339

DUET STUDY 407

Improvements in Sleep Inertia With Low-Sodium Oxybate Treatment in Participants With Narcolepsy Type 1 and Type 2 in the DUET Study

Deborah A. Nichols, MS[1]; Logan D. Schneider, MD[2]; David T. Plante, MD, PhD[3]; Jing Dai, PhD[1]; Teresa L. Steininger, PhD[1]; Marisa Whalen, PharmD[4]; Alyssa Cairns, PhD[4]; Chad M. Ruoff, MD[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]Department of Psychiatry, University of Wisconsin-Madison, Madison, WI, USA; [4]Jazz Pharmaceuticals, Philadelphia, PA, USA; [5]Mayo Clinic, Scottsdale, AZ, 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 individuals with narcolepsy type 1 (NT1) or type 2 (NT2) compared with baseline (BL)

Conclusions

- Participants with narcolepsy exhibited objective sleep inertia upon awakening at BL (prior to LXB treatment), which was more pronounced in participants with NT1
- After LXB treatment, substantial improvements in objective sleep inertia magnitude (based on Psychomotor Vigilance Test) and reduced subjective sleep inertia severity (based on Patient Global Impression of Severity) were observed in the overall narcolepsy cohort, and in NT1 and NT2 subcohorts
- Subjective sleepiness ratings upon awakening (based on Karolinska Sleepiness Scale) indicated that subjective sleep inertia magnitude was small and similar at BL and EOT, but there was less subjective sleepiness at EOT overall
- These data suggest that sleep inertia in narcolepsy manifests primarily as objective cognitive impairment, which is effectively mitigated by LXB treatment

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.** Dauvilliers Y, et al. *Neurology.* 2019;92(15):e1754-e1762. **5.** Alexander JK, et al. Poster presented at: Annual Meeting of the Associated Professional Sleep Societies; June 8-11, 2025; Seattle, WA. **6.** Trotti LM. *Continuum (Minneapolis, Minn).* 2020;26(4):890-907. **7.** Barateau L, et al. *Sleep.* 2024;47(5):zsad323. **8.** Dauvilliers Y, et al. *Sleep Med Rev.* 2022;66:101709. **9.** Kim JR, et al. *PLoS One.* 2026;21(1):e0337992. **10.** Hilditch CJ & McHill AW. *Nat and Sci Sleep.* 2019:155-165. **11.** Roth B. *Int J Neurol.* 1981;15(1-2):108-118. **12.** Xywav® (calcium, magnesium, potassium, and sodium oxybates) oral solution, CIII [prescribing information]. Palo Alto, CA: Jazz Pharmaceuticals, Inc. **13.** Nichols DA, et al. *Neurol Ther.* 2025;14(4):1705-1727. **14.** Trotti LM, et al. *J Clin Sleep Med.* 2022;18:1395–1403. **15.** Schneider LD, et al. *Neurol Ther.* 2026; April 30 Online Ahead of Print.

Support and Acknowledgments: This study was supported by Jazz Pharmaceuticals. Under the direction of the authors, Peloton Advantage, LLC (an OPEN Health company) employees Martina Kusi-Mensah, PharmD, and Emily Bruggeman, PhD, provided medical writing support and an editor provided editorial support for this poster, which were funded by Jazz Pharmaceuticals.

Disclosures: DA Nichols, J Dai, M Whalen, and **A Cairns** are full-time employees of Jazz Pharmaceuticals who, in the course of this employment, have received stock options exercisable for, and other stock awards of, ordinary shares of Jazz Pharmaceuticals, plc. TL Steininger is a former full-time employee and current contract worker for Jazz Pharmaceuticals who has received shares of Jazz Pharmaceuticals, plc. **LD Schneider** has received personal compensation for serving on advisory boards and speakers bureaus for Avadel, Eisai, and Jazz Pharmaceuticals. **DT Plante** is a consultant and advisory board member for Jazz Pharmaceuticals. He has also served as a consultant/advisory board member for Alkermes, Centessa, Harmony Biosciences, and Takeda; an advisory board member for Apnimed; and a consultant for Aditum Bio and Teva Pharmaceuticals (Australia). **CM Ruoff** has served as an advisory board member for Alkermes, Eisai, Jazz Pharmaceuticals, and Takeda, and has received grant funding from Jazz Pharmaceuticals. **HPA Van Dongen** serves as a consultant to Jazz Pharmaceuticals plc.

