Investor Update

July 2, 2019

Jazz Pharmaceuticals®

Forward-Looking Statements

"Safe Harbor" Statement Under the Private Securities Litigation Reform Act of 1995

This slide deck and the accompanying oral presentation contain forward-looking statements, including, but not limited to, statements related to Jazz Pharmaceuticals' current plans and expectations regarding the commercial launch and availability of Sunosi™ (solriamfetol) in the U.S. (including launch timing); the commercial opportunity for Sunosi in the U.S., including pricing expectations, GTN estimates, projected future Sunosi net sales and performance, and market access; planned sales and marketing activities for Sunosi; planned development activities and potential novel opportunities for additional Sunosi indications; the potential benefits of Sunosi; and other statements that are not historical facts. These forward-looking statements are based on the company's current plans, objectives, estimates, expectations and intentions and inherently involve significant risks and uncertainties. Actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation, risks and uncertainties associated with: the company's ability to effectively commercialize Sunosi and achieve and maintain commercial success thereof; obtaining and maintaining appropriate pricing and reimbursement for Sunosi; delays or problems in the supply or manufacture of Sunosi; complying with applicable regulatory requirements; the time-consuming and uncertain regulatory approval process, including the risk that the company's regulatory submissions, including the MAA for solriamfetol, may not be approved by applicable regulatory authorities in a timely manner or at all; pharmaceutical product development and the uncertainty of clinical success, including risks related to failure or delays in initiating or completing clinical trials; the ability to achieve expected future financial performance and results; and other risks and uncertainties affecting the company, including those described from time to time under the caption "Risk Factors" and elsewhere in Jazz Pharmaceuticals plc's Securities and Exchange Commission filings and reports (Commission File No. 001-33500), including the company's Quarterly Report on Form 10-Q for the quarter ended March 31, 2019 and future filings and reports by the company. Other risks and uncertainties of which the company is not currently aware may also affect the company's forward-looking statements and may cause actual results and the timing of events to differ materially from those anticipated. The forward-looking statements made in this slide deck and the accompanying oral presentation are made only as of the date hereof or as of the dates indicated in the forward-looking statements, even if they are subsequently made available by the company on its website or otherwise.

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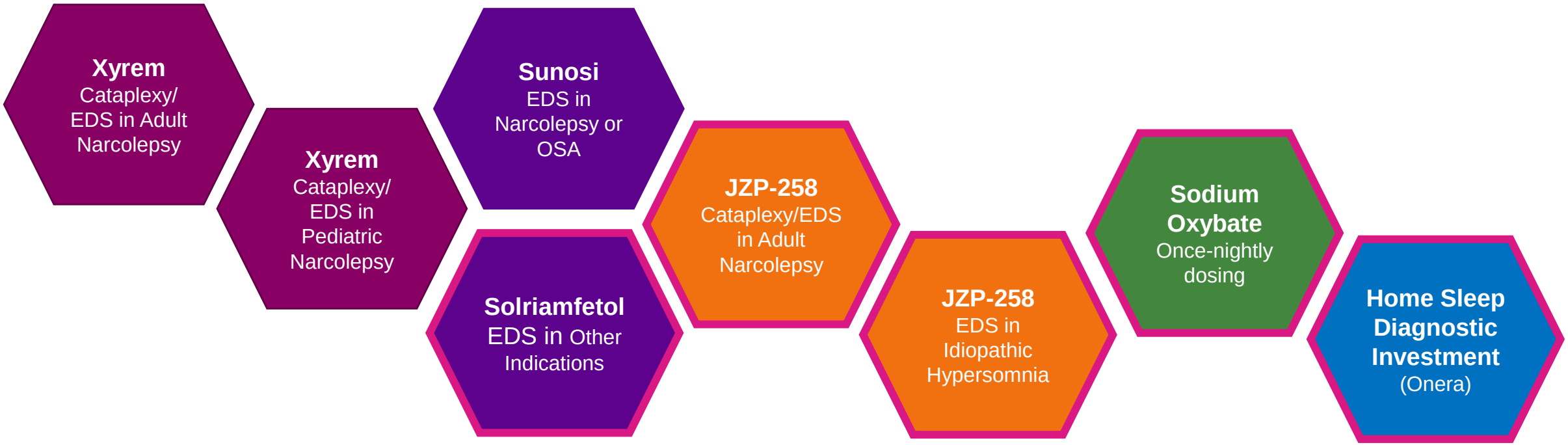
Agenda

Bruce Cozadd Chairman and Chief Executive Officer, Jazz Pharmaceuticals	Introduction
Michael J. Thorpy, MD Director of the Sleep-Wake Disorders Center at Montefiore Medical Center Professor of Clinical Neurology at Albert Einstein College of Medicine	Narcolepsy Disease Overview / Clinical Data
Richard K. Bogan, MD, FCCP, FAASM President of Bogan Sleep Consultants, LLC Associate Clinical Professor, University of South Carolina School of Medicine	OSA Disease Overview / Clinical Data
Lawrence Carter, Ph.D. Executive Director, Global Development Lead, Solriamfetol, Jazz Pharmaceuticals	Sunosi Label and Product Development
Mike Miller Executive Vice President, U.S. Commercial, Jazz Pharmaceuticals	Commercial Initiatives
Matt Young Executive Vice President and Chief Financial Officer, Jazz Pharmaceuticals	Financial Update
Dan Swisher President and Chief Operating Officer, Jazz Pharmaceuticals	Q&A
Robert Iannone, M.D., M.S.C.E. Executive Vice President, Research and Development, Jazz Pharmaceuticals	Q&A
Allen Yang, M.D. Senior Vice President, Clinical Development, Jazz Pharmaceuticals	Q&A
Jed Black, M.D. Senior Vice President, Sleep and CNS Medicine, Jazz Pharmaceuticals	Q&A

Jazz: Growing our Leadership in Sleep Medicine

Investing in Our Sleep Therapeutic Area



= planned or current investigational program

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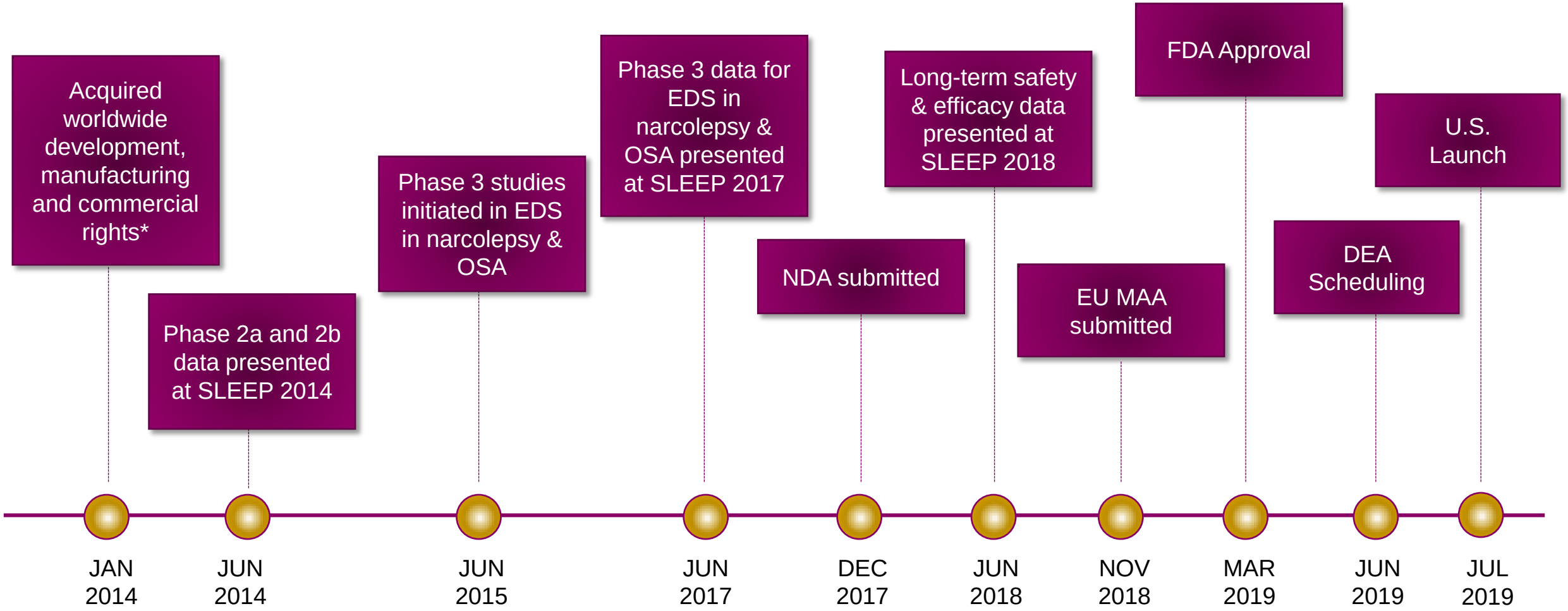
Sunosi Timeline



* Other than in certain jurisdictions in Asia where SK Biopharmaceuticals retains rights

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Excessive Daytime Sleepiness in Narcolepsy

Michael J. Thorpy, M.D.
Director of the Sleep-Wake Disorders Center at Montefiore Medical Center
Professor of Neurology at Albert Einstein College of Medicine

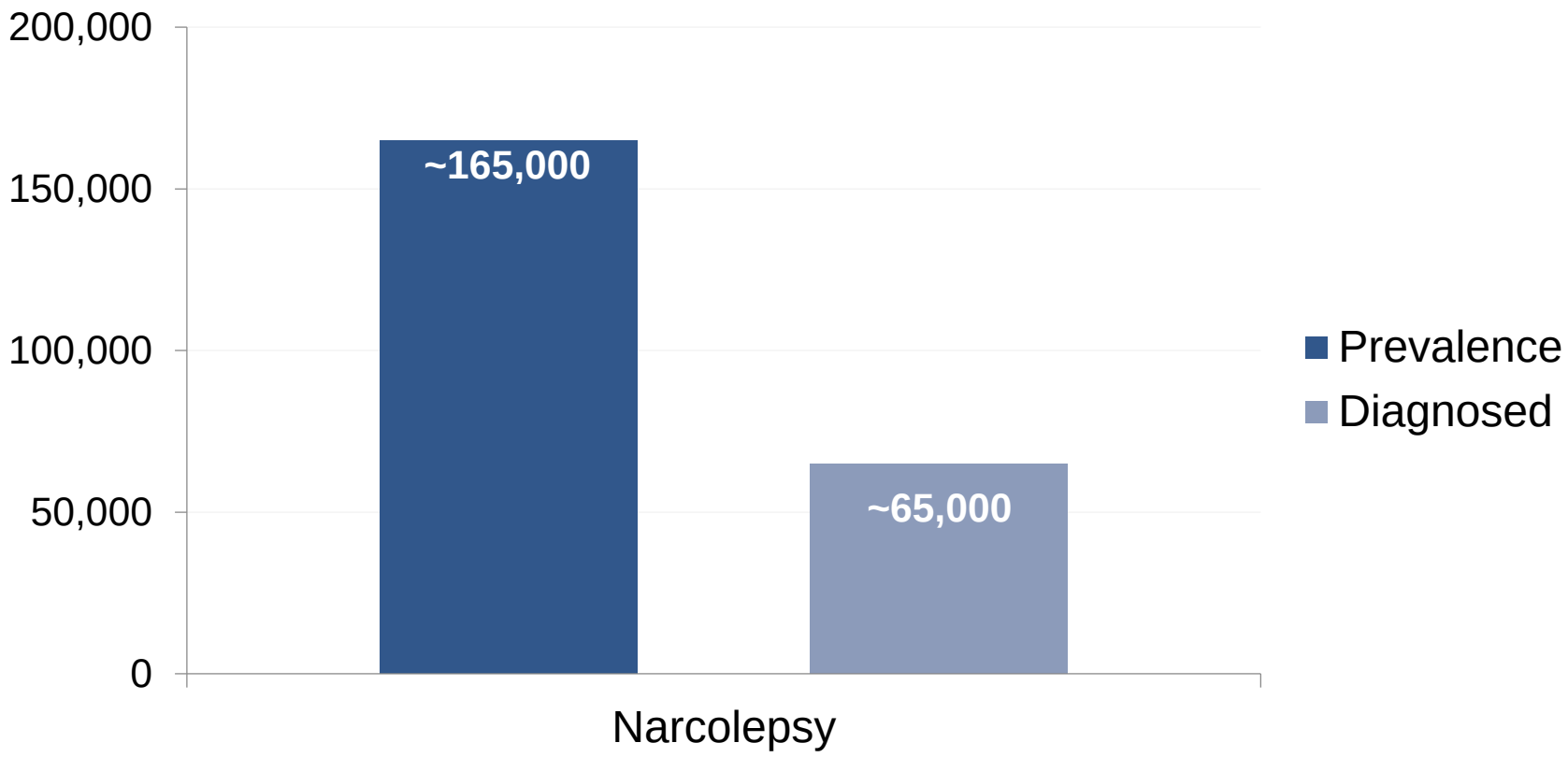
Narcolepsy Overview

- Narcolepsy prevalence in the U.S.
- Sleep/Wake pathophysiology
- Symptoms
- Diagnostic criteria
- Comorbidities associated with narcolepsy

