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DUET STUDY 407

Improvements in Sleep Inertia With Low-Sodium Oxybate Treatment in Participants With Idiopathic Hypersomnia in the DUET Study

Deborah A. Nichols, MS[1]; Logan D. Schneider, MD[2]; Chad M. Ruoff, MD[3]; Jing Dai, PhD[1]; Teresa L. Steininger, PhD[1]; Marisa Whalen, PharmD[4]; Alyssa Cairns, PhD[4]; David T. Plante, MD, PhD[5]; Hans P. A. Van Dongen, PhD[6]

[1]Jazz Pharmaceuticals, Palo Alto, CA, USA; [2]Stanford Sleep Medicine Center, Redwood City, CA, USA; [3]Mayo Clinic, Scottsdale, AZ, USA; [4]Jazz Pharmaceuticals, Philadelphia, PA, USA; [5]Department of Psychiatry, University of Wisconsin-Madison, Madison, WI, USA; [6]Sleep and Performance Research Center, Washington State University Health Sciences Spokane, Spokane, WA, USA

Objective

- To evaluate effectiveness of low-sodium oxybate (LXB) on changes in objective and subjective sleep inertia in DUET participants with idiopathic hypersomnia, stratified by long sleep need

Conclusions

- At baseline (BL), participants with idiopathic hypersomnia exhibited subjective and objective sleep inertia, with prolonged dissipation after awakening
  - At BL, objective sleep inertia was particularly severe in participants with long sleep need (>9 hours)
- After LXB treatment, participants with idiopathic hypersomnia showed substantial improvements in objective and subjective sleep inertia magnitude, which occurred in the overall idiopathic hypersomnia cohort and in both subcohorts stratified by long sleep need
- After LXB treatment, most participants in both subcohorts also reported reduced subjective sleep inertia severity on the Patient Global Impression of Severity

References: 1. Tassi P, Muzet A. *Sleep Med Rev.* 2000;4(4):341-353. **2.** Lundholm KR, et al. *Clocks Sleep.* 2021;3(2):298-311. **3.** Trotti LM. *Sleep Med Rev.* 2017;35:76-84. **4.** American Academy of Sleep Medicine. *International Classification of Sleep Disorders – Third Edition, Text Revision*. Darien, IL: American Academy of Sleep Medicine; 2023. **5.** Kim JR, et al. *PLoS One.* 2026;21(1):e0337992. **6.** Hilditch CJ & McHill AW. *Nat Sci Sleep.* 2019:155-165. **7.** Roth B. *Int J Neurol.* 1981;15(1-2):108-118. **8.** Dauvilliers Y, et al. *Neurology.* 2019;92(15):e1754-e1762. **9.** Trotti LM. *Continuum (Minneapolis, Minn).* 2020;26(4):890-907. **10.** Alexander JK, et al. Presented at: Annual Meeting of the Associated Professional Sleep Societies; June 8-11, 2025; Seattle, WA. **11.** Xywav® (calcium, magnesium, potassium, and sodium oxybates) oral solution, CIII [prescribing information]. Palo Alto, CA: Jazz Pharmaceuticals, Inc. **12.** Nichols DA, et al. *Neurol Ther.* 2025;14(4):1705-1727. **13.** Plante DT, et al. *CNS Drugs.* 2026;Mar 14 Online Ahead of Print. **14.** Trotti LM, et al. *J Clin Sleep Med.* 2022;18:1395-1403.

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Presented at SLEEP 2026, the 40th Annual Meeting of the Associated Professional Sleep Societies, LLC (APSS); June 14–17, 2026; Baltimore, MD, USA

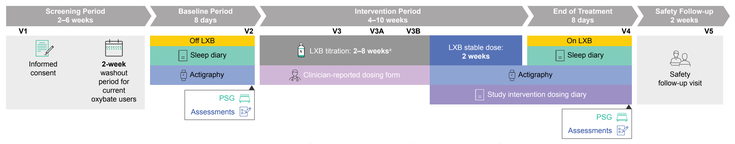
Introduction

- People with idiopathic hypersomnia often experience sleep inertia, which is characterized by profound difficulty waking up, with grogginess, disorientation, and cognitive impairment immediately after awakening[1-4]
- In the general population, sleep inertia may last 15 to 30 minutes[5,6]; in people with idiopathic hypersomnia, it can last several hours[7]
  - As many as 78% to 90% of individuals with idiopathic hypersomnia report sleep inertia as a core symptom[8-10]
- Low-sodium oxybate (LXB; Xywav®) is approved for the treatment of cataplexy or excessive daytime sleepiness in patients aged 7 years of age and older with narcolepsy and for the treatment of idiopathic hypersomnia in adults[11]
- Prior to DUET, sleep inertia magnitude had not been evaluated in people with idiopathic hypersomnia taking LXB