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

Scan this code to access this poster and supplemental material online. This code is not for promotional purposes.

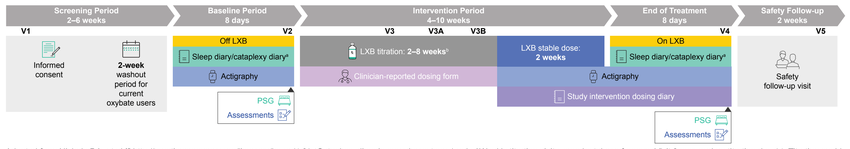
Introduction

- Sleep inertia is characterized by profound difficulty waking up, with grogginess, disorientation, and cognitive impairment immediately after awakening[1-3]
  - While sleep inertia is a core symptom of idiopathic hypersomnia (reported by 78% to 90%),[4,5] it may also be experienced by individuals with narcolepsy[6-8]
  - In the general population, sleep inertia may last 15 to 30 minutes[9,10]; in people with idiopathic hypersomnia, it can last for several hours[11]; in narcolepsy, sleep inertia is not well documented
- Low-sodium oxybate (LXB; Xywav®) is approved for the treatment of cataplexy or excessive daytime sleepiness in patients aged 7 years and older with narcolepsy and for the treatment of idiopathic hypersomnia in adults[12]
- Sleep inertia magnitude has not yet been evaluated in people with narcolepsy taking LXB

Methods

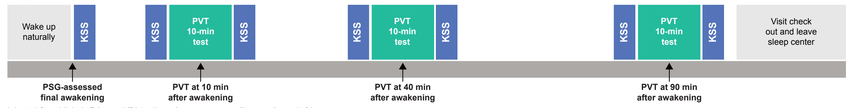
Figure 1. Study Design

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.[13] http://creativecommons.org/licenses/by-nc/4.0/. [a]Cataplexy diary in narcolepsy type 1 only. [b]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.[13] http://creativecommons.org/licenses/by-nc/4.0/.
BL, baseline; EOT, end of treatment; KSS, Karolinska Sleepiness Scale; min, minutes; PSG, polysomnography; PVT, Psychomotor Vigilance Test.

- 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 narcolepsy cohort, and separately for the NT1 and NT2 subcohort 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 decay 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) assessed in the NT1 and NT2 subcohorts have been previously reported[15] (and are reported in the Supplemental Information, accessed via the QR code)
- For additional Methods details, see the Supplemental Information, accessed via the QR code

Results

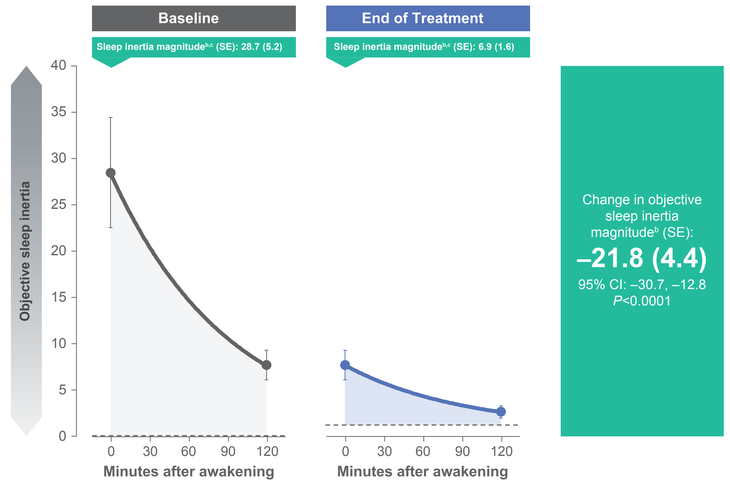
Table 1. Demographics and Baseline Characteristics[a]

