes. From a clinical perspective, these contrasting patterns suggest that TST misperception in hypersomnolence is not a uniform phenomenon: patients with consolidated long sleep may be prone to underestimation, whereas

those with more fragmented sleep or shorter TSTs may tend toward overestimation. Recognizing these distinct profiles could have implications for tailoring both diagnostic assessments and patient counseling. Future studies employing spectral power analysis or high-density EEG could explore whether TST misperception in IH varies according to sleep duration (short vs long TST) or the presence of sleep drunkenness and nonrestorative napping. Alternatively, patients with IH may display a unique mechanism of sleep misperception, distinct from the cortical hyperarousal model proposed in insomnia. One possible contributor to TST misperception in IH could be the occurrence of wake (or local wake) episodes during sleep.[47]

Can PSG findings explain SSM?

Once we established that patients with CDH underestimate TST compared to healthy controls, we aimed to understand the nature of these discrepancies. While the percentage of N1 and the number of awakenings were negatively associated to sleep perception in healthy controls, no association between SSMi and sleep fragmentation variables was observed in the CDH groups. This aligns with insomnia research, which suggests that reliable PSG correlates of SSM typically emerge only in individuals with severe sleep misperception (TST discrepancy ≥ 2 hours) compared to healthy sleepers.[16,48] However, most studies have failed to identify consistent PSG macrostructural differences between patients with insomnia with TST misperception and healthy controls.[7–9,12] This suggests that conventional sleep staging may lack the sensitivity to detect underlying mechanisms, such as hyperarousal. Consequently, these findings underscore the need for advanced EEG techniques in hypersomnia research to better explore the role of hyperarousal, microsleep or other contributing factors in sleep misperception.

Misperception or inherent limits of the PSG?

The discrepancy between self-reported sleep perception and objective measurements may stem from both genuine perceptual differences and the limitations of current sleep recording techniques.[12] While PSG is the gold standard for sleep assessment, its reliance on limited electrodes and long averaging periods may overlook regional brain activity variations, challenging the prevailing view that sleep consists of discrete, easily identifiable states with distinct neurophysiological signatures.[49,50] Evidence suggests that wake-like activity can occur during sleep, possibly linked to noradrenergic activation, which may not always be detectable via standard scalp EEG.[47,51] Brief shifts toward faster brain activity may allow partial awareness of the environment, balancing rest with responsiveness.[52] Such intrusions of fast activity have been associated with poor sleep quality and SSM, particularly in insomnia.[7] This raises the possibility that SSM may be a "mismeasurement" of standard PSG recordings rather than a true perceptual distortion.[12] Future studies utilizing high-density EEG could be crucial in uncovering the subtle brain dynamics that underlie these experiences. Interestingly, increased fast activity has been linked to the sensation of being awake even in healthy individuals, suggesting that this phenomenon is not exclusive to sleep disorders.[53] A multidimensional approach integrating sleep processes and states could improve our understanding of sleep perception and its neural mechanisms across various populations, including those with insomnia and hypersomnia.

Clinical implications

These findings have important clinical implications, particularly for IH. While self-reported TST is not considered a formal diagnostic criterion, it may provide valuable supporting evidence, especially in cases where objective confirmation is unavailable or inconclusive. Given this, integrating objective sleep measures, such as 7-day actigraphy or an extended 32-hour ad libitum-PSG protocol, is recommended to complement patient self-reports.[54–56] Additionally, investigating sleep misperception during ad libitum PSG could provide further insights into its direction and significance. However, it is important to note that patients' perception of their sleep is a valuable source of information, offering critical insights into how sleep disorders impact their daily functioning and quality of life. A comprehensive understanding of the mechanisms underlying sleep misperception could enhance both diagnosis and treatment strategies for CDH. In patients with insomnia, for example, behavioral sleep education targeting the mismatch between PSG data and self-reported reports has been shown to improve the accuracy of TST perception.[57] However, a key distinction between insomnia and hypersomnolence lies in how sleep is perceived: while patients with insomnia typically report insufficient sleep and tend to underestimate sleep duration, making self-reported recalibration a relevant therapeutic target, patients with hypersomnolence may also underestimate their sleep, but the clinical relevance of this misperception is reduced when excessive sleep duration is the primary complaint. Instead, this observation has diagnostic implications, suggesting that reported prolonged sleep durations in hypersomnolence may be lower than actual recorded values, necessitating adjustments to diagnostic thresholds.