Methods

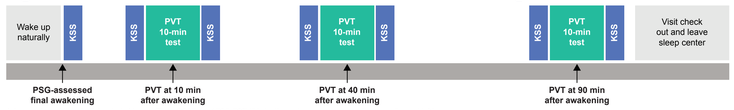
Figure 1. Study Design

DUET (**D**evelop hypersomnia **U**nderstanding by **E**valuating low-sodium oxybate **T**reatment; NCT05875974) was a phase 4, prospective, single-arm, open-label study



Adapted from Nichols DA, et al. http://creativecommons.org/licenses/by-nc/4.0/.[12] Primary results from DUET have been reported.[13]
[a]Weekly titration visits were by teleconference. Visit 3 occurred on titration day 14. Titration could take between 2 and 8 weeks. Additional in-clinic visits were scheduled for day 35 (visit 3A) and day 56 (visit 3B), as needed. Investigator could optimize participant dosage and move to SDP at visit 3, 3A, or 3B, but not during intervening weekly teleconferences.
LXB, low-sodium oxybate; PSG, polysomnography; SDP, stable-dose period; V, visit.

Figure 2. Schedule of PVT and KSS Assessments on the Morning After PSG at BL and EOT



Adapted from Nichols DA, et al. *Neurol Ther.* 2025;14:1705-727.[13] http://creativecommons.org/licenses/by-nc/4.0/.
BL, baseline; EOT, end of treatment; KSS, Karolinska Sleepiness Scale; min, minute; PSG, polysomnography; PVT, Psychomotor Vigilance Test.

- This exploratory analysis evaluated data from participants stratified by patient-reported ideal nighttime sleep duration (with [>9 hours] and without [≤9 hours] long sleep need [LSN]), based on their responses to Idiopathic Hypersomnia Severity Scale (IHSS) item 1 at BL (Response options: "≥11 hours," ">9 and <11 hours," "7 to 9 hours," or "<7 hours")[8]
- Sleep inertia outcomes included the Psychomotor Vigilance Test (PVT; objective sustained attention as measured by number of PVT lapses) and Karolinska Sleepiness Scale (KSS; subjective sleepiness as measured by KSS ratings); both were administered multiple times (**Figure 2**) at BL (Visit 2) and end of treatment (EOT; Visit 4), in the morning after overnight polysomnography
- Nonlinear, mixed-effects regression models were applied to PVT lapses (reaction time >500 ms) and KSS ratings for the overall idiopathic hypersomnia cohort, and separately for the LSN subcohorts analysis, to estimate objective and subjective sleep inertia, respectively, over the course of time since awakening[2,14]
- These statistical models also evaluated the main characteristics of sleep inertia, including the sleep inertia magnitude and the dissipation rate[2]
  - Sleep inertia magnitude was calculated by subtracting the model-derived asymptote (the point at which the sleep inertia dissipation reached a plateau) from the estimated sleep inertia immediately upon awakening
  - The sleep inertia dissipation time constant is defined as a standard measure of exponential dissipation that indicates how long it took for sleep inertia to reduce by 63.2%.[2] It was calculated for each model separately and was generated from the data at both BL and EOT timepoints combined
- The Patient Global Impression of Severity (PGI-S) was administered and analyzed descriptively at BL and EOT to assess subjective sleep inertia severity
- Treatment-emergent adverse events (TEAEs) were assessed in the subcohorts with or without LSN (reported in the Supplemental Information, accessed via the QR code)
- For additional Methods details, see the Supplemental Information, accessed via the QR code

Results

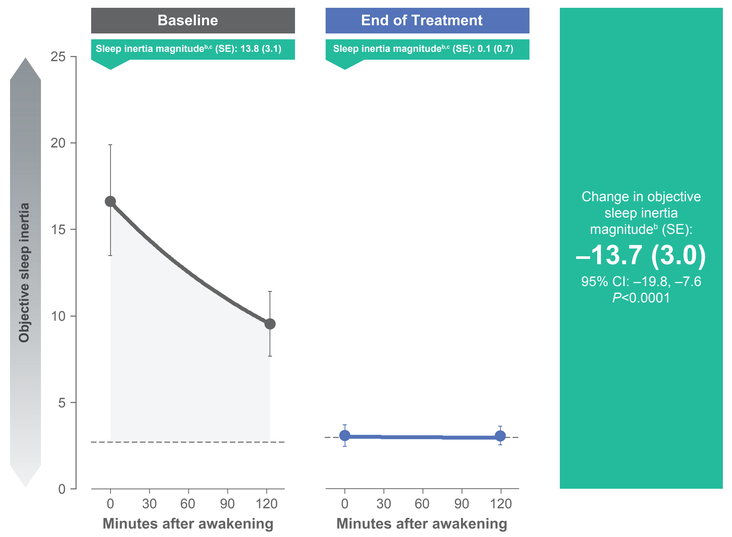
Table 1. Demographics and Baseline Characteristics[a]