Characteristic	NT1 (n=16)	NT2 (n=18)	Total Narcolepsy Cohort (n=34)
Age (years) Mean (SD)	35.6 (14.2)	35.9 (14.7)	35.7 (14.2)
Female, n (%)	13 (81.3)	12 (66.7)	25 (73.5)
Concomitant alerting agents,[b] n (%)	10 (62.5)	10 (55.6)	20 (58.8)

[a]Completer analysis set. [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.
BL, baseline; max, maximum; min, minimum; NT1, narcolepsy type 1; NT2, narcolepsy type 2; SD, standard deviation.

- Fifty-five participants with narcolepsy enrolled, and 34 participants completed the study; 13 participants transferred to a separate >9 g narcolepsy cohort and 8 participants discontinued the study

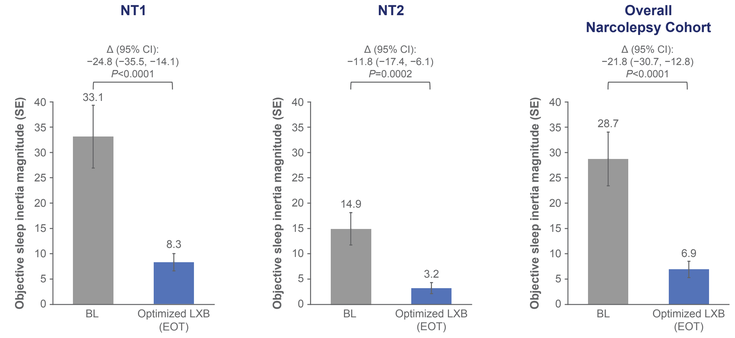
Figure 3. Objective Sleep Inertia (Based on PVT Lapses) Reduced With Optimized LXB Treatment After Awakening in the Overall Narcolepsy Cohort[a]



[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 overall narcolepsy cohort was 0 at BL and 0.8 at EOT.
BL, baseline; CI, confidence interval; EOT, end of treatment; LXB, low-sodium oxybate; NT1, narcolepsy type 1; NT2, narcolepsy type 2; 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 NT1 and NT2 subcohorts, are displayed in the Supplement, accessed via the QR code

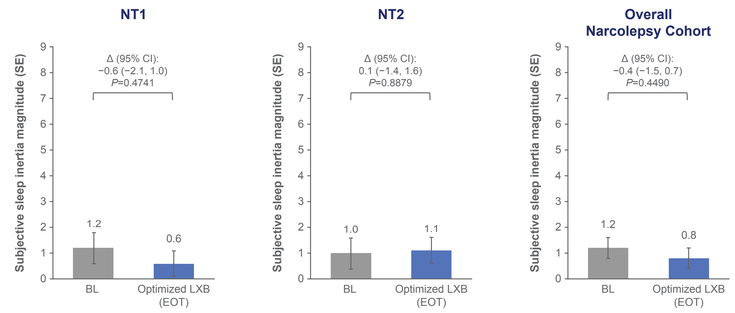
Figure 4. Objective Sleep Inertia Magnitude (Based on PVT Lapses) Reduced With Optimized LXB Treatment in Participants With Narcolepsy[a]



[a]Completer analysis set.
BL, baseline; CI, confidence interval; EOT, end of treatment; NT1, narcolepsy type 1; NT2, narcolepsy type 2; PVT, Psychomotor Vigilance Test; LXB, low-sodium oxybate.