TONES 2 and TONES 5 Clinical Trial Overview

- Solriamfetol Phase 3 clinical trial overview

Narcolepsy Remains an Underdiagnosed and Undertreated Disease

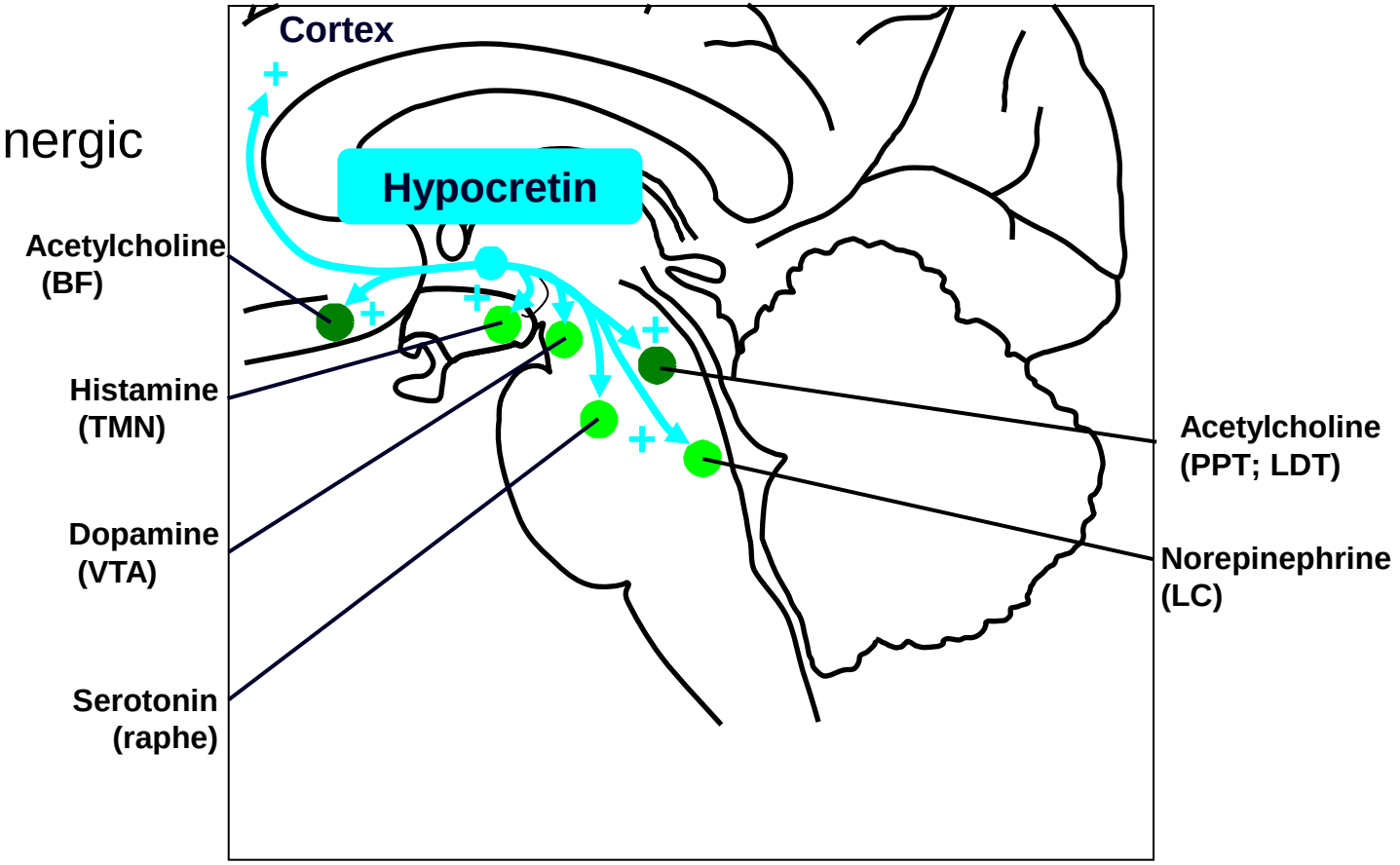


Source: SHA Claims Data Jul 2010 to Mar 2015

Pathophysiology: Neurotransmitter Dysregulation

- Animal model studies of narcolepsy[1] have suggested that hypocretin loss leads to dysregulation of dopaminergic and noradrenergic neurons and may result in:
  - Incomplete neuronal inhibition at night
  - Destabilized wakefulness during the day

Hypocretin/Orexin: Wake Regulation

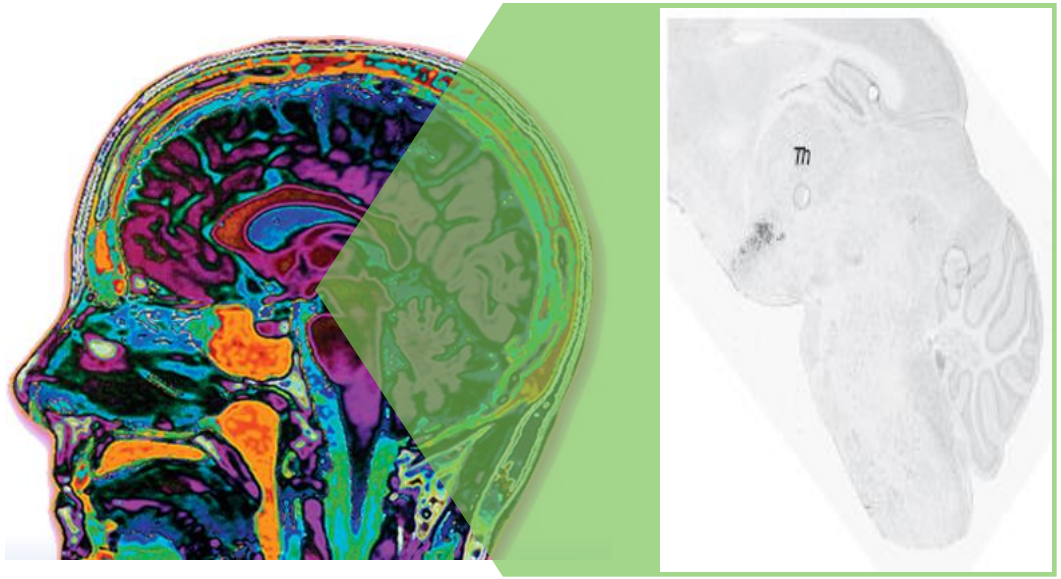


Adapted from Scammell and Black, 2004

[1] Xie X, et al. In: *Narcolepsy and Hypersomnia;* 2006:583-619.

Narcolepsy

- Chronic neurologic disorder[1]
  - No known cure
  - Typical onset in childhood
  - Lifelong treatment is required
- Narcolepsy with cataplexy—caused by a selective loss of hypocretin neurons in the hypothalamus, which may be mediated via autoimmune mechanisms[2]
  - Hypocretin is believed to stabilize sleep/wake states
- Results from dysregulation of the sleep/wake cycle and the intrusion of sleep into wakefulness[3]

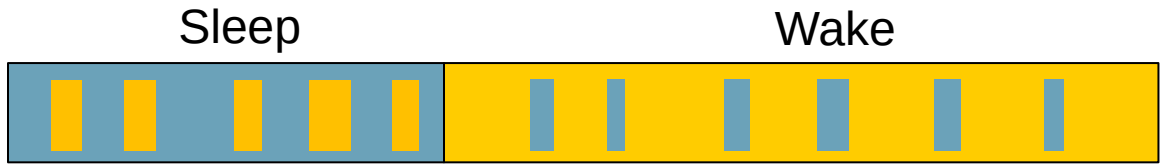


[1] National Institute of Neurological Disorders and Stroke (NINDS). Narcolepsy fact sheet. 2013. http://www.ninds.nih.gov/disorders/narcolepsy/detail_narcolepsy.htm#241193201; [2] Kornum BR, et al. *Curr Opin Neurobiol*. 2011;21(6):897-903; [3] Nishino S. *Sleep Med.* 2007; 8(4):373-399.

Narcolepsy Symptoms
Excessive Daytime Sleepiness—Most Common Symptom of Narcolepsy

Narcolepsy is a chronic disorder that involves poor control of the sleep/wake cycle[1-3]

- Sleep intrudes into wakefulness
  - EDS
  - Cataplexy (muscle atonia typically associated with REM occurs during the day)
- Wakefulness intrudes into sleep
  - Disrupted night time sleep
  - Hypnagogic/hypnopompic hallucinations
  - Sleep paralysis



Symptom	Prevalence Within Narcolepsy	Major Characteristics
EDS	100%	• Chronic pervasive sleepiness • Sleep attacks and inadvertent naps triggered by irresistible overwhelming urges to sleep
Cataplexy	~70%	• Triggered by strong emotions (e.g., laughter) • Atonia (loss of muscle tone)

[1] Houghton WC, et al. *Sleep Med Rev.* 2004;8(5):355-366; [2] Saper CB, et al. *Trends Neurosci.* 2001;24(12):726-731; [3] American Academy of Sleep Medicine (AASM). *International Classification of Sleep Disorders.* 3rd ed. 2014; [4] NINDS. Narcolepsy fact sheet. July 18, 2013. http://www.ninds.nih.gov/disorders/narcolepsy/detail_narcolepsy.htm.

Narcolepsy Diagnosis Criteria: ICSD-3 and DSM-5

ICSD-3

Narcolepsy Type 1 (narcolepsy with cataplexy)

- Chronic EDS (daily for at least 3 months) and
- Presence of one or both of the following:
  - Cataplexy **+** mean sleep latency ≤ 8 minutes; 2 or more SOREMPs on MSLT; SOREMP preceding PSG
  - CSF hypocretin-1 level is either ≤ 110 pg/mL or < 1/3 of mean values

Narcolepsy Type 2 (narcolepsy without cataplexy)[1,2]

- Chronic EDS (daily—at least 3 months)
- Mean sleep latency of ≤ 8 minutes + 2 or more SOREMPs on an MSLT; SOREMP on preceding PSG
- Cataplexy absent
- CSF hypocretin-1 concentration not measured **or** CSF hypocretin-1 level is > 110 pg/mL or > 1/3 mean values
- Hypersomnolence and/or MSLT findings not explained by other causes

DSM-5

- EDS ≥ 3 times/week for past 3 months (required)

One of the following:

- Cataplexy (a few times per month) defined as:
  - Hypocretin deficiency in CSF, with values ≤ 33% (healthy subjects) or ≤ 110 pg/mL, not in context of acute brain injury, inflammation, or infection

OR

  - Nocturnal PSG showing REM sleep latency ≤ 15 minutes, or MSLT showing mean sleep latency ≤ 8 minutes and 2 or more SOREMPs

[1] Patients classified as narcolepsy Type 2 that later develop cataplexy or should be reclassified as narcolepsy Type 1. [2] If CSF hypocretin-1 is tested later and found to be either ≤ 110 pg/mL or < 1/3 of mean normal control values, then the patient should be reclassified as narcolepsy Type 1.

AASM. *The International Classification of Sleep Disorders.* 3rd ed. 2014. http://www.aasmnet.org/library/default.aspx.

Medical Comorbidity in Narcolepsy:
Burden of Narcolepsy Disease (BOND) Study[1]

Objective: to evaluate medical comorbidity patterns in patients with a narcolepsy diagnosis in the U.S.

Patients/Methods: retrospective medical claims data analysis identified individuals ≥18 years of age with 1 diagnosis code for narcolepsy or continuously insured between 2006 and 2010, and controls without narcolepsy matched 5:1 on age, gender, region, and payer*

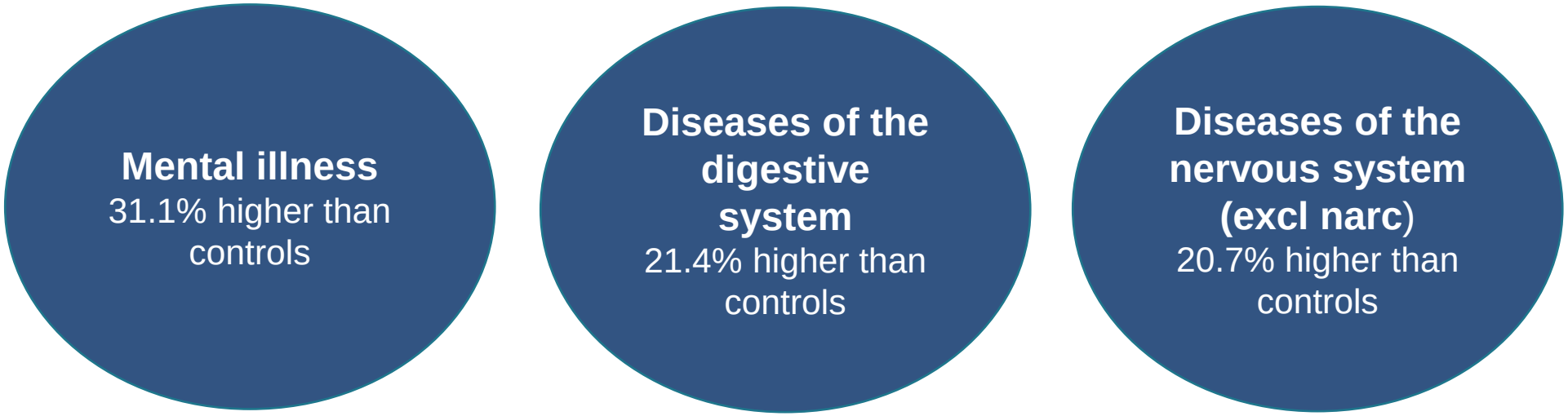
Results: The final study group included 9312 subjects with narcolepsy and 46,559 controls (each group: average age, 46.1 years; 59% female)

*Narcolepsy and control subjects were compared for frequency of comorbid conditions, identified by the appearance of >1 diagnosis code(s) mapped to a Clinical Classification System (CCS) level 1 category any time during the study period, and on specific subcategories, including recognized narcolepsy comorbidities of obstructive sleep apnea (OSA) and depression.

[1]Black, J, et al. *SleepMed. 2017*; 33: 13-18.

BOND Study: Narcolepsy Cohort Demonstrated Significantly Higher Comorbidities Compared to Controls[1]

- The greatest comorbidities in the narcolepsy cohort was seen for

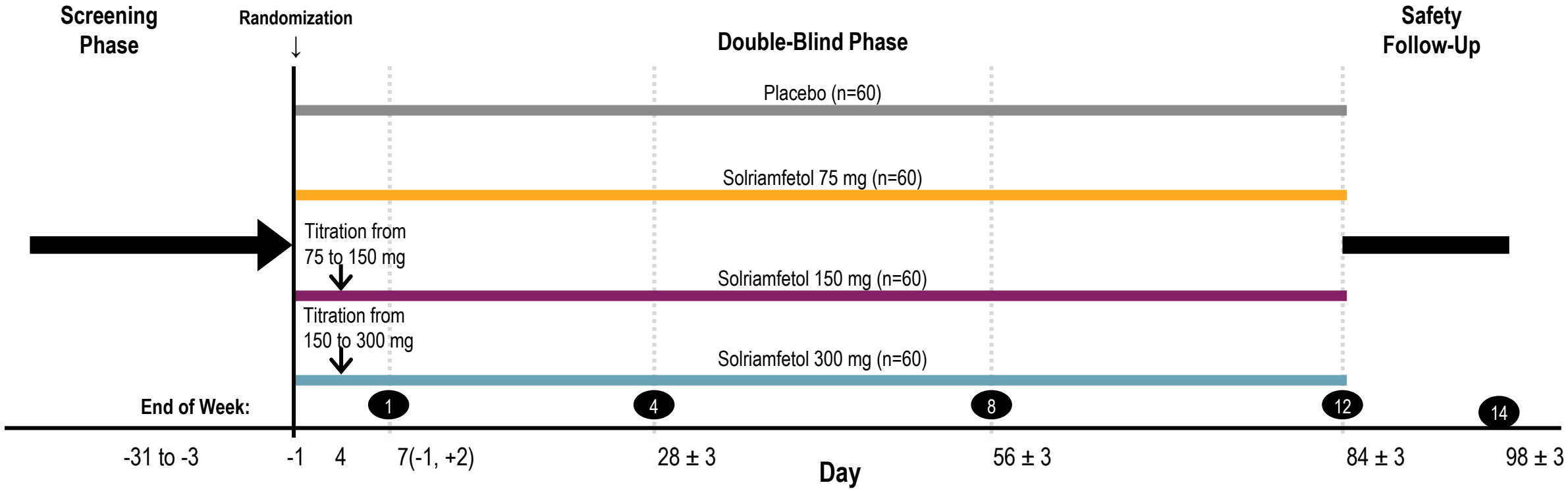


- Diseases of the circulatory system had high prevalence in both narcolepsy and control groups, but demonstrated excess prevalence of 16.6% in the narcolepsy group

[1]Black, J, et al. *SleepMed. 2017*; 33: 13-18.