Characteristic	With LSN (>9 h) n=20[b]	Without LSN (≤9 h) n=19[b]	Total Idiopathic Hypersomnia Cohort n=40
Age (years) Mean (SD)	37.7 (11.6)	38.7 (12.8)	38.7 (12.3)
Female, n (%)	20 (100)	11 (57.9)	31 (77.5)
Concomitant alerting agents,[c] n (%)	5 (25.0)	9 (47.4)	15 (37.5)

[a]Completer analysis set. [b]Of 40 enrolled participants with idiopathic hypersomnia, 39 had BL IHSS data (20 reported LSN [>9 hours] and 19 reported no LSN [≤9 hours]). [c]Alerting agents were defined as stimulants or wake-promoting agents; participants must have taken the same dosage for ≥1 month before screening visit 1 with no plan to adjust dosage during the study.
BL, baseline; IHSS, Idiopathic Hypersomnia Severity Scale; LSN, long sleep need; max, maximum; min, minimum; SD, standard deviation.

- Twenty of 24 participants with LSN and 19/21 participants without LSN completed the study

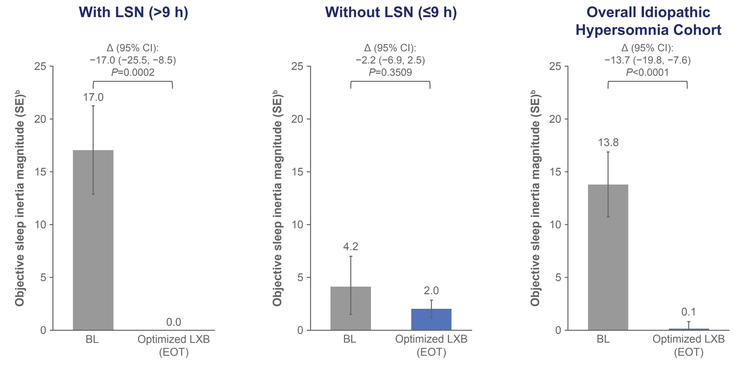
Figure 3. Objective Sleep Inertia (Based on PVT Lapses) Reduced With Optimized LXB Treatment After Awakening, in the Overall Idiopathic Hypersomnia Cohort[a]



[a]Completer analysis set. [b]At 0 minutes after awakening. The dotted line indicates model-derived asymptote to where the sleep inertia dissipates over time since awakening. Sleep inertia magnitude was calculated by subtracting the asymptote from the sleep inertia immediately upon awakening. [c]The asymptote for the total idiopathic hypersomnia cohort was 2.7 at BL and 3.0 at EOT.
CI, confidence interval; LSN, long sleep need; LXB, low-sodium oxybate; PVT, Psychomotor Vigilance Test; SE, standard error.

- This curve displays the nonlinear, mixed-effects regression model using PVT lapses from which the estimated objective sleep inertia magnitude (**Figure 4**) was derived
- The full set of curves for PVT and KSS, including by subcohorts with or without LSN, are displayed in the Supplement, accessed via the QR code

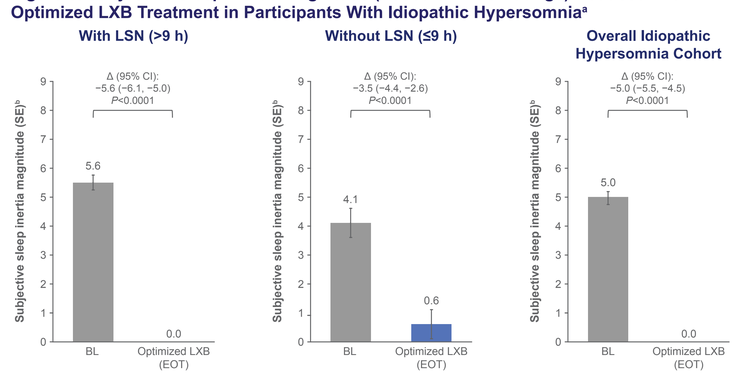
Figure 4. Objective Sleep Inertia Magnitude (Based on PVT Lapses) Reduced With Optimized LXB Treatment in Participants With Idiopathic Hypersomnia[a]



[a]Completer analysis set; 20/24 participants with LSN and 19/21 participants without LSN completed the study. [b]Objective sleep inertia was estimated from PVT lapses.
BL, baseline; CI, confidence interval; EOT, end of treatment; h, hour; LSN, long sleep need; LXB, low-sodium oxybate; PVT, Psychomotor Vigilance Test.