- Participants with narcolepsy exhibited objective sleep inertia upon awakening at BL; this was more pronounced in NT1 than in NT2
- Participants in both subcohorts showed reductions in objective sleep inertia magnitude with LXB treatment
- The objective sleep inertia dissipation time constant (SE) was estimated to be 1.5 (0.3) hours for the overall narcolepsy cohort model
- For additional figures, see the Supplemental Information, accessed via the QR code

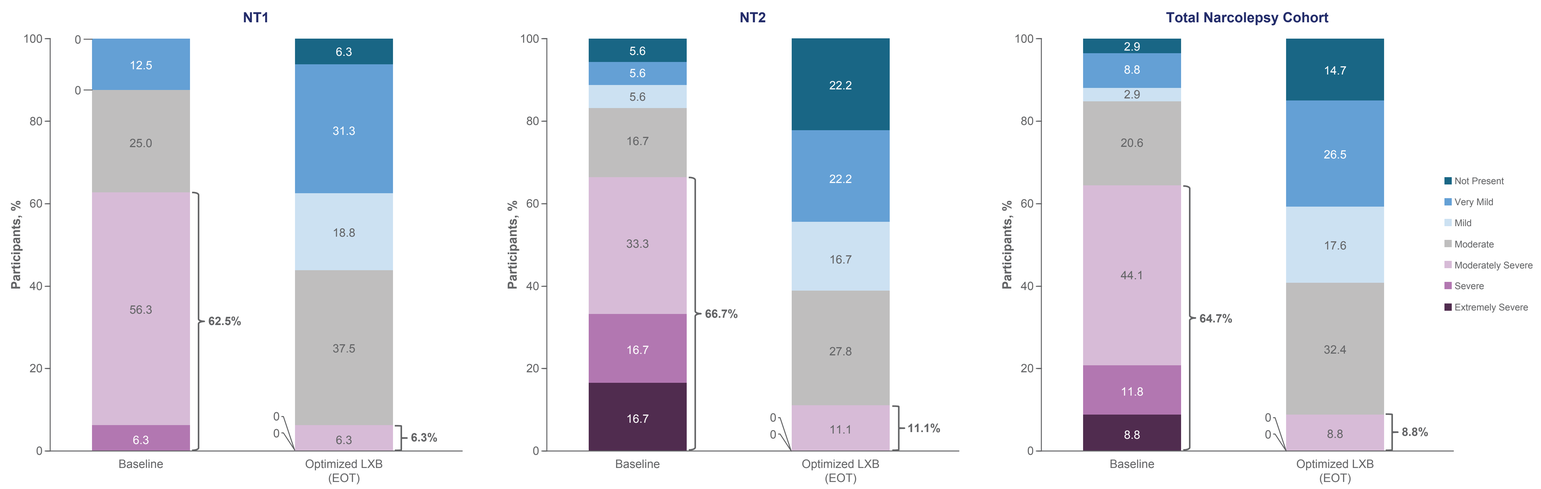
Figure 5. Subjective Sleep Inertia Magnitude (Based on KSS Ratings) Was Low at BL and Similar With Optimized LXB Treatment in Participants With Narcolepsy[a]



[a]Completer analysis set.
BL, baseline; CI, confidence interval; EOT, end of treatment; NT1, narcolepsy type 1; NT2, narcolepsy type 2; KSS, Karolinska Sleepiness Scale; LXB, low-sodium oxybate.

- Participants with NT1 and NT2 showed low subjective sleep inertia magnitude upon awakening at BL, which was similar at EOT
  - Subjective sleepiness estimated from modeling of KSS scores upon awakening were 6.2 at BL and 4.6 at EOT in the overall narcolepsy cohort (see Supplement, accessed via the QR code)
- The subjective sleep inertia dissipation time constant (SE) was estimated to be 0.5 (0.2) hours for the overall narcolepsy cohort model

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



The sums of percentages shown may not equal 100% due to rounding.
EOT, end of treatment; LXB, low-sodium oxybate; NT1, narcolepsy type 1; NT2, narcolepsy type.