TONES 2 and TONES 5
CLINICAL DATA

TONES 2[1]: A 12 Week Randomized, Placebo-controlled, Double-blind, Parallel-group Study to Assess Efficacy and Safety of Solriamfetol (Narcolepsy)

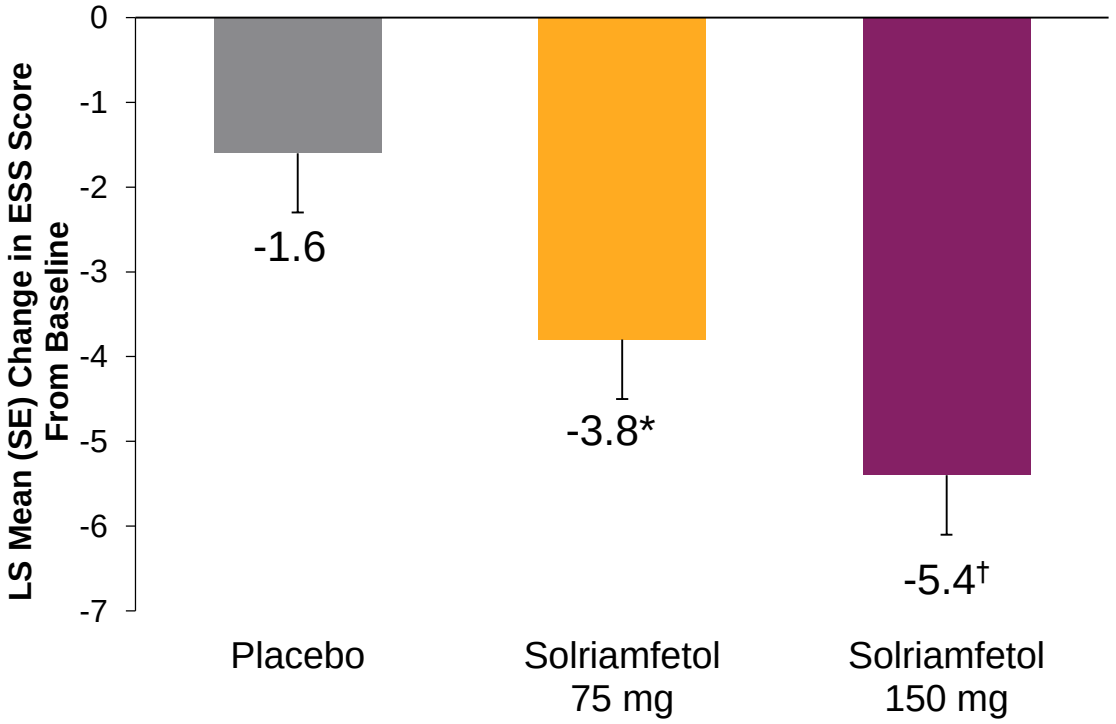


- Co-primary endpoints
  - Change from baseline to week 12 in mean sleep latency on 40-minute MWT
  - Change from baseline to week 12 in mean ESS score
- Key secondary endpoint
  - Percentage of patients reporting improvement on the Patient Global Impression of Change (PGI-C) at week 12

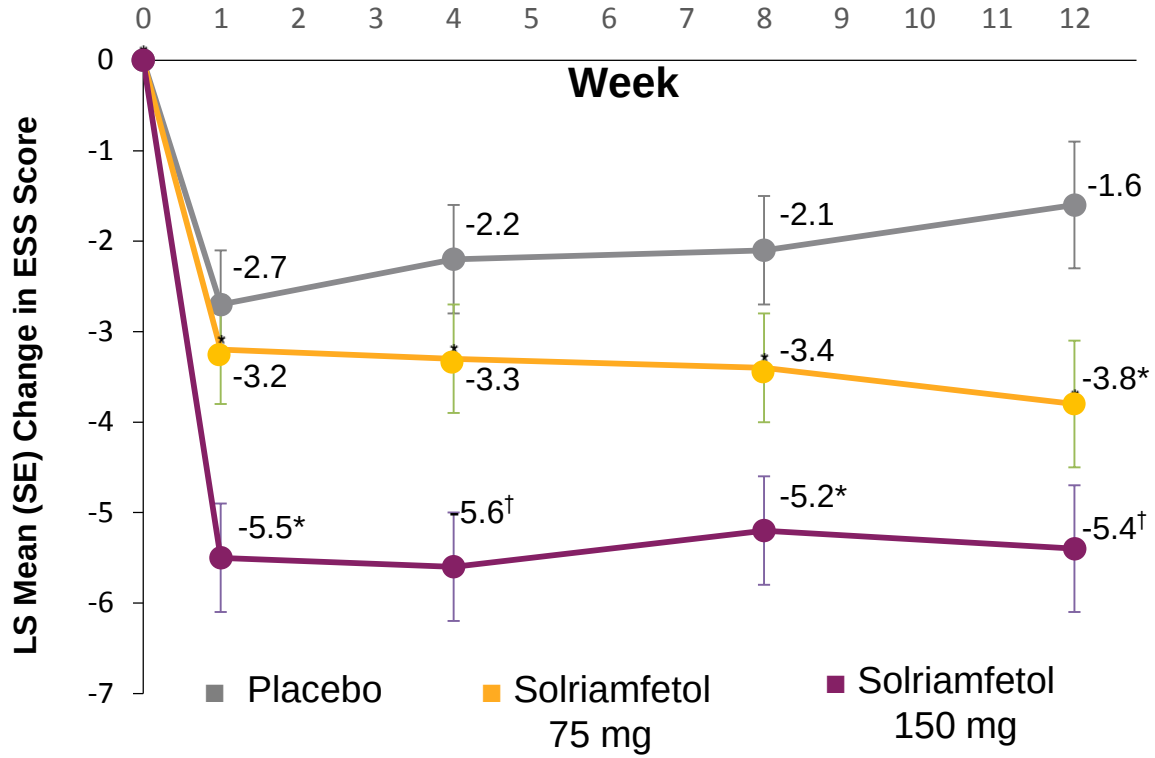
[1] Thorpy MJ, et al. *Ann Neurol*. 2019; 85(3):359-370.
Note: numbers indicate safety population.

Epworth Sleepiness Scale Score: Effects Observed as Early as Week 1 and Maintained over 12 weeks

Significant improvement from baseline to Week 12 in solriamfetol treatment groups (mITT Population)[1]



Improvement from Week 1 through Week 12 with solriamfetol 150 mg (mITT Population)[1]

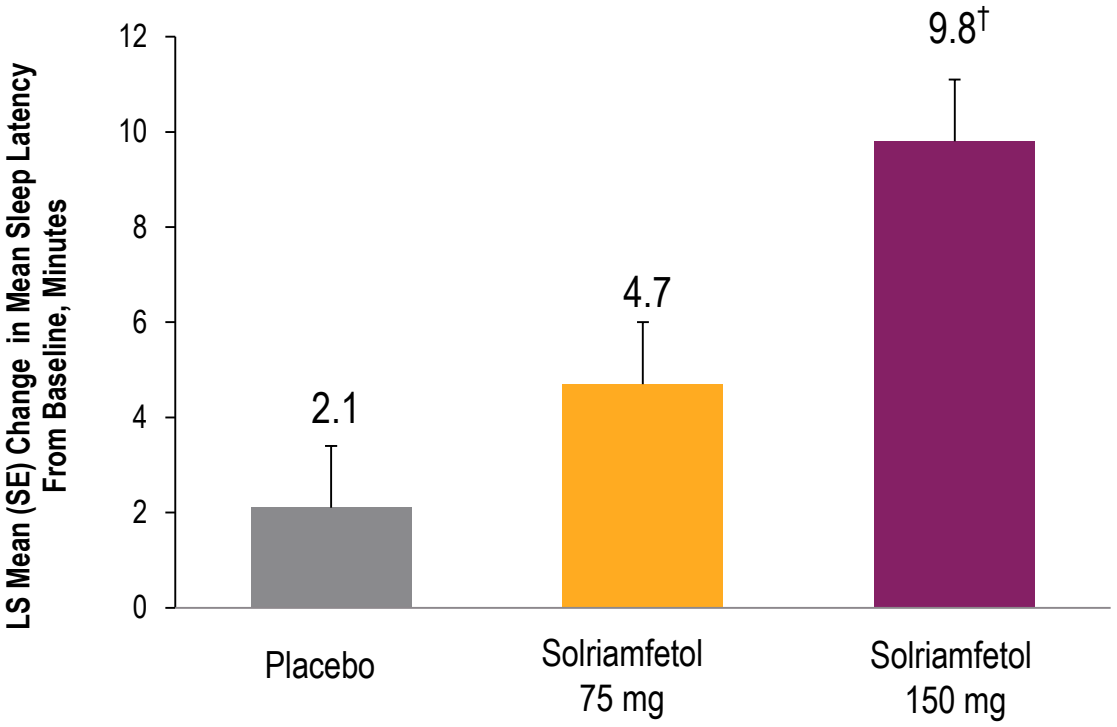


[1] Thorpy MJ, et al. *Ann Neurol*. 2019; 85(3):359-370. [2]300 mg data not shown

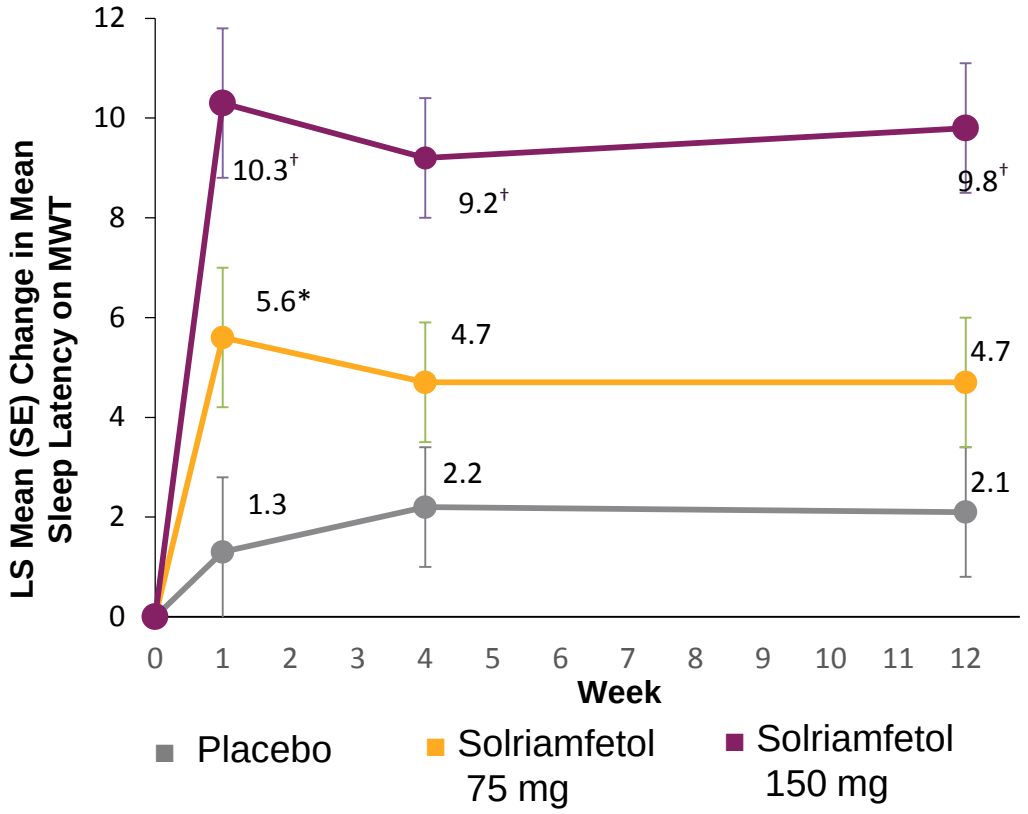
*$P < 0.05$ and †$P < 0.0001$ vs placebo

Maintenance of Wakefulness Test: Effects Observed as Early as Week 1 and Maintained over 12 weeks[2]

Significant improvement from baseline at Week 12 with solriamfetol 150 mg (mITT Population)[1]



Improvement from Week 1 through Week 12 with solriamfetol 150 mg (mITT Population)[1]



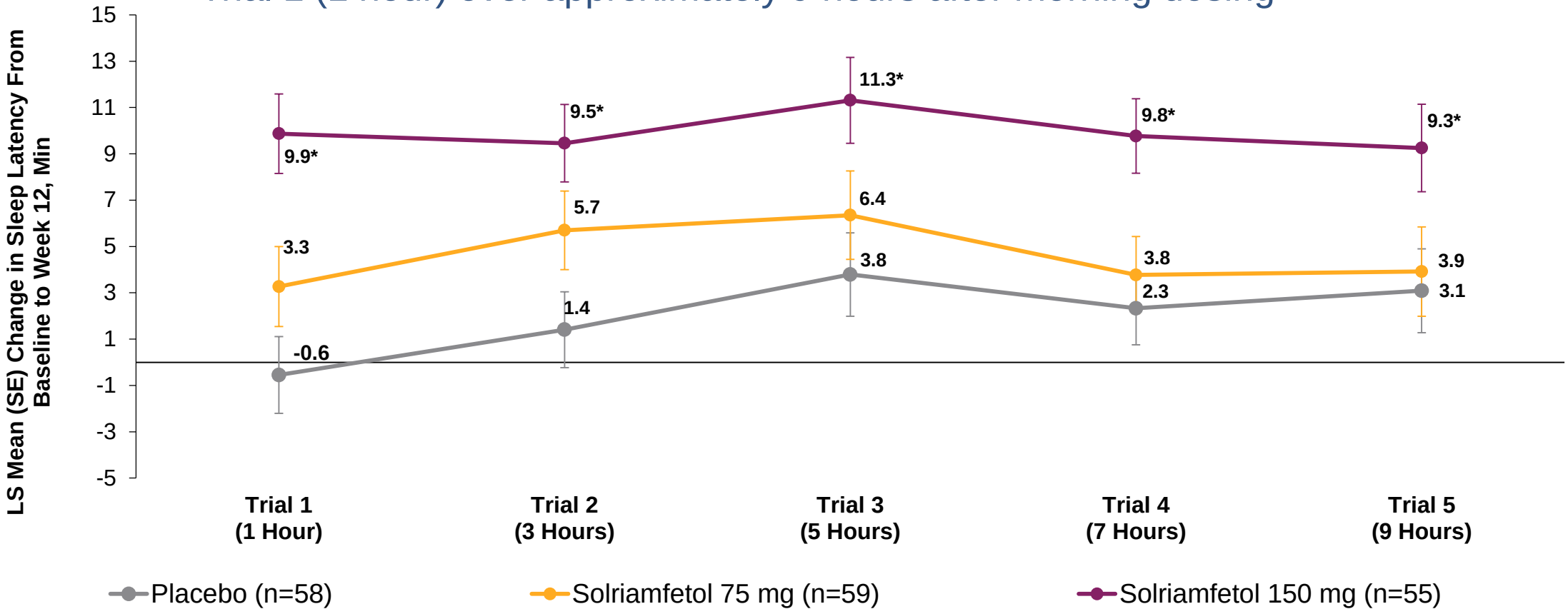
Negative change from baseline denotes improvement.