- Participants with idiopathic hypersomnia exhibited objective sleep inertia upon awakening at BL, which was especially pronounced in participants with LSN
- Participants in both subcohorts showed reductions in objective sleep inertia, while participants with LSN also showed reductions in objective sleep inertia magnitude with LXB treatment
- The objective sleep inertia dissipation time constant (SE) was estimated to be 2.9 (1.2) hours for the overall idiopathic hypersomnia cohort model
- For additional figures, see the Supplemental Information, accessed via the QR code

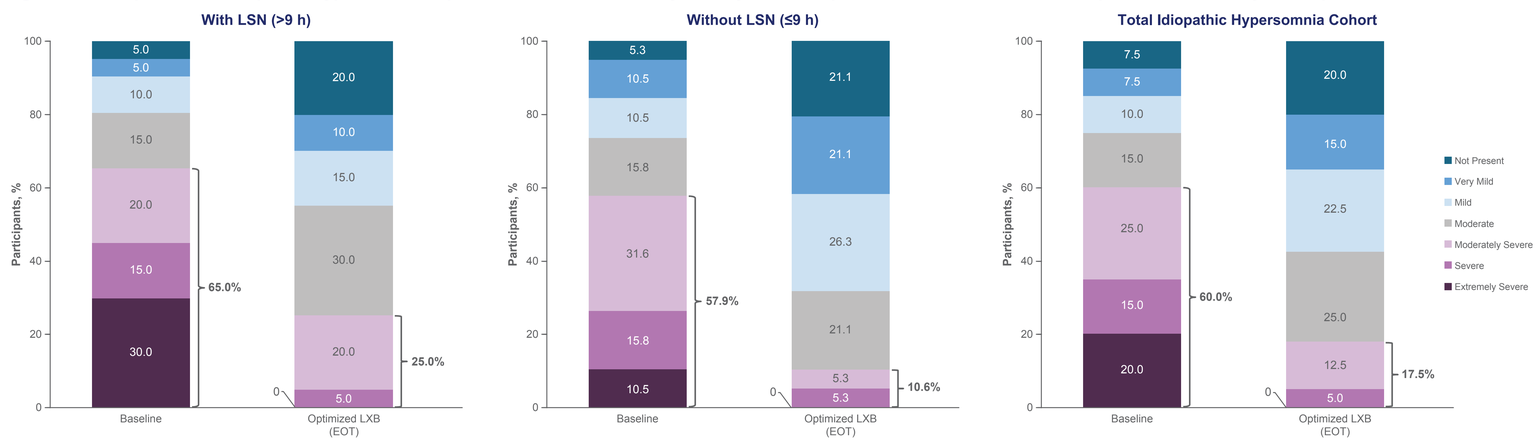
Figure 5. Subjective Sleep Inertia Magnitude (Based on KSS Ratings) Reduced With Optimized LXB Treatment in Participants With Idiopathic Hypersomnia[a]



[a]Completer analysis set; 20/24 participants with LSN and 19/21 participants without LSN completed the study. [b]Subjective sleep inertia was estimated from KSS ratings.
BL, baseline; CI, confidence interval; EOT, end of treatment; h, hour; KSS, Karolinska Sleepiness Scale; LSN, long sleep need; LXB, low-sodium oxybate.

- Participants in both subcohorts reported profound subjective sleep inertia upon awakening at BL, which improved with LXB treatment
- The subjective sleep inertia dissipation time constant (SE) was estimated to be 12.4 (3.9) hours for the overall idiopathic hypersomnia cohort model
- For additional figures, see the Supplemental Information, accessed via the QR code

Figure 6. Participants With Idiopathic Hypersomnia Reported Reduction in Severity of Subjective Sleep Inertia on the Patient Global Impression of Severity With Optimized LXB Treatment



EOT, end of treatment; h, hours; LSN, long sleep need; LXB, low-sodium oxybate.

- At BL, 60.0% of participants in the total idiopathic hypersomnia cohort reported at least moderately severe sleep inertia, which decreased to 17.5% at EOT (*P*=0.0002; With LSN, *P*=0.0209; Without LSN, *P*=0.0027)

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References: 1. Dinges DF, Powell JW. *Behav Res Methods Instrum Comput.* 1985. 17(6):652–655. **2.** Van Dongen HP, et al. *Sleep.* 2001; 24(7):813-819. **3.** Shahid A, et al. *STOP, THAT and One Hundred Other Sleep Scales.* New York, NY: Springer New York; 2012:209-210. **4.** Lundholm KR, et al. *Clocks Sleep.* 2021;3(2):298-311.