- At BL, 64.7% of participants in the total narcolepsy cohort reported at least moderately severe sleep inertia, which decreased to 8.8% at EOT (P<0.0001; NT1, P=0.0027; NT2, P=0.0039)

339

DUET STUDY 407

Improvements in Sleep Inertia With Low-Sodium Oxybate Treatment in Participants With Narcolepsy Type 1 and Type 2 in the DUET Study

Deborah A. Nichols, MS[1]; Logan D. Schneider, MD[2]; David T. Plante, MD, PhD[3]; Jing Dai, PhD[1]; Teresa L. Steininger, PhD[1]; Marisa Whalen, PharmD[4]; Alyssa Cairns, PhD[4]; Chad M. Ruoff, MD[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]Department of Psychiatry, University of Wisconsin-Madison, Madison, WI, USA; [4]Jazz Pharmaceuticals, Philadelphia, PA, USA; [5]Mayo Clinic, Scottsdale, AZ, USA; [6]Sleep and Performance Research Center, Washington State University Health Sciences Spokane, Spokane, WA, USA

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.

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

Scan this code to access this poster and supplemental material online. This code is not for promotional purposes.

Supplemental Material

Supplemental Figure S1. Inclusion/Exclusion Criteria

Inclusion Criteria

- Adults 18–75 with a primary diagnosis of NT1 or NT2 (per ICSD-3 or DSM-5 criteria)
- ESS score >10[a]
- Concomitant stable use of anticataplectics or 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[d])
- 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; NT1, narcolepsy type 1; NT2, narcolepsy type 2; 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=55; NT1, n=26; NT2, n=29; 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=34; NT1, n=16; NT2, n=18; evaluated for Patient Global Impression of Severity [PGI-S], Psychomotor Vigilance Test [PVT], and Karolinska Sleepiness Scale [KSS] nonlinear modeling analyses)

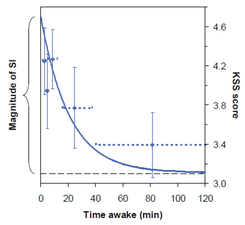
Outcomes

- 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 idiopathic hypersomnia study cohort was analyzed separately; results are reported in **Poster 338**
- 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.[4] https://creativecommons.org/licenses/by/4.0/.
KSS, Karolinska Sleepiness Scale; min, minutes; SI, sleep inertia.

Supplemental Table S1. Summary of TEAEs[a]