[1] Thorpy MJ, et al. *Ann Neurol*. 2019; 85(3):359-370. [2]300 mg data not shown

*$P < 0.05$ and †$P < 0.0001$ vs placebo

Maintenance of Wakefulness Test: Effects Observed Across the Day

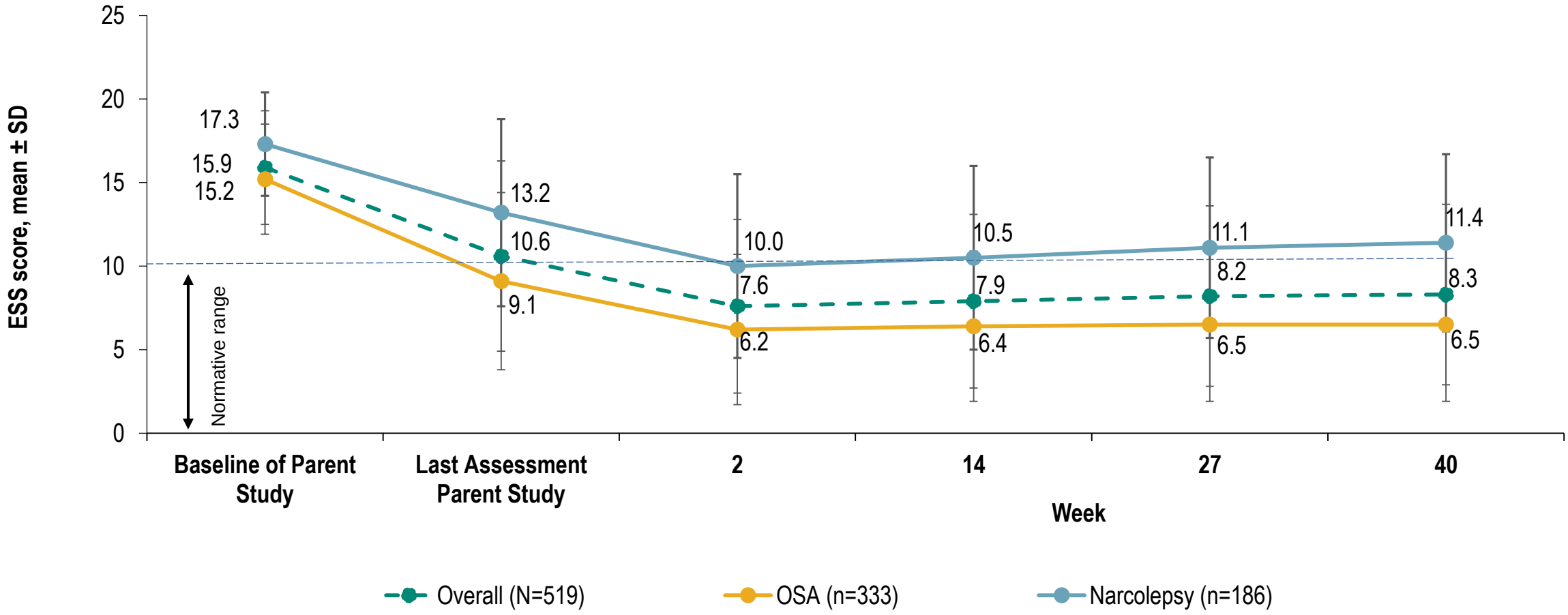
Solriamfetol 150 mg significantly increased sleep latency on MWT from Trial 1 (1 hour) over approximately 9 hours after morning dosing[1]



[1] Schweitzer PK et al. *Sleep*. 2018;41:A231; based on mITT Population. [2]300 mg data not shown
**P* <0.05.

TONES 5[1] Open-Label Phase: ESS Scores

Efficacy maintained throughout course of study with sustained reductions in mean ESS scores



[1] Malhotra A et al. *Sleep*. 2018;41(abstract suppl.):A230. [2]300 mg data not shown

TONES 2 and TONES 3: Adverse Events

Safety Population includes 300 mg dose per Product Information

Adverse event	Narcolepsy		OSA	
	Placebo (n = 59)	Solriamfetol combined (n = 177)	Placebo (n = 119)	Solriamfetol combined (n = 355)
Any adverse event, n (%)	27 (45.8)	121 (68.4)	57 (47.9)	241 (67.9)
Serious adverse event, n (%)	0	1 (0.6)	2 (1.7)	3 (0.8)
Adverse event leading to discontinuation, n (%)	1 (1.7)	9 (5.1)	4 (3.4)	26 (7.3)
Most common adverse events,[1] n (%)				
Headache	3 (5.1)	38 (21.5)	10 (8.4)	36 (10.1)
Nausea	1 (1.7)	19 (10.7)	7 (5.9)	28 (7.9)
Decreased appetite	1 (1.7)	19 (10.7)	1 (0.8)	27 (7.6)
Nasopharyngitis	3 (5.1)	16 (9.0)	8 (6.7)	18 (5.1)
Dry mouth	2 (3.4)	13 (7.3)	2 (1.7)	16 (4.5)
Anxiety	1 (1.7)	9 (5.1)	0	25 (7.0)

- Similar safety profile in narcolepsy and OSA and consistent with previous solriamfetol studies

[1]Reported in ≥5% of participants in combined solriamfetol group for both narcolepsy and OSA.

Rosenberg R, et al. Presented at: Annual Meeting of the AMCP Nexus; October 22-25, 2018; Orlando, FL.

Excessive Daytime Sleepiness in OSA

Richard K. Bogan, MD, FCCP, FAASM
President of Bogan Sleep Consultants, LLC
Associate Clinical Professor, University of South Carolina School of Medicine

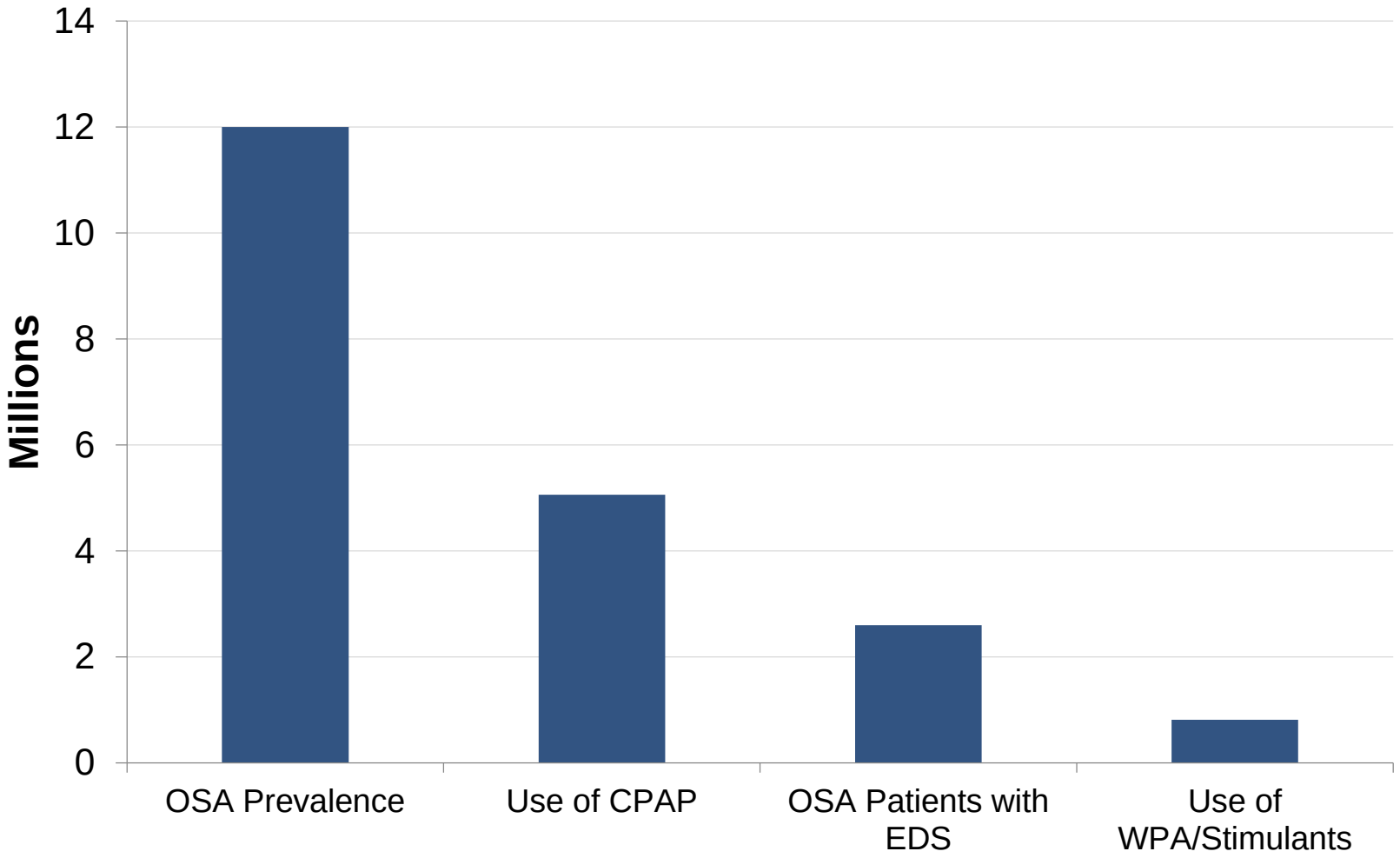
Obstructive Sleep Apnea Overview

- OSA prevalence in the U.S.
- Symptoms/consequences of OSA
- Current treatment options
- Excessive daytime sleepiness in OSA
- The burden of illness in OSA patients: Motor vehicle accidents, work-related accidents, and work productivity

TONES 3 and TONES 5 Study Overview

- Solriamfetol Phase 3 clinical trial overview

Estimated Prevalence – EDS in OSA

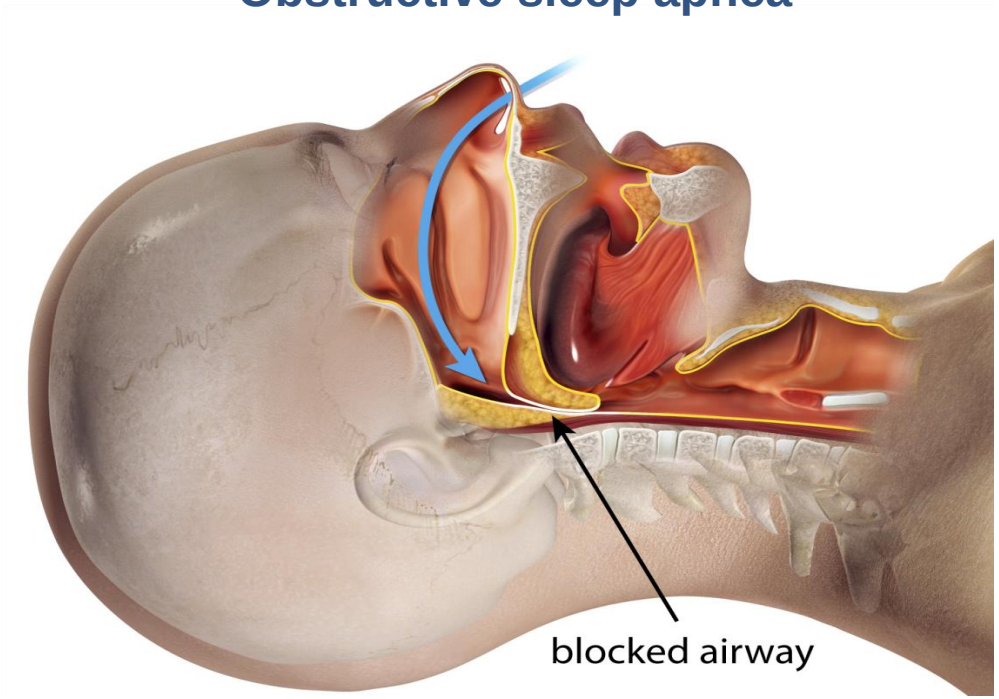


- Estimated 12M diagnosed OSA patients in 2016 in large U.S. health care claims database[1]
- 44% of OSA patients (5.0M) had evidence of CPAP use, while 56% (6.4M) did not[1]
- 20% - 25% (~2.7M) of OSA patients are estimated to have EDS after primary treatment[2,3]
- ~6% of OSA patients (720K) had ≥ 1 EDS medication prescription claim in a ~5 year claims database analysis[4,5]

[1] Won C, Hess G, Acquavella J, Bron M, Bujanover S, Villa KF. Poster presented at Annual meeting of the Academy of Managed Care Pharmacy. October 2018; Orlando, FL; [2] Pagel, JF. Am Fam Physician. 2009; 79(5):391-396; [3] Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. N Engl J Med. 1993; 328:1230-5; [4] Ohayon MM, Profant J, Gibson SP, Scheckner B, Milesi C. Poster presented at: 32nd Annual Meeting of the Associated Professional Sleep Societies; June 2018; Baltimore, MD; [5] Gasa et al, *J Sleep Res.* (2013) 22, 389–397

Patients With OSA Present With A Variety of Signs and Symptoms

Obstructive sleep apnea



Consequences of OSA[1]

- Sleep fragmentation
- Hypoxemia
- Hypercapnia
- Changes in intrathoracic pressure
- Increased sympathetic activity

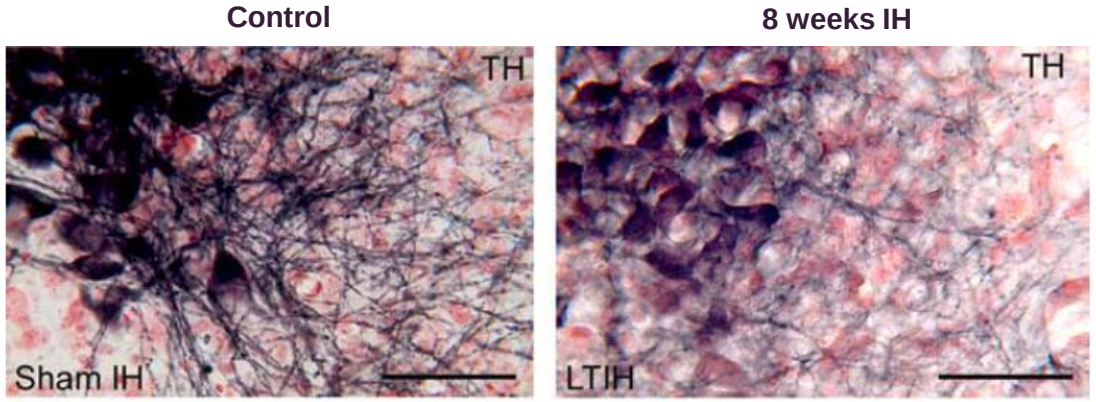
Clinical OSA Symptoms[1]

- EDS
- Snoring
- Interrupted breathing
- Awakenings due to gasping or choking

[1] Epstein LJ, et al. *J Clin Sleep Med*. 2009;5(3):263-276.