Limits and future prospective

The study's reliance on a single night of in-laboratory PSG limits the generalizability of findings to broader sleep patterns. Future research should incorporate multinight assessments or at-home monitoring to improve ecological validity. Assessing test-retest consistency over time could provide insights into the stability of findings and the impact of the first-night effect. Additionally, IH participants followed a clinical protocol that restricted ad libitum sleep, potentially leading to an underestimation of TST due to imposed schedules and next-day MSLT requirements. Access to time cues and windows in the sleep environment may have also influenced TST perception. Another limitation is the potential for rounding bias in self-reported TST, as patients typically provided their estimates in half-hour intervals, rather than in exact minutes. Although participants were asked to estimate their sleep duration after the PSG night using a standardized morning questionnaire, they were not instructed to report with minute-level precision, which may have introduced some variability in the misperception index. Moreover, another limitation is the variation in diagnostic work-up procedures between sites, particularly regarding the use of cerebrospinal fluid orexin measurement and extended

PSG protocols. In France, cerebrospinal fluid orexin testing and 32-hour bedrest recordings are part of routine assessment in national reference centers, whereas in Canada, orexin testing is rarely performed and long-duration PSG is not routinely available. Although both cohorts followed *International Classification of Sleep Disorders*, third edition, text revision criteria, these differences may have resulted in a more heterogeneous diagnostic composition, especially within the IH group, which may include individuals with and without objectively confirmed long sleep duration. This variability may represent an important direction for future studies aiming to differentiate phenotypes within CDH populations. Although including 2 cohorts enhanced generalizability, it also introduced complexity that probably reflect the real-world clinical situation. Only the Montreal cohort included healthy controls, limiting direct CDH–control comparisons in Montpellier. While nonsignificant subgroup differences were replicated, cohort-specific variability emerged, particularly in NT2 underestimator rates (10% vs ∼25%), likely influenced by small NT2 sample size, and sampling variation. This variability calls for cautious interpretation, reinforces the need to avoid overgeneralization, and warrants further investigation into potential moderators of self-reported-objective sleep discrepancy. Replicating the study across different centers would help address potential sampling biases and identify possible endophenotypic variations. Furthermore, additional factors such as medication status (drug-naive vs withdrawal), nightmares, first-night effect, and the perception of sleep as nonrestorative should be explored to refine the understanding of sleep misperception mechanisms.

CONCLUSIONS

In conclusion, our findings provide compelling evidence that TST underestimation is a widespread phenomenon across the CDH subgroups, with significant implications for clinical practice. Underestimation is consistent across all CDH subgroups as compared to healthy controls. In the Canadian cohort, all CDH subgroups significantly underestimated TST relative to controls, and NT1 had the highest proportion of underestimators. In the French cohort, no significant subgroup differences were observed, but the IH group had the highest proportion of underestimators. Both groups (NT1 and IH) showed SSMi below the Lecci et al[16] cut-off classifying them as underestimators. These findings suggest that NT1 and IH may be more prone to underestimation, but conclusions should be interpreted cautiously as subgroup differences were not statistically significant in either cohort. These findings highlight the need for clinicians to consider the potential impact of sleep misperception when diagnosing and treating patients with CDH.

ABBREVIATIONS

CDH, central disorders of hypersomnolence
EDS, excessive daytime sleepiness
EEG, electroencephalogram
IH, idiopathic hypersomnia
MSLT, Multiple Sleep Latency Test
NSH, nonspecified hypersomnia
NT1, narcolepsy type 1
NT2, narcolepsy type 2
PSG, polysomnography
REM, rapid eye movement
SSM, sleep state misperception
SSMi, Sleep State Misperception Index
TST, total sleep time

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The data underlying this article will be shared on reasonable request to the corresponding author.

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