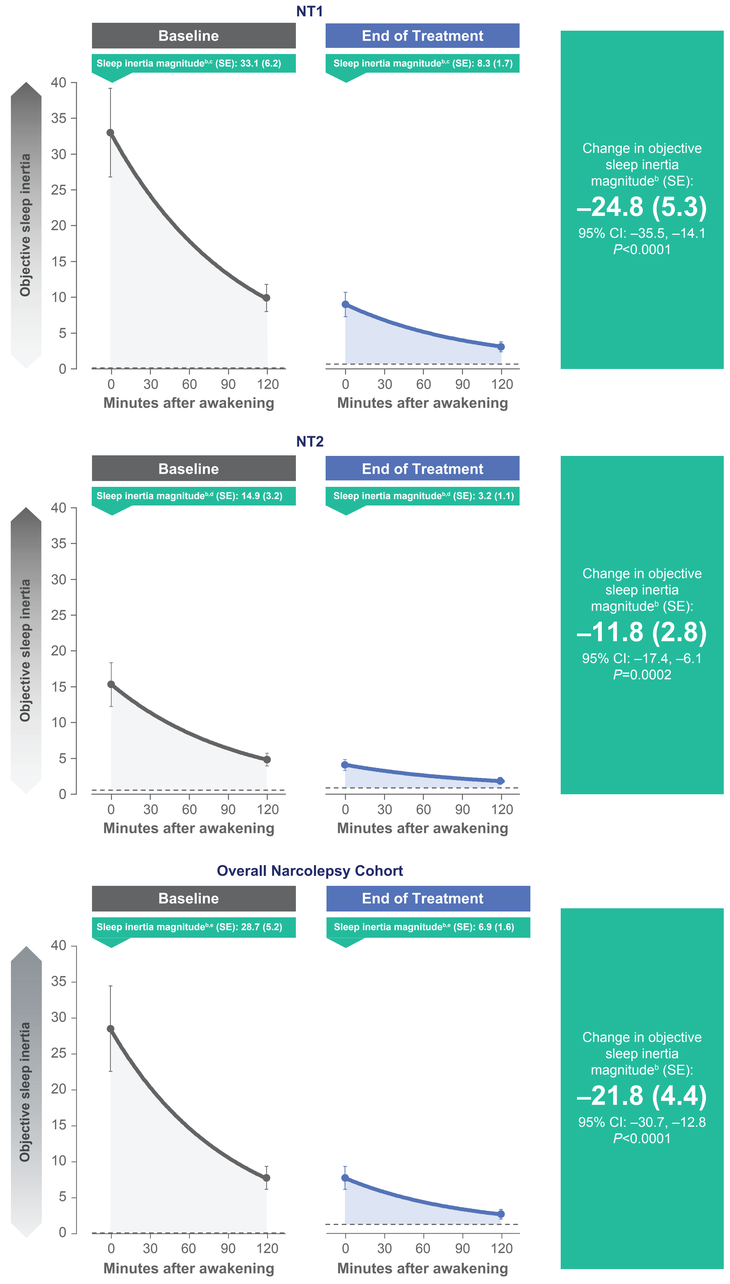
Participants, n (%)	NT1 (n=26)	NT2 (n=29)	Total Narcolepsy Cohort (N=55)
With ≥1 TEAE	16 (61.5)	18 (62.1)	34 (61.8)
With ≥1 serious TEAE	0	0	0
With ≥1 TEAE related to treatment	15 (57.7)	15 (51.7)	30 (54.5)
With ≥1 TEAE leading to discontinuation	0	4 (13.8)	4 (7.3)
TEAEs occurring in ≥5% of participants			
Nausea	6 (23.1)	7 (24.1)	13 (23.6)
Dizziness	5 (19.2)	3 (10.3)	8 (14.5)
Headache	2 (7.7)	5 (17.2)	7 (12.7)
Somnolence	0	6 (20.7)	6 (10.9)
Vomiting	3 (11.5)	3 (10.3)	6 (10.9)
Anxiety	1 (3.8)	3 (10.3)	4 (7.3)
Nasal congestion	1 (3.8)	3 (10.3)	4 (7.3)
Oropharyngeal pain	2 (7.7)	2 (6.9)	4 (7.3)
Brain fog	0	3 (10.3)	3 (5.5)
Cough	0	3 (10.3)	3 (5.5)
Decreased appetite	1 (3.8)	2 (6.9)	3 (5.5)
Enuresis	3 (11.5)	0	3 (5.5)
Hypoesthesia	2 (7.7)	1 (3.4)	3 (5.5)

[a]Safety analysis set includes all participants who enrolled in the study and took ≥1 dose of the study drug after the BL period.
BL, baseline; NT1, narcolepsy type 1; NT2, narcolepsy type 2; TEAE, treatment-emergent adverse event.