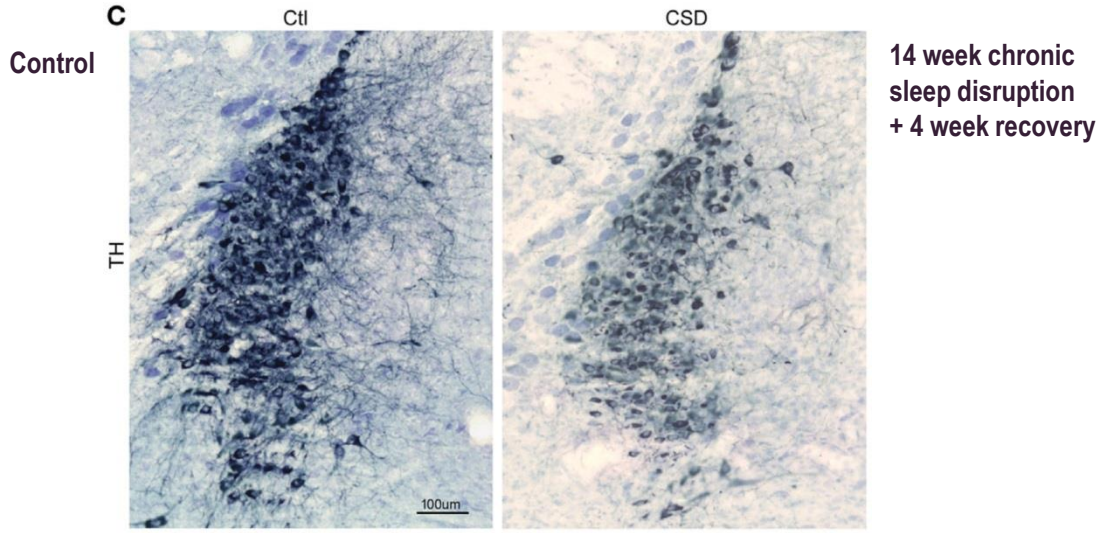
Intermittent Hypoxia/Sleep Fragmentation Effects on Wake-Promoting Regions of the Brain

- Two leading and connected theories for EDS in OSA
  - chronic intermittent hypoxia
  - sleep fragmentation
- Based on animal data, chronic intermittent hypoxia can lead to neuronal injury in wake-promoting regions of the brain[1,2]
  - Mice exposed to LTIH showed significant damage to/reductions in noradrenergic and dopaminergic wake-active neurons[1]



Loss of medial dendrites in locus coeruleus* was observed in mice exposed to 8 weeks of IH compared to controls.

- In animal studies, isolated sleep fragmentation was associated with neuronal injury in wake-promoting regions of the brain
  - Animal models of OSA have been produced by exposing animals to several weeks of chronic sleep disruption (24 hr. day)[1,2]
  - Models have shown significant reductions in catecholaminergic axonal projections to the frontal cortex[1], reduced locus coeruleus neuron excitability[1] and significant neuronal loss in wake-promoting regions of the brain[2]



Chronic sleep disruption resulted in degeneration of locus coeruleus wake-activated neurons[2]

[1] Zhu Y, et al. *J Neuro*. 2007;27(37):10060-10071; [2] Veasey S, et al. *Sleep*. 2004;27(2):194-201. *Locus coeruleus are the norepinephrine-containing neurons in the brain.

Current Treatments for OSA[1]

Current treatments[1]
Primary airway therapies Continuous positive airway pressure (CPAP) Bilevel positive airway pressure (BiPAP) Autotitrating positive airway pressure (APAP) Oral appliances - Mandibular-repositioning appliances - Tongue-retaining devices Surgical procedures
Behavioral therapies Weight loss Exercise Positional therapy Avoidance of alcohol/sedatives before bedtime
Adjunctive therapies Oxygen supplementation Bariatric surgery
Drug therapies Wake promoting agents and stimulants Topical nasal corticosteroids

- CPAP therapy is a standard of care for the treatment of the upper airway in OSA[1]
- EDS in OSA often persists despite optimized treatment of the upper airway[2]
- Airway therapies, such as CPAP, are associated with issues of acceptance, adherence, and tolerability[3]

[1] Epstein LJ, et al. *J Clin Sleep Med*. 2009;5(3):263-276. [2] Weaver TE, et al. Sleep. 2007; 30(6):711-719. [3] Weaver TE, et al. *Proc Am Thorac Soc*. 2008;5(2):173-178.

Overview of EDS in OSA

EDS is common among individuals with OSA and can persist despite primary OSA therapy

- OSA results from repeated collapse of the upper airway[1]
- OSA is one of the more common causes of EDS[2]
  - OSA patients feel unrested and sleepy during the day and
  - OSA patients may experience impairments in alertness, cognitive function, and quality of life[3]
- In a retrospective study, the prevalence of EDS before treatment was 87.2% by MSLT[4], and EDS has been shown to persist in 9 – 22% of patients following primary treatment[5-8]
- According to the American Academy of Sleep Medicine diagnostic criteria for OSA and the third edition of the International Classification of Sleep Disorders, a diagnosis of OSA must include positive polysomnography (PSG) findings. In addition, symptoms such as EDS may occur[3,8]
  - Patients may present with a major complaint of EDS in many, but not all, cases[9]
  - The severity of OSA—determined by the number of apnea/hypopnea events and/or the level of oxygen desaturation[8]

[1] Young T, et al. *N Engl J Med*. 1993;328(17):1230-1235. [2] Ramar K, Guilleminault C. *Sleep Med Clin*. 2006;1:63-78. [3] Kapur VK, et al. *J Clin Sleep Med*. 2017;13(3):479-504. [4] Seneviratne U, Puvanendran K. Sleep Med. 2004;5(4):339-343. [5] Gasa M, et al. J Sleep Res. 2013; 22(4):389-397. [6] Koutsourelakis I, et al. Eur Respir J. 2009;34(3):687-693. [7] Weaver TE, et al. Sleep. 2007;30(6):711-719. [8] Pepin J-L, et al. *Eur Respir J.* 2009;33:1062-1067 [9] American Academy of Sleep Medicine. The International Classification of Sleep Disorders – Third Edition (ICSD-3). 2014.

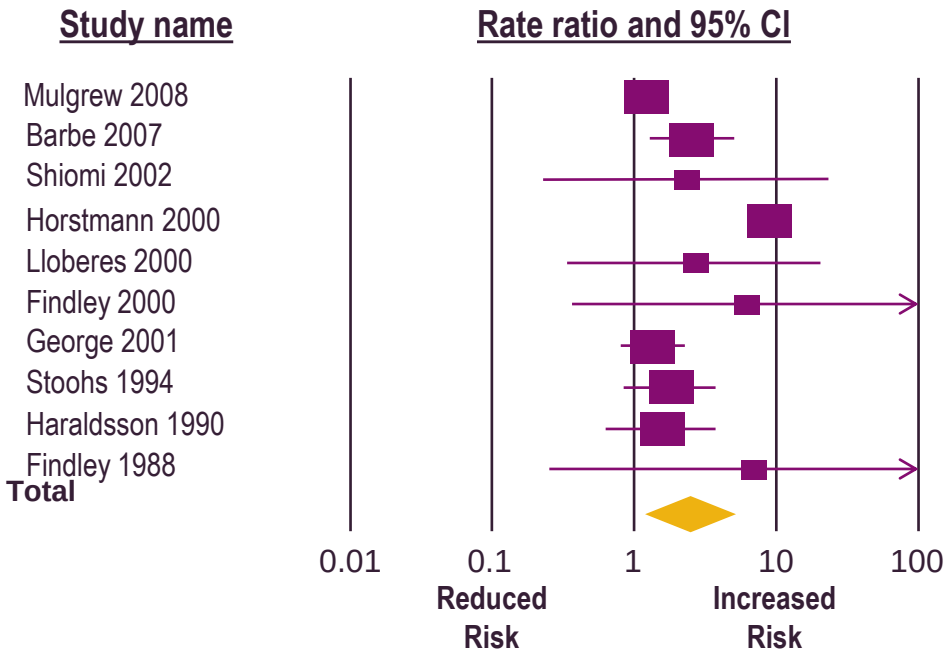
Diagnostic Challenges of EDS in OSA

- EDS is one of the most common symptoms of OSA[1,2]
- Patients do not always present with EDS as a symptom; they may be more sleepy as rated by objective measures compared with self-reported measures[1,3]
- At any level of CPAP compliance, the percentage of EDS is lower based upon subjective patient-reported results from the ESS compared with objective results from the MSLT, suggesting patient underrecognition[4]
- EDS may be difficult to distinguish from fatigue[5]
- Caffeine intake in OSA patients is higher than in participants without OSA[6]
- Current objective diagnostic tools, such as the MSLT and MWT, are not practical as screening tools or for monitoring treatment[1]
- While subjective measures, such as the ESS, are inexpensive and convenient, they may not fully capture the impairment of the sleepy patient,[1] and are only weakly correlated with measures from objective tests, such as the MSLT[2]

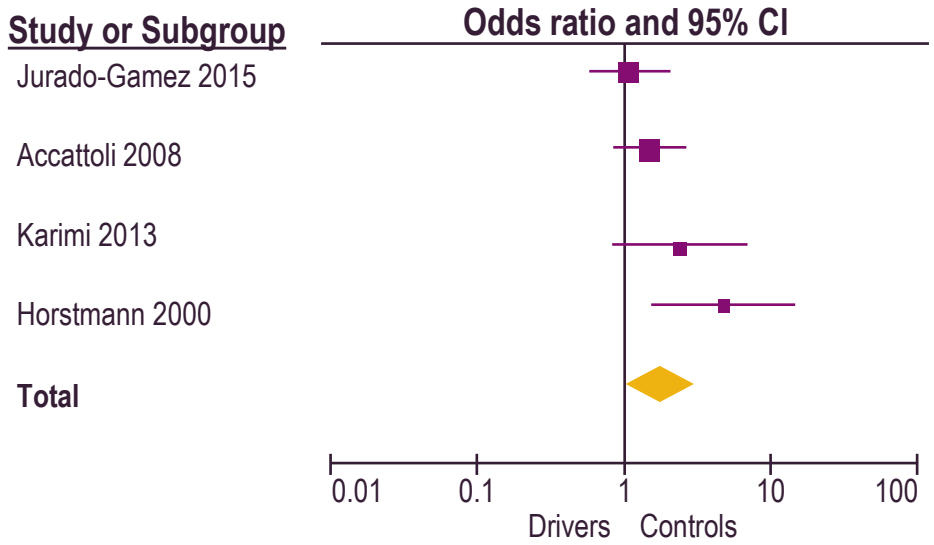
[1] Pagel JF. *Am Fam Physician*. 2009;79(5):391-396. [2] Dongol EM, Williams EJ. *Curr Opin Pulm Med*. 2016;22(6):589-594. [3] Ramar K, Guilleminault C. *Sleep Med Clin*. 2006;1:63-78. [4] Weaver TE, et al. *Sleep*.2007;30(6):711-719. [5] Ruggles K, Hausman M. *Wisc Med J*. 2003;102(1):21-24. [6] Bardwell WA, et al. *J Sleep Res*. 2000;9(3):269-272.

Meta-Analysis on Risk of MVA and Work-Related Accidents/Injuries in OSA Patients

- Controlled studies assessing crash risk suggested OSA patients were 2.43 times more likely to experience a MVA than individuals without OSA (*P* = 0.013)[1]
- Guidelines suggest thorough examination of high-risk drivers can improve driving safety[2,3]
- Crash risk was increased among individuals with OSA compared to controls (random-effects meta-analysis)[1]



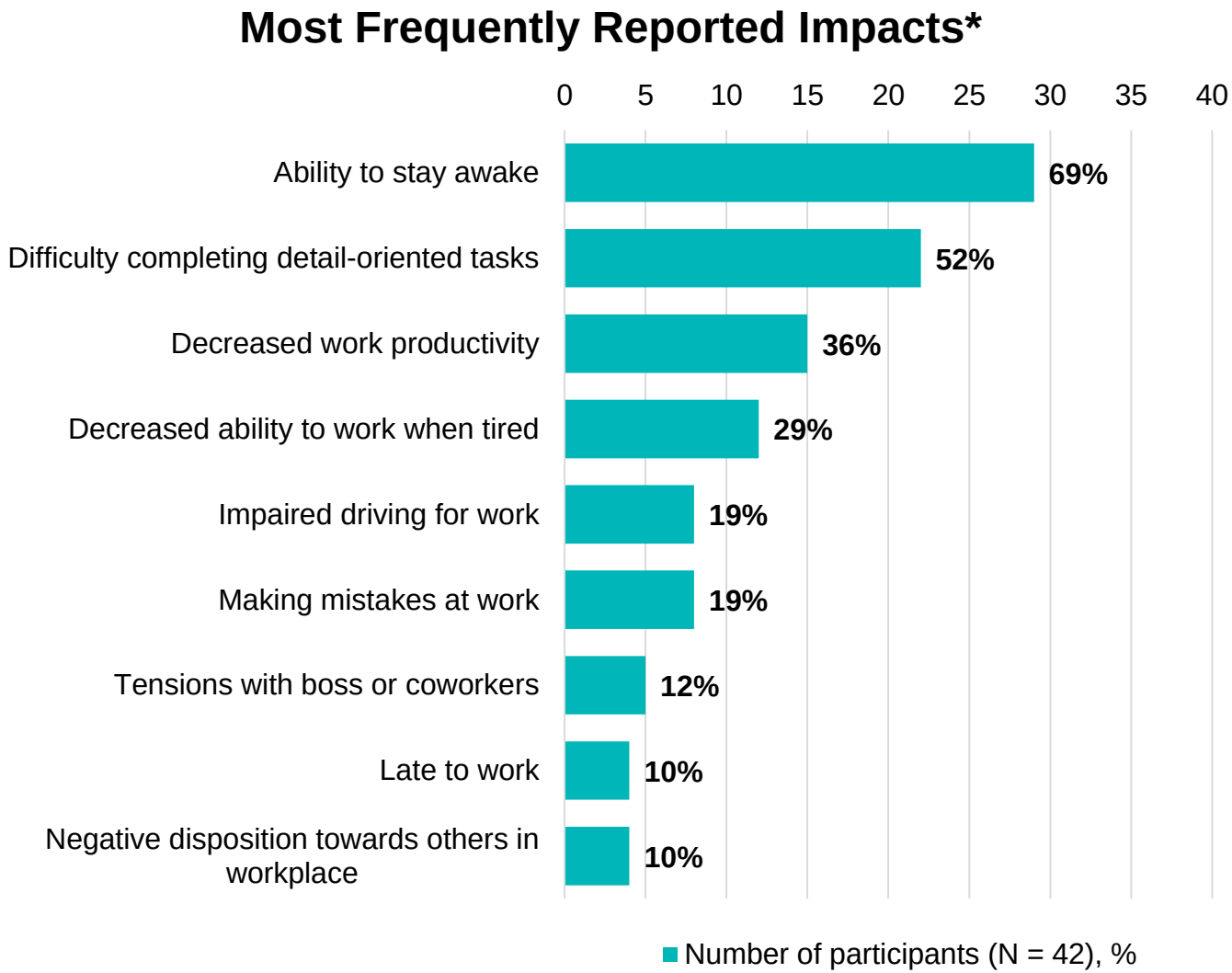
- Studies evaluating the risk of work-related accidents in confirmed OSA patients determined that workers with OSA were 1.78 (95% CI, 1.03-3.07) times more likely to have occupational accidents than workers without OSA[4]
- Workers with various sleep problems, including those with OSA, were 1.62 to 2.23 times more likely to be injured than workers without sleep problems[5,6]
- Occupational driving accidents increased among individuals with OSA compared to controls[4]



[1] Tregear S, et al. *J Clin Sleep Med*. 2009;5(6):573-581. [2] Strohl KP, et al. *Am J Respir Crit Care Med*. 2013;187(11):1259-1266. [3] Colvin LJ, Collop NA. *J Clin Sleep Med*. 2016;12(1):113-125. [4] Garbarino S, et al. *Sleep*. 2016;39(6):1211-1218. [5] Uehli K, et al. *Sleep Med Rev*. 2014;18(1):61-73. [6] Melamed S, Oksenberg A. *Sleep*. 2002;25(3):315-322.