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Supplemental Material

Supplemental Figure S1. Inclusion/Exclusion Criteria

Inclusion Criteria

- Adults 18–75 with a primary diagnosis of idiopathic hypersomnia (per ICSD-3 criteria)
- ESS score >10[a]
- Concomitant stable use of alerting agents allowed[b]

Exclusion Criteria

- Untreated/inadequately treated sleep-disordered breathing (AHI >10)[c]
- History/presence of other untreated, inadequately treated, or unstable disorder or condition (eg, sleep, medical, behavioral/psychiatric, neurologic[c])
- Medication(s) contraindicated, with known drug-drug interaction, or similar EEG effects as LXB or known to have clinically significant CNS sedating effects

[a]At screening visit 1 or, if taking an oxybate medication at screening, at visit 2. [b]Alerting agents were defined as stimulants or wake-promoting agents; participants must have taken the same dosage for ≥1 month before screening visit 1 with no plan to adjust dosage during the study. [c]Hypopnea definition included a ≥4% desaturation as per Rule 1B of *The AASM Manual for the Scoring of Sleep and Associated Events*, as assessed during the baseline PSG visit. [d]Disorder or condition that might affect the participant's safety and/or interfere with the conduct of the study, in the opinion of the investigator.
AASM, American Academy of Sleep Medicine; AHI, apnea-hypopnea index; CNS, central nervous system; EEG, electroencephalogram; ESS, Epworth Sleepiness Scale; ICSD-3, *International Classification of Sleep Disorders – Third Edition*; LXB, low-sodium oxybate; PSG, polysomnography.

Analyses Sets

- The safety analysis set includes all participants who enrolled in the study and took their prescribed low-sodium oxybate (LXB; Xywav®) regimen for ≥1 night after the baseline (BL) period (N=46; with long sleep need [LSN], n=24; without LSN, n=21; no baseline IHSS to derive LSN subcohort, n=1; evaluated for demographics/BL characteristics, dosing, treatment-emergent adverse events [TEAEs])
- The completer analysis set includes all participants who enrolled in the study, took their prescribed LXB regimen for ≥1 night after the BL period, completed the stable-dose period, and completed the polysomnography end-of-treatment (EOT) visit (n=40; with LSN, n=20; without LSN, n=19; no baseline IHSS to derive LSN subcohort, n=1; evaluated for Patient Global Impression of Severity [PGI-S], Psychomotor Vigilance Test [PVT], and Karolinska Sleepiness Scale [KSS] nonlinear modeling analyses)

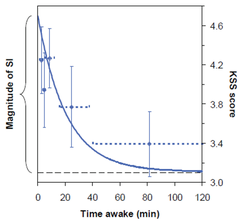
Outcomes

- The 10-minute PVT, a reaction-time test, was completed at 10, 40, and 90 minutes after awakening, and required participants to respond as quickly and accurately as possible to visual stimulus[1,2]
  - The PVT evaluates the ability to sustain attention and measures behavioral alertness; the key outcome was lapses of attention (reaction times >500 ms)
- The KSS, a single-item, self-reported, 9-point categorical scale, was completed upon awakening (within 2 minutes) and immediately before and after each PVT assessment
  - The KSS measures situational sleepiness in the present moment using a 9-point scale (1="extremely alert" to 9="very sleepy, great effort to keep awake, fighting sleep")[3]

Statistical Analyses

- The models accounted for systematic interindividual differences and heteroskedastic error variance; *P* values are nominal
- The narcolepsy study cohort was analyzed separately; results are reported in **Poster 339**
- Post hoc *P* values for comparisons of proportions of participants at BL versus EOT reporting "moderately severe/severe/extremely severe" on the PGI-S assessments were obtained from McNemar-Bowker test

Supplemental Figure S2. Example of Nonlinear Mixed-Effects Regression Model With KSS Administered as a Function of Time Since PSG-Assessed Final Awakening



- Curve displays the group model for sleepiness (KSS rating)
- Magnitude of sleep inertia was calculated by subtracting the model-derived asymptote from the estimated sleep inertia immediately upon awakening
- Sleep inertia dissipation time constant: a standard measure of exponential decay that indicates how long it took for sleep inertia to reduce by 63.2%

Figure re-used without modification from Lundholm K, et al. *Clocks Sleep.* 2021.3:298–311, under Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/).[4]
KSS, Karolinska Sleepiness Scale; min, minutes; SI, sleep inertia.