Lowest Highest | Lowest Highest | Lowest Highest

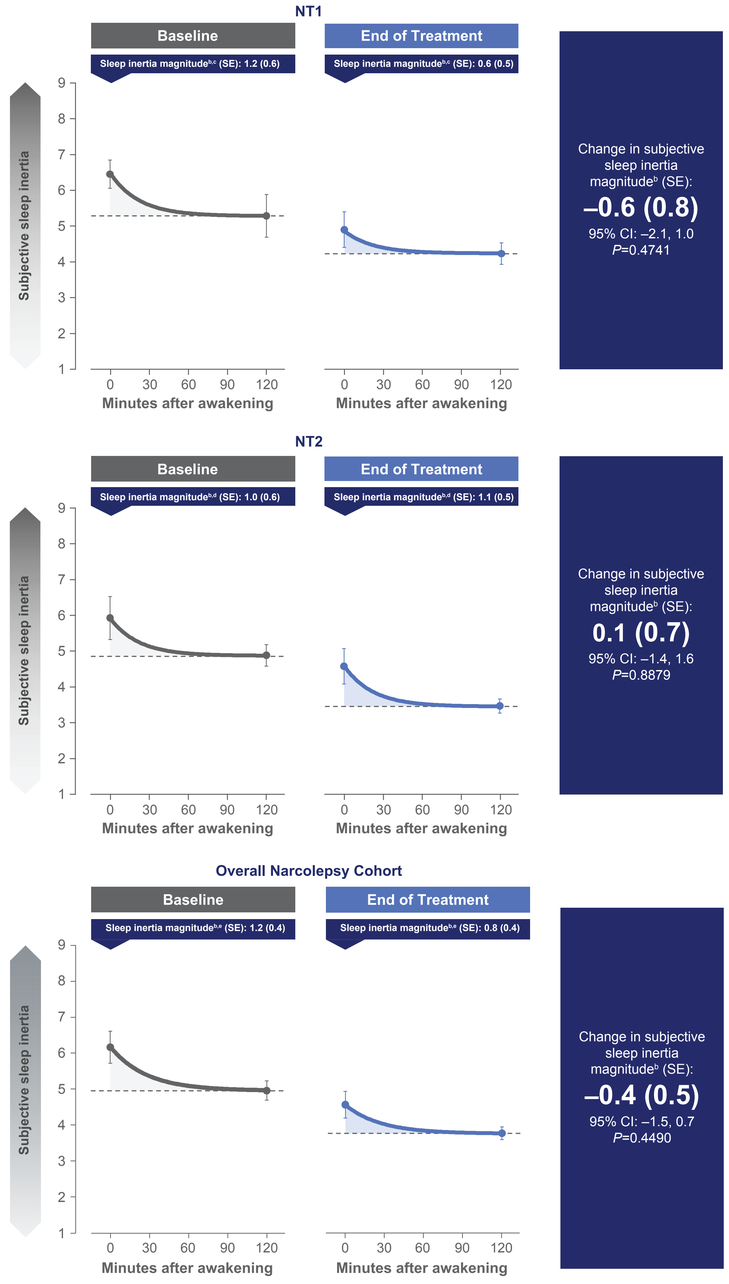
- Comparable proportions of participants with NT1 (61.5%) or NT2 (62.1%) reported ≥1 TEAE
- Most TEAEs were mild (34.6%, 27.6%) or moderate (26.9%, 34.5%) in severity for participants with NT1 or NT2, respectively

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



[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 participants with NT1 was 0 at BL and 0.6 at EOT. [d]The asymptote for participants with NT2 was 0.5 at BL and 0.8 at EOT. [e]The asymptote for the overall narcolepsy cohort was 0 at BL and 0.8 at EOT.
BL, baseline; CI, confidence interval; EOT, end of treatment; LXB, low-sodium oxybate; NT1, narcolepsy type 1; NT2, narcolepsy type 2; PVT, Psychomotor Vigilance Test; SE, standard error.

Supplemental Figure S4. Subjective Sleep Inertia (Based on KSS Ratings) Showed No Change With Optimized LXB Treatment in Participants With Narcolepsy[a] After Awakening



[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 participants with NT1 was 5.3 at BL and 4.2 at EOT. [d]The asymptote for participants with NT2 was 4.9 at BL and 3.5 at EOT. [e]The asymptote for the overall narcolepsy cohort was 5.0 at BL and 3.8 at EOT.
BL, baseline; CI, confidence interval; EOT, end of treatment; KSS, Karolinska Sleepiness Scale; NT1, narcolepsy type 1; NT2, narcolepsy type 2; LXB, low-sodium oxybate; SE, standard error.

339

Improvements in Sleep Inertia With Low-Sodium Oxybate Treatment in Participants With Narcolepsy Type 1 and Type 2 in the DUET Study

Deborah A. Nichols, MS[1]; Logan D. Schneider, MD[2]; David T. Plante, MD, PhD[3]; Jing Dai, PhD[1]; Teresa L. Steininger, PhD[1]; Marisa Whalen, PharmD[4]; Alyssa Cairns, PhD[4]; Chad M. Ruoff, MD[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]Department of Psychiatry, University of Wisconsin-Madison, Madison, WI, USA; [4]Jazz Pharmaceuticals, Philadelphia, PA, USA; [5]Mayo Clinic, Scottsdale, AZ, USA; [6]Sleep and Performance Research Center, Washington State University Health Sciences Spokane, Spokane, WA, USA