Impacts of EDS Associated With OSA on Work Productivity

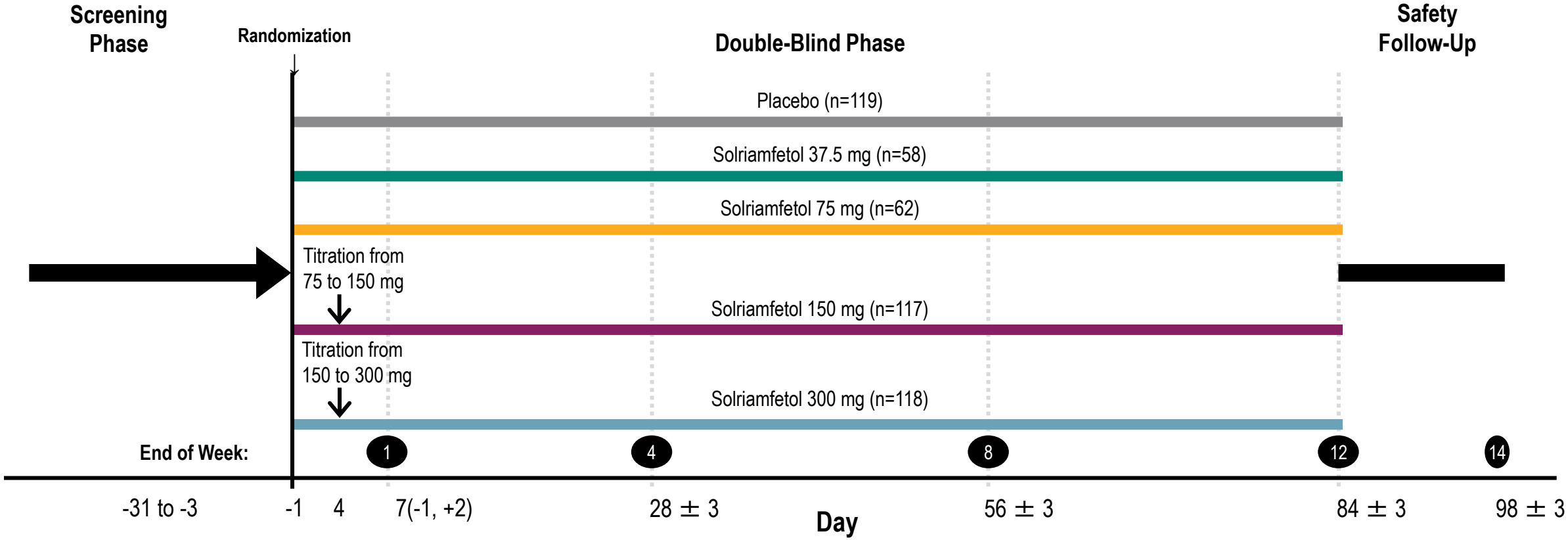
- 42 participants with EDS associated with OSA underwent semi-structured 2-hour focus groups to evaluate how EDS impacts work productivity
- 90% reported current or previous EDS-related work impacts
  - Of those currently experiencing EDS-related work impacts, 55% currently used PAP and 17% currently used an oral device
  - Nearly half of participants that currently experienced work impacts slept an average ≥7 hours per night



Waldman LT, et al. *Sleep* 2018;41:A175
*Impacts reported by ≥10% of participants

TONES 3 CLINICAL DATA

TONES 3[1]: A 12 Week Randomized, Placebo-controlled, Double-blind, Parallel-group Study to Assess Efficacy and Safety of Solriamfetol (OSA)



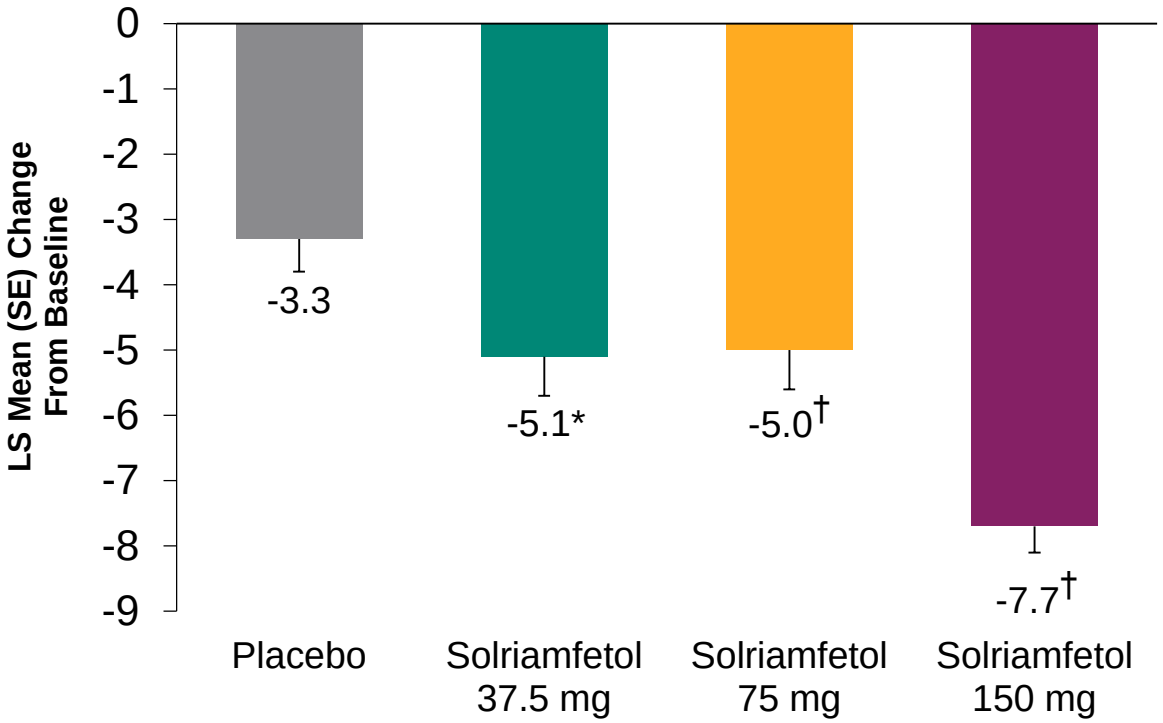
- Co-primary endpoints
  - Change from baseline to week 12 in mean sleep latency on 40-minute MWT
  - Change from baseline to week 12 in mean ESS score
- Key secondary endpoint
  - Percentage of patients reporting improvement on the Patient Global Impression of Change (PGI-C)[1] at week 12

[1] Schweitzer PK, et al. *Am J Respir Crit Care Med.* 2018 Dec 6. doi: 10.1164/rccm.201806-1100OC. [Epub ahead of print].

Note: numbers indicate safety population.

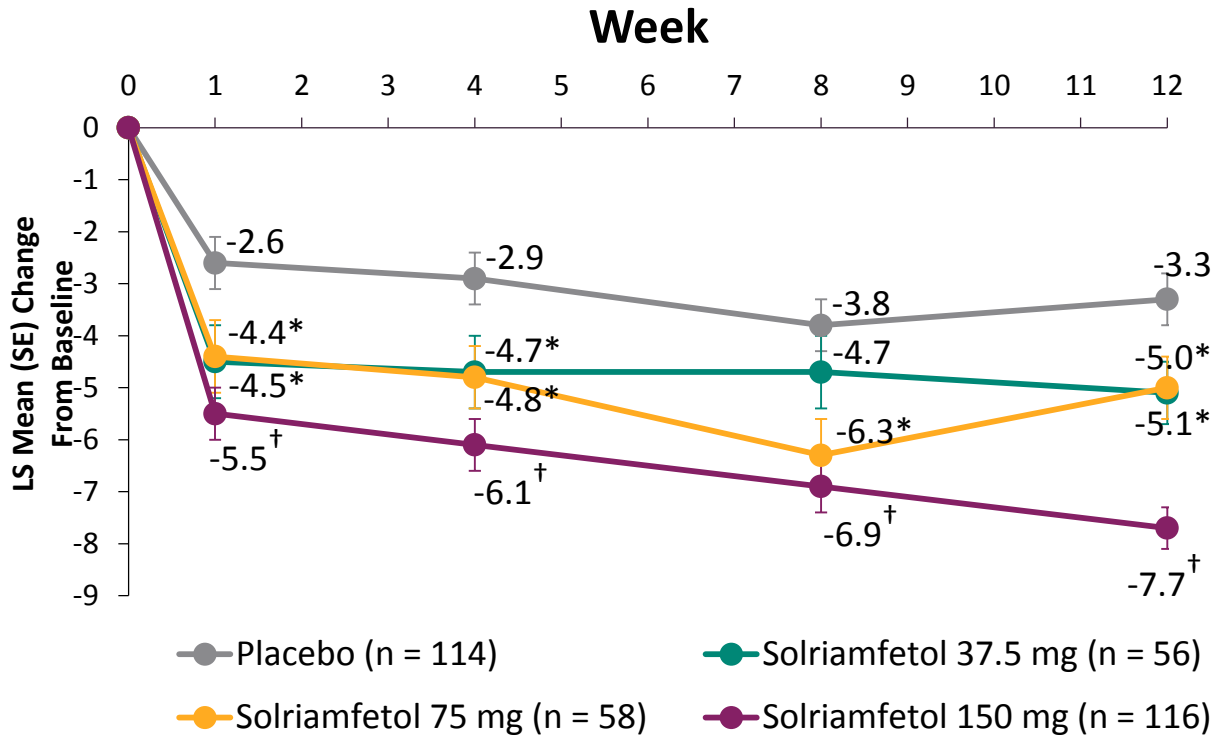
ESS Score

Significant improvement from Baseline to Week 12 in all solriamfetol treatment groups (mITT Population)[1]



Sample size: placebo, n=102; solriamfetol 37.5 mg, n=49; 75 mg, n=54; 150 mg, n=106; 300 mg, n=94. Negative change from baseline indicates improvement.

Improvement observed from Week 1 through Week 12 with solriamfetol 150 mg (mITT Population)[1]

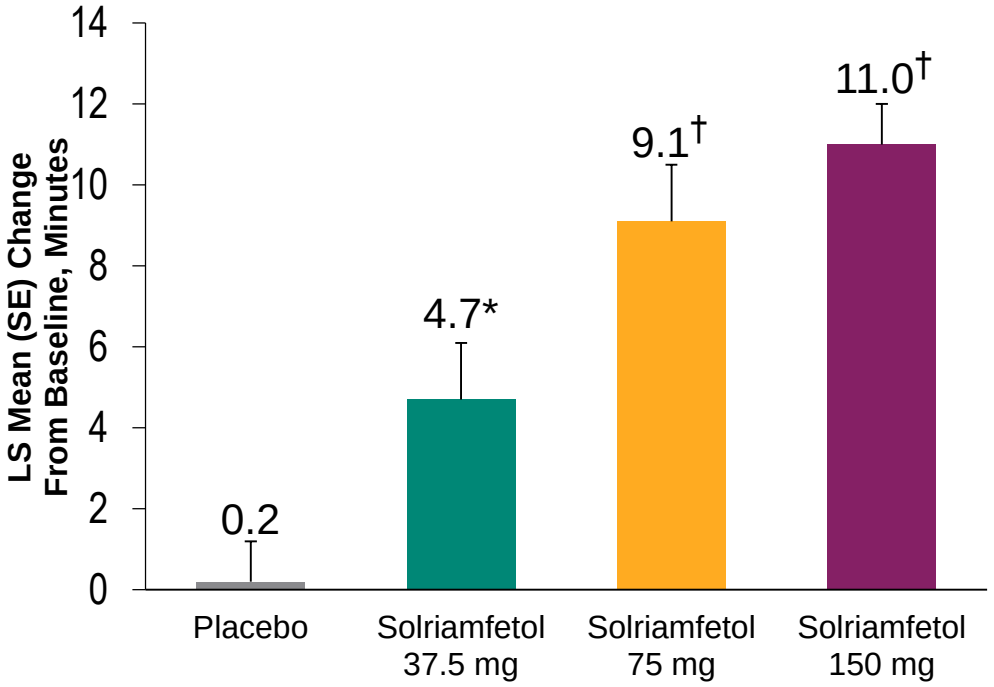


Values are for the modified intent-to-treat population (n=344).

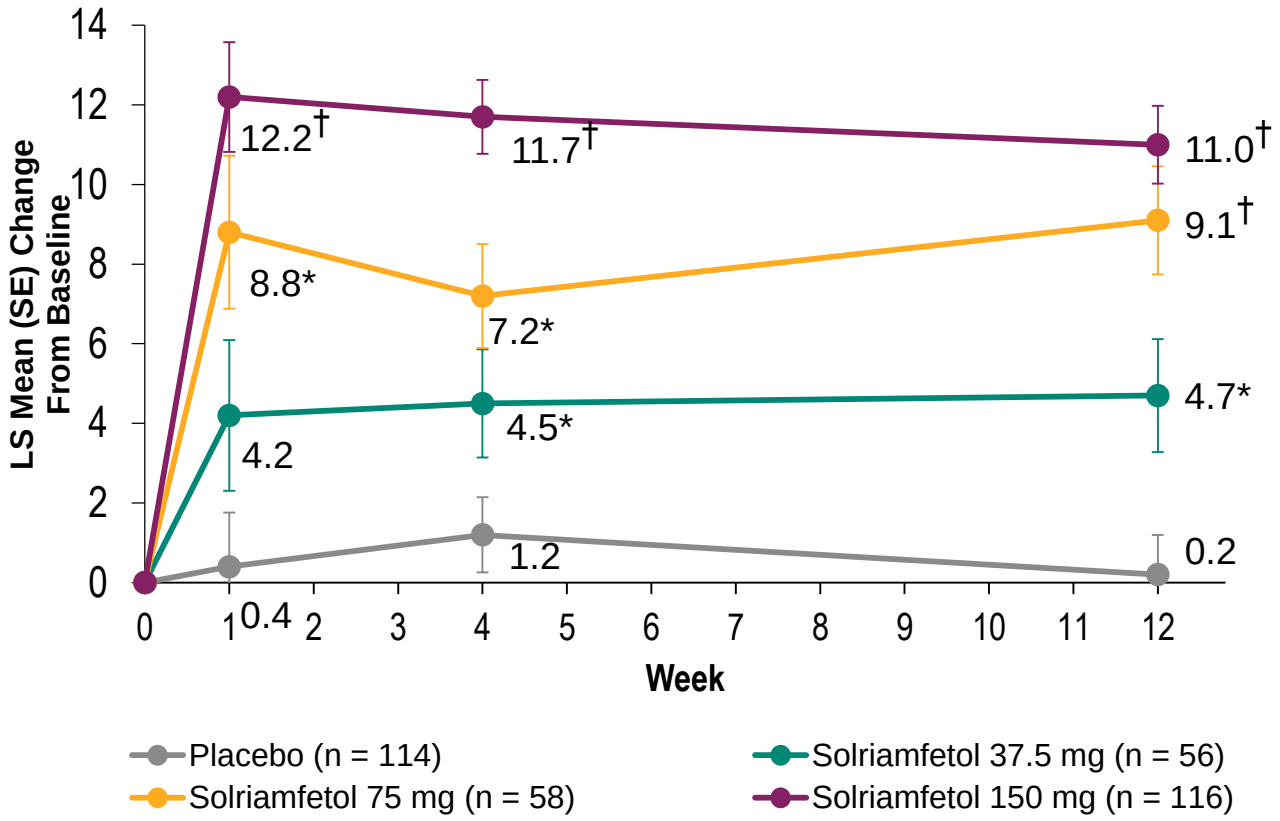
[1] Schweitzer PK, et al. *Am J Respir Crit Care Med.* 2018 Dec 6. doi: 10.1164/rccm.201806-1100OC. [Epub ahead of print]. [2]300 mg data not shown
*$P < 0.05$ and †$P < 0.0001$ vs placebo