Supplemental Table S1. Summary of TEAEs[a]

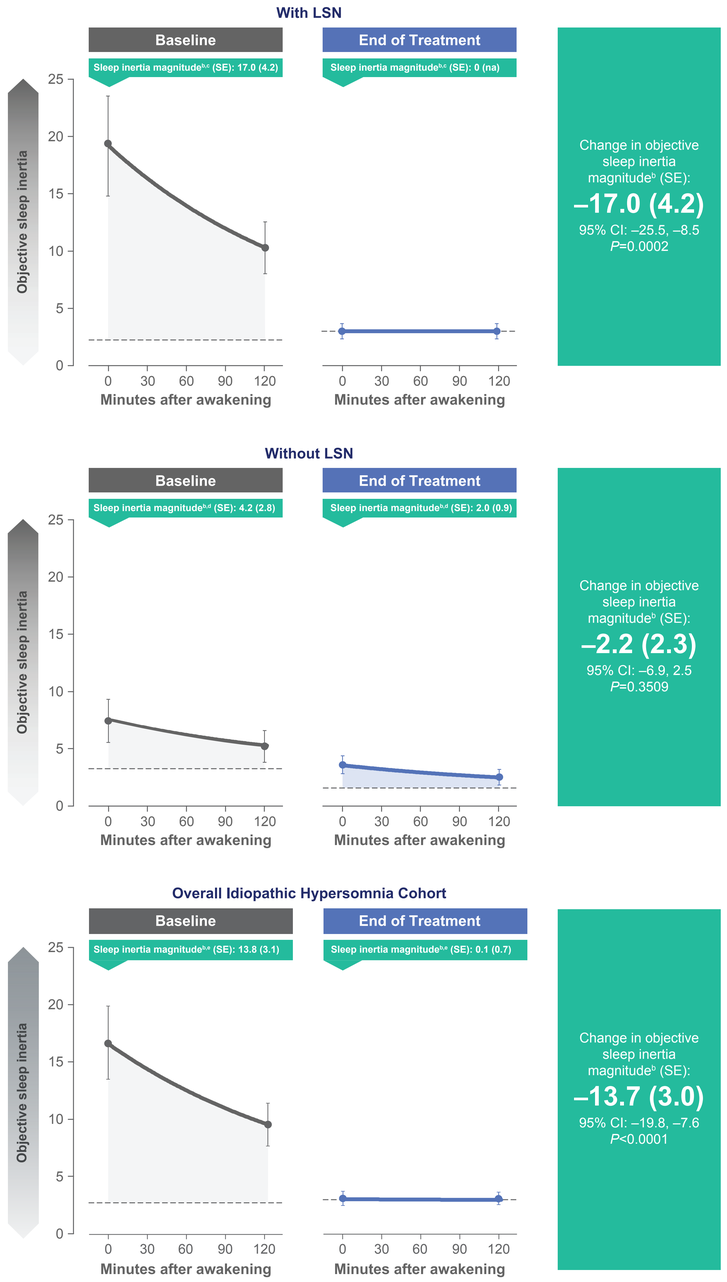
Participants, n (%)	With LSN (>9 h) n=24[b]	Without LSN (≤9 h) n=21[b]	Total Idiopathic Hypersomnia Cohort N=46
With ≥1 TEAE	19 (79.2)	14 (66.7)	34 (73.9)
With ≥1 serious TEAE	0	1 (4.8)	1 (2.2)
With ≥1 TEAE related to treatment	19 (79.2)	11 (52.4)	30 (65.2)
With ≥1 TEAE leading to discontinuation	1 (4.2)	0	1 (2.2)
TEAEs occurring in ≥5% of participants			
Nausea	7 (29.2)	2 (9.5)	9 (19.6)
Dizziness	6 (25.0)	2 (9.5)	8 (17.4)
Headache	4 (16.7)	4 (19.0)	8 (17.4)
Vomiting	4 (16.7)	0	5 (10.9)
Middle insomnia	1 (4.2)	3 (14.3)	4 (8.7)
Anxiety	2 (8.3)	1 (4.8)	3 (6.5)
Decreased appetite	1 (4.2)	2 (9.5)	3 (6.5)
Enuresis	2 (8.3)	1 (4.8)	3 (6.5)
Somnolence	2 (8.3)	1 (4.8)	3 (6.5)
Bruxism	0	2 (9.5)	2 (4.3)
Cough	0	2 (9.5)	2 (4.3)
Hyperhidrosis	2 (8.3)	0	2 (4.3)
Nasal congestion	0	2 (9.5)	2 (4.3)

[a]Safety analysis set includes all participants who enrolled in the study and took ≥1 dose of the study drug after the BL period. [b]Of 46 enrolled participants with idiopathic hypersomnia, 45 had BL IHSS data (24 reported LSN [>9 hours] and 21 reported no LSN [≤9 hours]).
BL, baseline; h, hours; LSN, long sleep need; TEAE, treatment-emergent adverse event.