Supplemental Material

Supplemental Table S2. Demographics and Baseline Characteristics[a]

Characteristic	NT1 (n=26)	NT2 (n=29)	Total Narcolepsy Cohort (N=55)
Age (years)			
Mean (SD)	34.6 (12.6)	32.4 (13.2)	33.4 (12.9)
Median (min, max)	31.5 (18, 68)	28.0 (20, 75)	29.0 (18, 75)
Sex at birth, n (%)			
Male	7 (26.9)	8 (27.6)	15 (27.3)
Female	19 (73.1)	21 (72.4)	40 (72.7)
Race, n (%)			
White	19 (73.1)	25 (86.2)	44 (80.0)
Black or African American	4 (15.4)	3 (10.3)	7 (12.7)
Asian	1 (3.8)	1 (3.4)	1 (1.8)
Multiple[b]	2 (7.7)	0	2 (3.6)
Unknown	0	0	1 (1.8)
Ethnicity, n (%)			
Hispanic or Latino	1 (3.8)	2 (6.9)	3 (5.5)
Not Hispanic or Latino	25 (96.2)	27 (93.1)	52 (94.5)
Body mass index (kg/m^2), mean (SD)	30.3 (6.9)	28.7 (6.6)	29.5 (6.7)
Healthy weight 18.5 to <25	8 (30.8)	11 (37.9)	19 (34.5)
Overweight 25 to <30	8 (30.8)	9 (31.0)	17 (30.9)
Obese ≥30	10 (38.5)	9 (31.0)	19 (34.5)
Concomitant alerting agents,[c] n (%)			
Yes	16 (61.5)	15 (51.7)	31 (56.4)
No	10 (38.5)	14 (48.3)	24 (43.6)

[a]Safety analysis set. [b]Participant reported more than 1 race. [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; max, maximum; min, minimum; NT1, narcolepsy type 1; NT2, narcolepsy type 2; SD, standard deviation.

Supplemental Table S3. Demographics and Baseline Characteristics[a]

Characteristic	NT1 (n=16)	NT1 (n=18)	Total Narcolepsy Cohort (n=34)
Age (years)			
Mean (SD)	35.6 (14.2)	35.9 (14.7)	35.7 (14.2)
Median (min, max)	30.5 (20, 68)	31.0 (22, 75)	31.0 (20, 75)
Sex at birth, n (%)			
Male	3 (18.8)	6 (33.3)	9 (26.5)
Female	13 (81.3)	12 (66.7)	25 (73.5)
Race, n (%)			
Asian	2 (12.5)	0	2 (5.9)
Black or African American	3 (18.8)	1 (5.6)	4 (11.8)
White	10 (62.5)	17 (94.4)	27 (79.4)
Unknown	1 (6.3)	0	1 (2.9)
Ethnicity, n (%)			
Hispanic or Latino	1 (6.3)	2 (11.1)	3 (8.8)
Not Hispanic or Latino	15 (93.8)	16 (88.9)	31 (91.2)
Body mass index (kg/m^2)			
Mean (SD)	28.9 (6.5)	29.2 (6.0)	29.1 (6.2)
Median (min, max)	27.3 (20, 40.6)	27.8 (21.7, 41.2)	27.4 (20, 41.2)
Concomitant alerting agents,[b] n (%)	10 (62.5)	10 (55.6)	20 (58.8)
No concomitant alerting agents	6 (37.5)	8 (44.4)	14 (41.2)

[a]Completer analysis set. [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.
max, maximum; min, minimum; NT1, narcolepsy type 1; NT2, narcolepsy type 2; SD, standard deviation.

- Thirty-four participants completed the study (NT1, n=16; NT2, n=18); 13 participants transferred to a separate >9 g narcolepsy cohort and 8 participants discontinued the study

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.

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

Scan this code to access this poster and supplemental material online. This code is not for promotional purposes.