Mean Sleep Latency on MWT

Significant improvement from Baseline at Week 12 in solriamfetol treatment groups (mITT Population)[1]



Improvement was observed from Week 1 through Week 12 with solriamfetol 75 and 150 mg (mITT Population)[1]



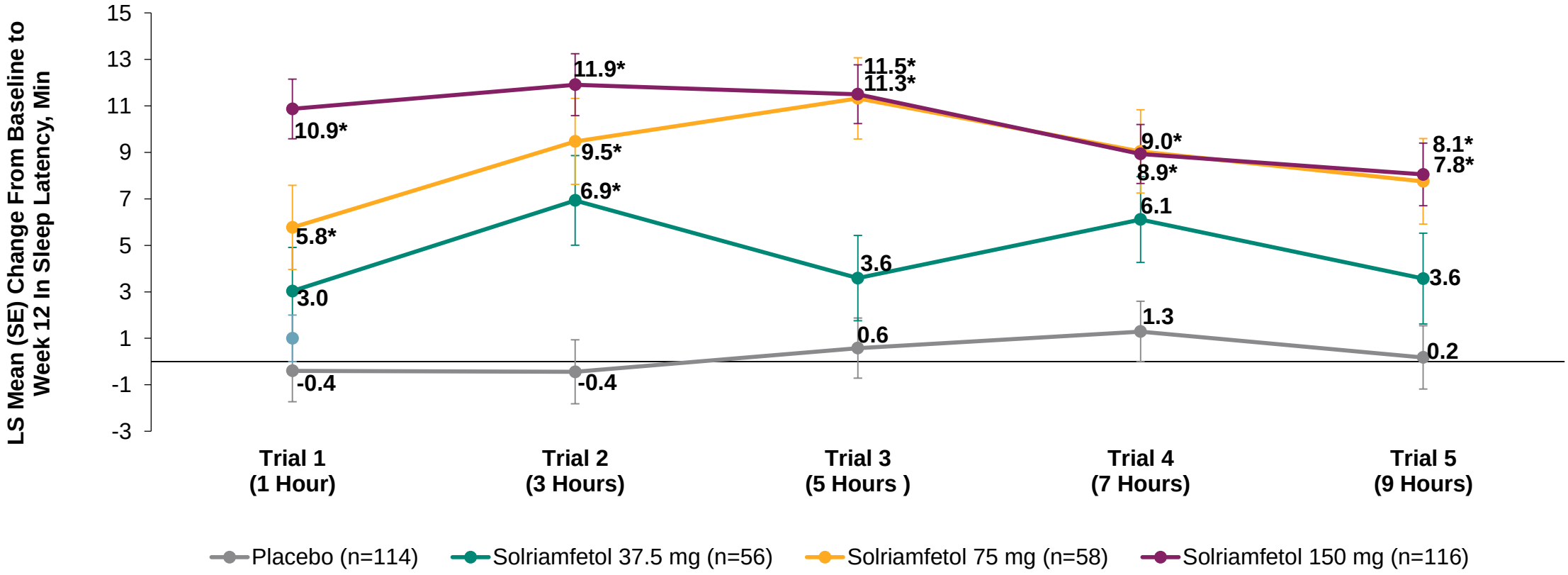
Values are for the modified intent-to-treat population (n=344).

[1] Schweitzer PK, et al. *Am J Respir Crit Care Med.* 2018 Dec 6. doi: 10.1164/rccm.201806-1100OC. [Epub ahead of print]. [2]300 mg data not shown

*$P < 0.05$ and †$P < 0.0001$ vs placebo

MWT Across the Day[1]

Solriamfetol at 75 and 150 mg Significantly Increased Sleep Latency on MWT from Trial 1 Over 9 Hours after Single Morning Dose[2]

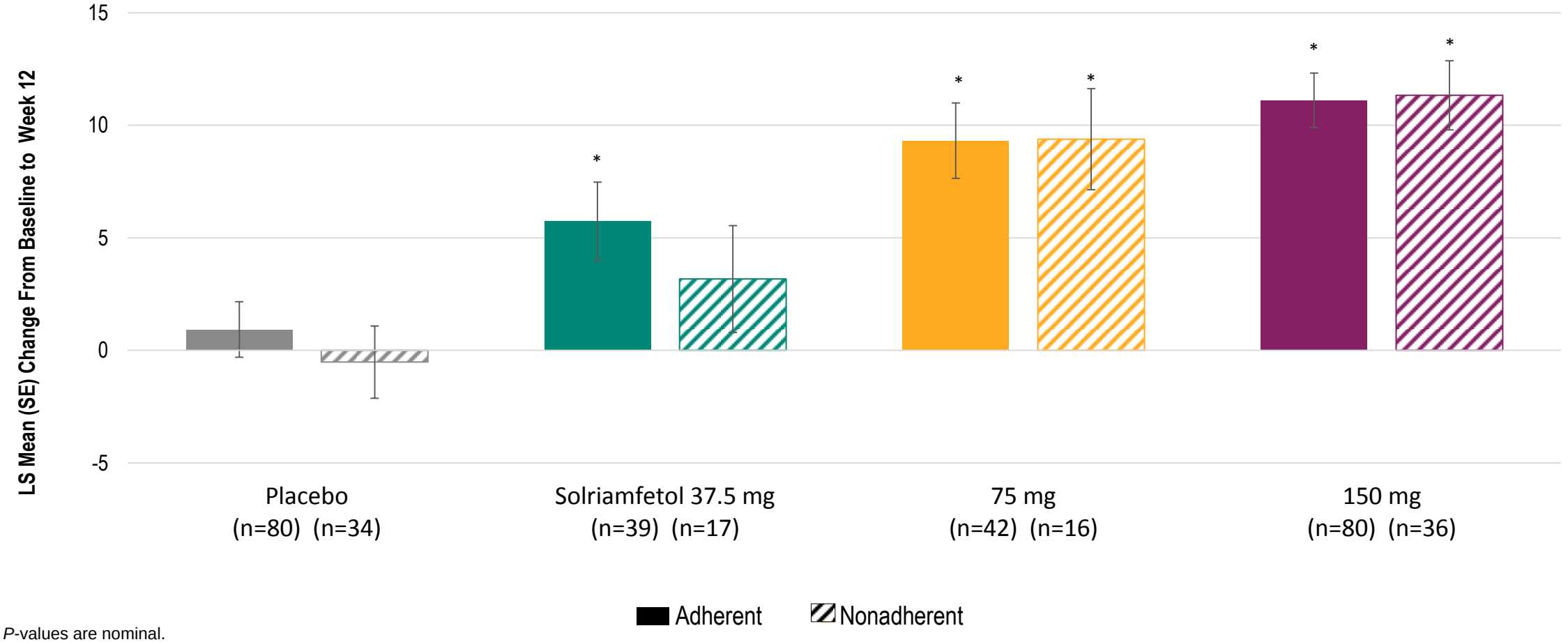


[1] Schweitzer PK, et al. *Am J Respir Crit Care Med.* 2018 Dec 6. doi: 10.1164/rccm.201806-1100OC. [Epub ahead of print]. [2] 300 mg data not shown

*$P < 0.05$

CPAP Adherence vs. Nonadherence[1]: MWT

Compared to Placebo, Solriamfetol Produced Similar Improvement in Mean Sleep Latency on MWT Regardless of Adherence to Primary OSA therapy (mITT population)[2,3,4]



*P<0.05, P-values are nominal.

[1] Observed in a secondary analysis; [2] Schweitzer PK et al. *Am J Respir Crit Care Med*. 2018; 197:A4396. [3] Definition of compliant/non-compliant use of primary OSA therapy included in Appendix. [4] 300 mg data not shown

TONES 2 and TONES 3: Adverse Events

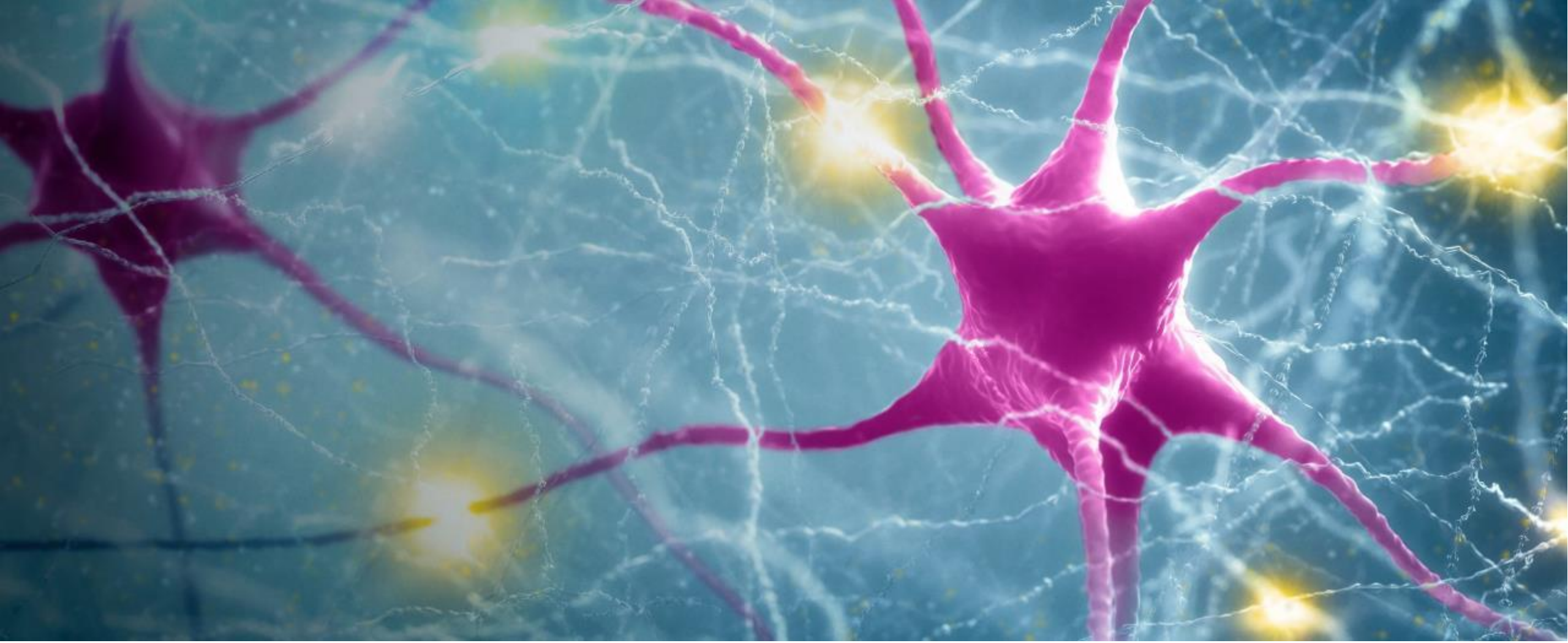
Safety Population includes 300 mg dose per Product Information

Adverse event	Narcolepsy		OSA	
	Placebo (n = 59)	Solriamfetol combined (n = 177)	Placebo (n = 119)	Solriamfetol combined (n = 355)
Any adverse event, n (%)	27 (45.8)	121 (68.4)	57 (47.9)	241 (67.9)
Serious adverse event, n (%)	0	1 (0.6)	2 (1.7)	3 (0.8)
Adverse event leading to discontinuation, n (%)	1 (1.7)	9 (5.1)	4 (3.4)	26 (7.3)
Most common adverse events,[1] n (%)				
Headache	3 (5.1)	38 (21.5)	10 (8.4)	36 (10.1)
Nausea	1 (1.7)	19 (10.7)	7 (5.9)	28 (7.9)
Decreased appetite	1 (1.7)	19 (10.7)	1 (0.8)	27 (7.6)
Nasopharyngitis	3 (5.1)	16 (9.0)	8 (6.7)	18 (5.1)
Dry mouth	2 (3.4)	13 (7.3)	2 (1.7)	16 (4.5)
Anxiety	1 (1.7)	9 (5.1)	0	25 (7.0)

- Similar safety profile in narcolepsy and OSA and consistent with previous solriamfetol studies

[1] Reported in ≥5% of participants in combined solriamfetol group for both narcolepsy and OSA.

Rosenberg R, et al. Presented at: Annual Meeting of the AMCP Nexus; October 22-25, 2018; Orlando, FL.



Sunosi Label Overview and Product Development

Lawrence Carter, Ph.D.
Executive Director, Global Development Lead, Solriamfetol, Jazz Pharmaceuticals

Jazz Pharmaceuticals

Sunosi™ (solriamfetol) tablets for oral use, C-IV

Approved March 20, 2019

SUNOSI is a dopamine and norepinephrine reuptake inhibitor (DNRI) indicated to improve wakefulness in adult patients with excessive daytime sleepiness associated with narcolepsy or obstructive sleep apnea (OSA).

Dosage in Narcolepsy
Initiate SUNOSI at 75 mg once daily in adults with narcolepsy. The recommended dose range for SUNOSI is 75 mg to 150 mg once daily. Based on efficacy and tolerability, the dosage of SUNOSI may be doubled at intervals of at least 3 days. The maximum recommended dose is 150 mg once daily. Dosages above 150 mg daily do not confer increased effectiveness sufficient to outweigh dose-related adverse reactions.

Dosage in OSA
Initiate SUNOSI at 37.5 mg once daily in adults with OSA. The recommended dosage range for SUNOSI is 37.5 mg to 150 mg once daily. Based on efficacy and tolerability, the dosage of SUNOSI may be doubled at intervals of at least 3 days. The maximum recommended dosage is 150 mg once daily. Dosages above 150 mg daily do not confer increased effectiveness sufficient to outweigh dose-related adverse reactions.

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Sunosi™ (solriamfetol) tablets for oral use, C-IV

SUNOSI contains solriamfetol, a Schedule IV controlled substance. SUNOSI has potential for abuse.
Physicians should carefully evaluate patients for a recent history of drug abuse, especially those with a history of stimulant (e.g., methylphenidate, amphetamine, or cocaine) or alcohol abuse, and follow such patients closely, observing them for signs of misuse or abuse of SUNOSI (e.g., incrementation of doses, drug-seeking behavior).

Limitations of Use
SUNOSI is not indicated to treat the underlying airway obstruction in OSA. Ensure that the underlying airway obstruction is treated (e.g., with CPAP) for at least one month prior to initiating SUNOSI for excessive daytime sleepiness. Modalities to treat the underlying airway obstruction should be continued during treatment with SUNOSI. SUNOSI is not a substitute for these modalities.

Contraindications
SUNOSI is contraindicated in patients receiving concomitant treatment with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitor, because of the risk of hypertensive reaction.