Lowest Highest Lowest Highest Lowest Highest

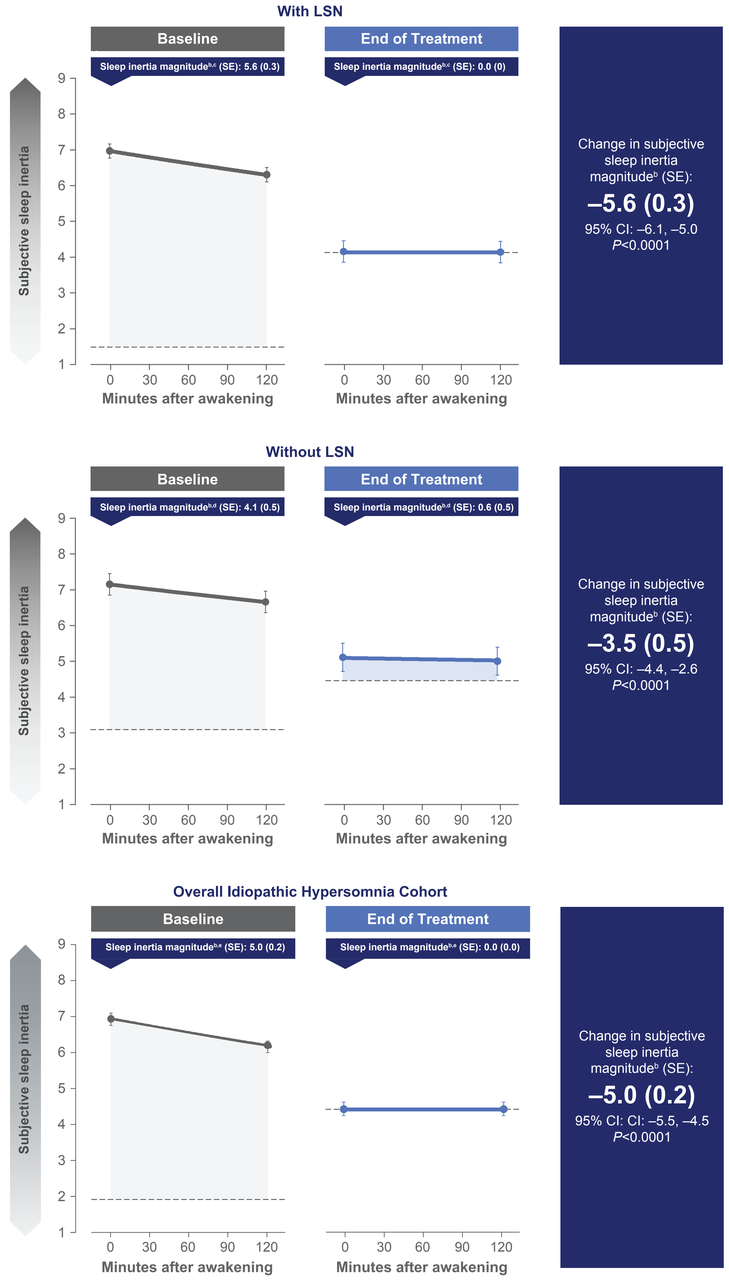
- Overall, 79.2% and 66.7% of participants with or without LSN, respectively, reported ≥1 TEAE
- Most TEAEs were mild (45.8%, 42.9%) or moderate (33.3%, 23.8%) in severity for participants with or without LSN, respectively

Supplemental Figure S3. Objective Sleep Inertia (Based on PVT Lapses) Reduced With Optimized LXB Treatment in Participants With Idiopathic Hypersomnia[a] After Awakening



[a]Completer analysis set; 20/24 participants with LSN and 19/21 participants without LSN completed the study. [b]At 0 minutes after awakening. The dotted line indicates model-derived asymptote to where the sleep inertia dissipates over time since awakening. Sleep inertia magnitude was calculated by subtracting the asymptote from the sleep inertia immediately upon awakening. [c]The asymptote for participants with LSN was 2.2 at BL and 3.0 at EOT. [d]The asymptote for participants without LSN was 3.3 at BL and 1.6 at EOT. [e]The asymptote for the total idiopathic hypersomnia cohort was 2.7 at BL and 3.0 at EOT.
CI, confidence interval; LSN, long sleep need; LXB, low-sodium oxybate; PVT, Psychomotor Vigilance Test; SE, standard error.

Supplemental Figure S4. Subjective Sleep Inertia (Based on KSS Ratings) Reduced With Optimized LXB Treatment in Participants With Idiopathic Hypersomnia[a] After Awakening



[a]Completer analysis set; 20/24 participants with LSN and 19/21 participants without LSN completed the study. [b]At 0 minutes after awakening. The dotted line indicates model-derived asymptote to where the sleep inertia dissipates over time since awakening. Sleep inertia magnitude was calculated by subtracting the asymptote from the sleep inertia immediately upon awakening. [c]The asymptote for participants with LSN was 1.4 at BL and 4.1 at EOT. [d]The asymptote for participants without LSN was 3.1 at BL and 4.5 at EOT. [e]The asymptote for the total idiopathic hypersomnia cohort was 1.9 at BL and 4.4 at EOT.
CI, confidence interval; long sleep need; KSS, Karolinska Sleepiness Scale; LXB, low-sodium oxybate; SE, standard error.

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Supplemental Material

Supplemental Table S2. Demographics and Baseline Characteristics[a]

Characteristic	With LSN (>9 h) n=24[b]	Without LSN (≤9 h) n=21[b]	Total Idiopathic Hypersomnia Cohort N=46
Age (years)			
Mean (SD)	36.8 (11.1)	38.6 (12.1)	38.1 (11.8)
Median (min, max)	33.5 (20, 62)	38.0 (22, 68)	37.5 (20, 68)
Sex at birth, n (%)			
Male	24 (100)	13 (61.9)	37 (80.4)
Female		8 (38.1)	9 (19.6)
Race, n (%)			
White	22 (91.7)	16 (76.2)	39 (84.8)
Black or African American	1 (4.2)	3 (14.3)	3 (6.5)
Asian			2 (4.3)
Native Hawaiian or other Pacific Islander		1 (4.8)	1 (2.2)
Multiple[c]	1 (4.2)	1 (4.8)	1 (2.2)
Ethnicity, n (%)			
Hispanic or Latino	8 (33.3)	2 (9.5)	10 (21.7)
Not Hispanic or Latino	16 (66.7)	18 (85.7)	35 (76.1)
Body mass index (kg/m^2), mean (SD)	28.3 (7.4)	28.6 (5.4)	28.5 (6.4)
Healthy weight 18.5 to <25			
Overweight 25 to <30			
Obese ≥30			
Concomitant alerting agents,[d] n (%)			
Yes	8 (33.3)	10 (47.6)	19 (41.3)
No	16 (66.7)	11 (52.4)	27 (58.7)

[a]Safety analysis set. [b]Of 46 enrolled participants with idiopathic hypersomnia, 45 had BL IHSS data (24 reported LSN [>9 hours] and 21 reported no LSN [≤9 hours]). [c]Participant reported more than 1 race. [d]Alerting agents were defined as stimulants or wake-promoting agents; participants must have taken the same dosage for ≥1 month before screening visit 1 with no plan to adjust dosage during the study.
BL, baseline; IHSS, Idiopathic Hypersomnia Severity Scale; LSN, long sleep need; max, maximum; min, minimum; SD, standard deviation.

Supplemental Table S3. Demographics and Baseline Characteristics[a]

Characteristic	With LSN (>9 h) n=20[b]	Without LSN (≤9 h) n=19[b]	Total Idiopathic Hypersomnia Cohort n=40
Age (years)			
Mean (SD)	37.7 (11.6)	38.7 (12.8)	38.7 (12.3)
Median (min, max)	34.0 (20, 62)	38.0 (22, 68)	38.0 (20, 68)
Sex at birth, n (%)			
Male	0	8 (42.1)	9 (22.5)
Female	20 (100)	11 (57.9)	31 (77.5)
Race, n (%)			
Asian	1 (5.0)	1 (5.3)	2 (5.0)
Black or African American	0	2 (10.5)	2 (5.0)
White	18 (90.0)	15 (78.9)	34 (85.0)
Native Hawaiian or Other Pacific Islander	0	1 (5.3)	1 (2.5)
Multiple[c]	1 (5.0)	0	1 (2.5)
Ethnicity, n (%)			
Hispanic or Latino	7 (35.0)	2 (10.5)	9 (22.5)
Not Hispanic or Latino	13 (65.0)	16 (84.2)	30 (75.0)
Body mass index (kg/m^2)			
Mean (SD)	28.8 (7.9)	29.5 (4.9)	29.2 (6.5)
Median (min, max)	25.9 (17.1, 45.1)	29.6 (20.8, 38.7)	28.9 (17.1, 45.1)
Concomitant alerting agents,[d] n (%)	5 (25.0)	9 (47.4)	15 (37.5)
No concomitant alerting agents	15 (75.0)	10 (52.6)	25 (62.5)

[a]Completer analysis set. [b]Of 40 enrolled participants with idiopathic hypersomnia who completed the study, 39 had BL IHSS data (20 reported LSN [>9 hours] and 19 reported no LSN [≤9 hours]). [c]Participant reported more than 1 race.
[d]Alerting agents were defined as stimulants or wake-promoting agents; participants must have taken the same dosage for ≥1 month before screening visit 1 with no plan to adjust dosage during the study.
BL, baseline; IHSS, Idiopathic Hypersomnia Severity Scale; LSN, long sleep need; max, maximum; min, minimum; SD, standard deviation.

- Twenty of 24 participants with LSN and 19/21 participants without LSN completed the study

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