Jazz Pharmaceuticals

Sunosi™ (solriamfetol) tablets for oral use, C-IV

Warnings and Precautions

Blood Pressure and Heart Rate Increases

- SUNOSI increases systolic blood pressure, diastolic blood pressure, and heart rate in a dose-dependent fashion. Epidemiological data show that chronic elevations in blood pressure increase the risk of major adverse cardiovascular events (MACE), including stroke, heart attack, and cardiovascular death. The magnitude of the increase in absolute risk is dependent on the increase in blood pressure and the underlying risk of MACE in the population being treated. Many patients with narcolepsy and OSA have multiple risk factors for MACE, including hypertension, diabetes, hyperlipidemia, and high body mass index (BMI).
- Assess blood pressure and control hypertension before initiating treatment with SUNOSI. Monitor blood pressure regularly during treatment and treat new-onset hypertension and exacerbations of pre-existing hypertension. Exercise caution when treating patients at higher risk of MACE, particularly patients with known cardiovascular and cerebrovascular disease, pre-existing hypertension, and patients with advanced age. Use caution with other drugs that increase blood pressure and heart rate.
- Periodically reassess the need for continued treatment with SUNOSI. If a patient experiences increases in blood pressure or heart rate that cannot be managed with dose reduction of SUNOSI or other appropriate medical intervention, consider discontinuation of SUNOSI.
- Patients with moderate or severe renal impairment could be at a higher risk of increases in blood pressure and heart rate because of the prolonged half-life of SUNOSI.

Jazz Pharmaceuticals

Sunosi™ (solriamfetol) tablets for oral use, C-IV

Warnings and Precautions

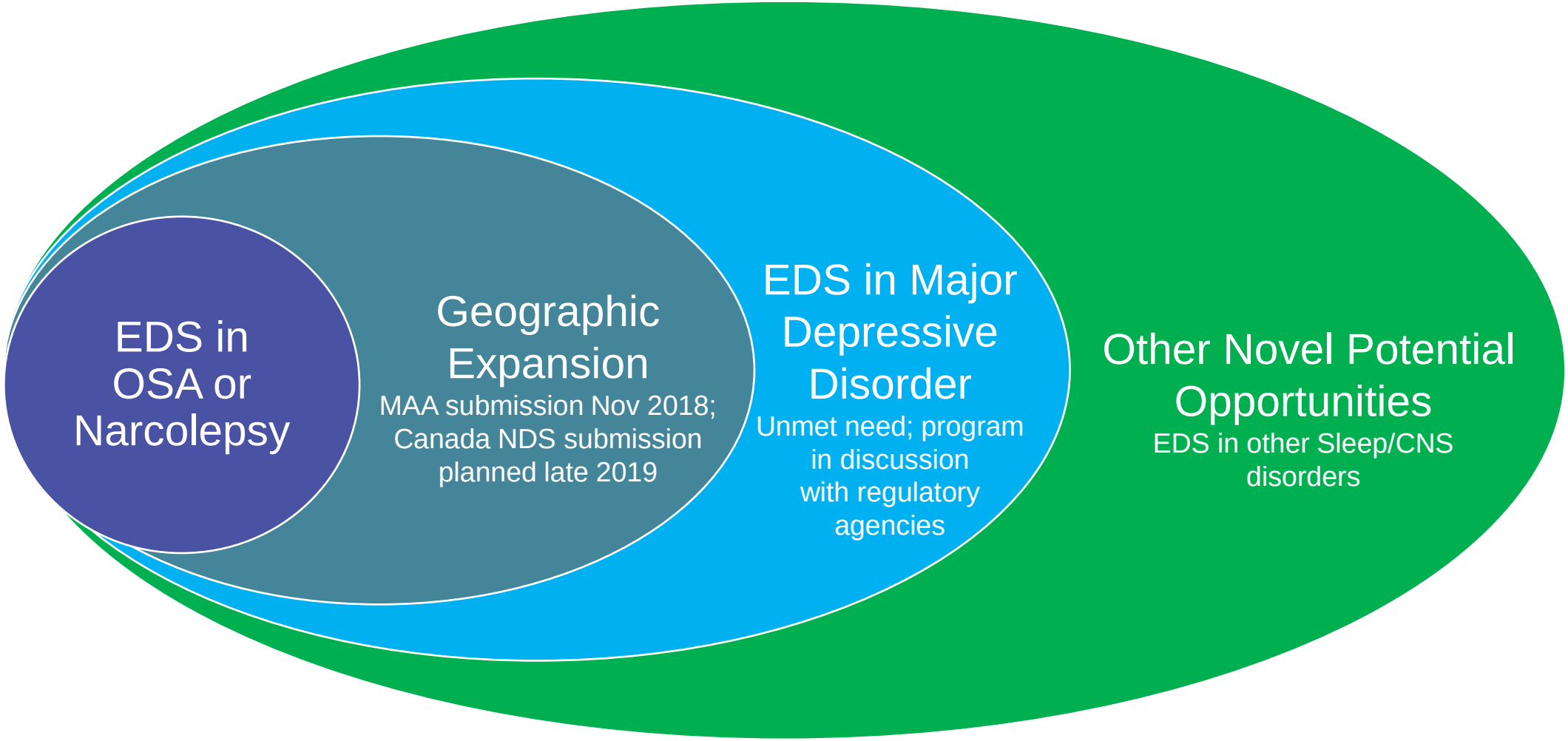
Psychiatric Symptoms

- Psychiatric adverse reactions have been observed in clinical trials with SUNOSI, including anxiety, insomnia, and irritability.
- Exercise caution when treating patients with SUNOSI who have a history of psychosis or bipolar disorders, as SUNOSI has not been evaluated in these patients.
- Patients with moderate or severe renal impairment may be at a higher risk of psychiatric symptoms because of the prolonged half-life of SUNOSI.
- Observe SUNOSI patients for the possible emergence or exacerbation of psychiatric symptoms. Consider dose reduction or discontinuation of SUNOSI if psychiatric symptoms develop.

Jazz Pharmaceuticals

Solriamfetol: Opportunities Being Explored Beyond Narcolepsy & OSA

Excessive Daytime Sleepiness Can Be Debilitating in Multiple Conditions



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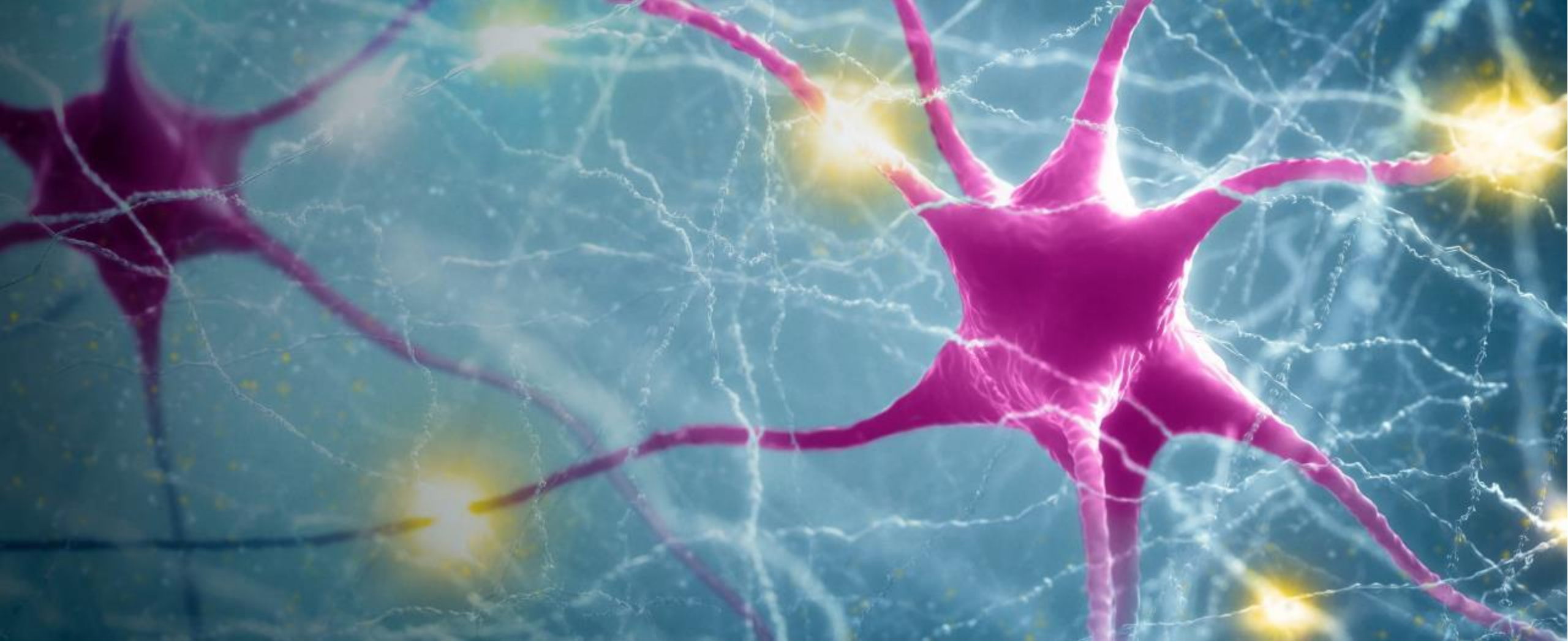
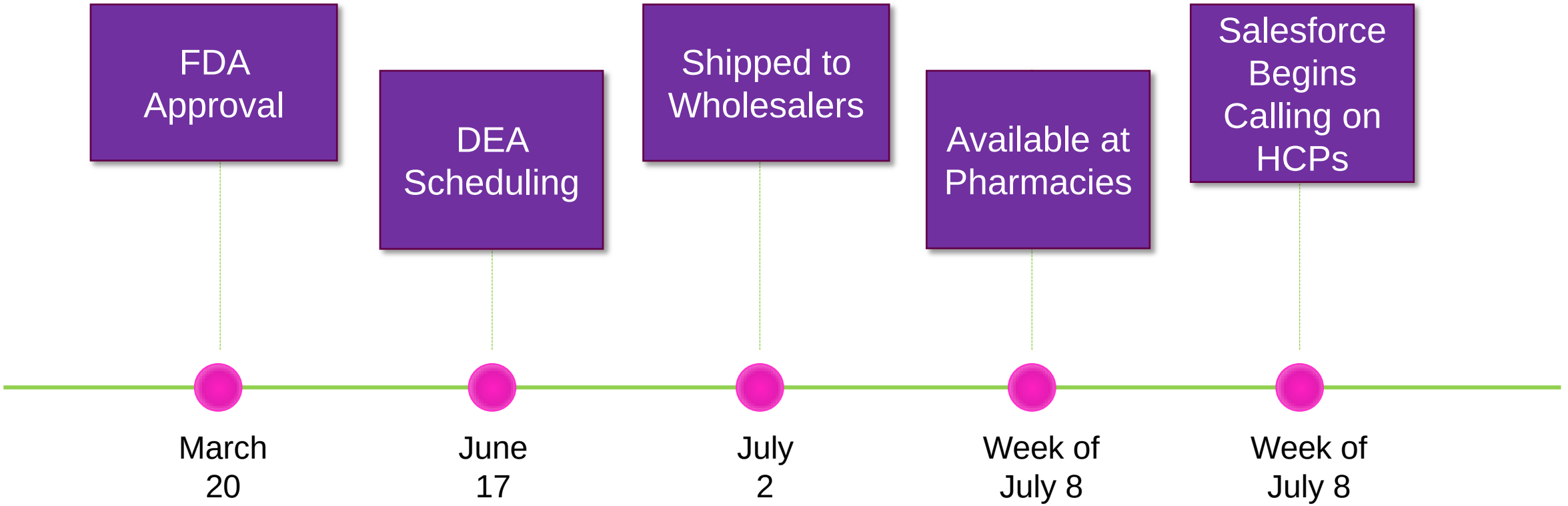


Launch Initiatives

Mike Miller
Executive Vice President, U.S. Commercial

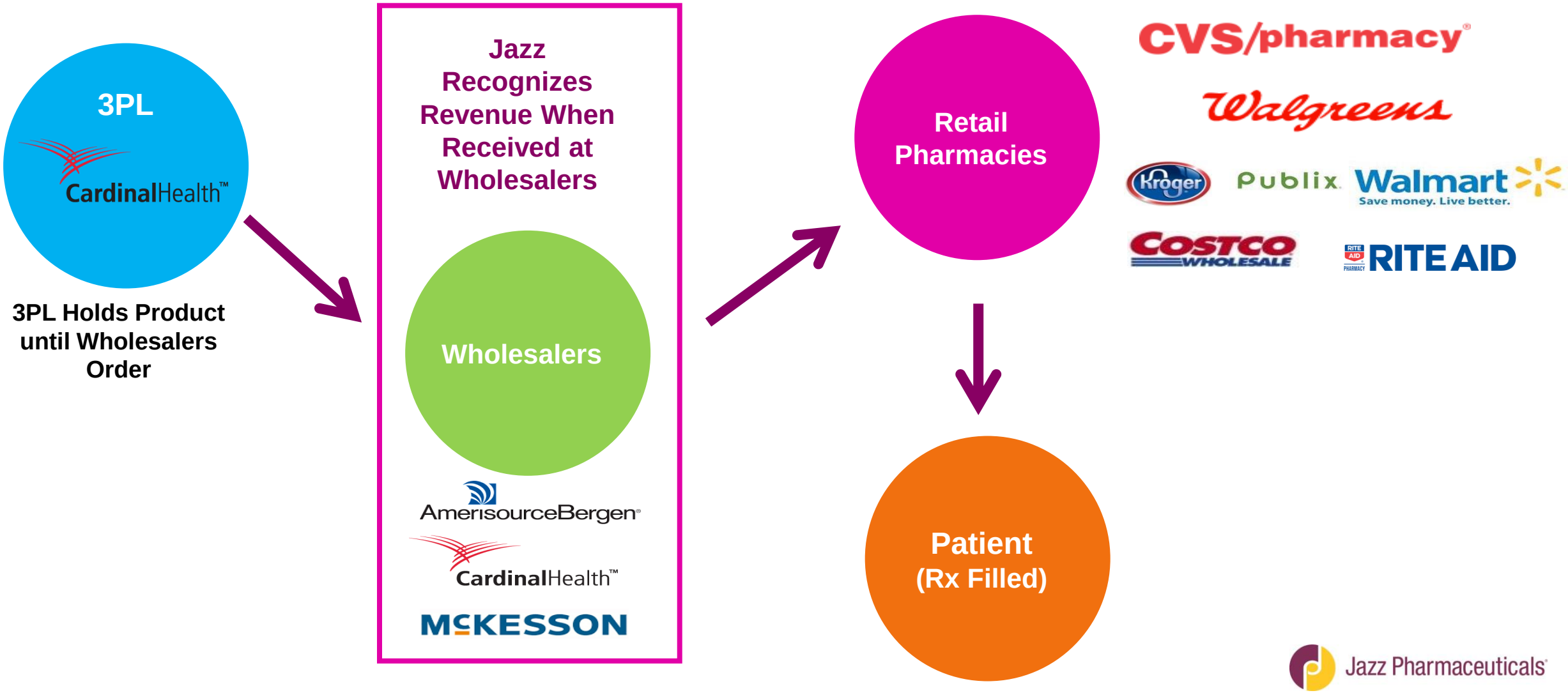
Sunosi Commercially Available in U.S. Week of July 8, 2019

DEA scheduling June 17 | Product labeling/packaging completed in June | Sunosi shipped to wholesalers in July



From Manufacturer to Patient

SUNOSI™ (solriamfetol) IV



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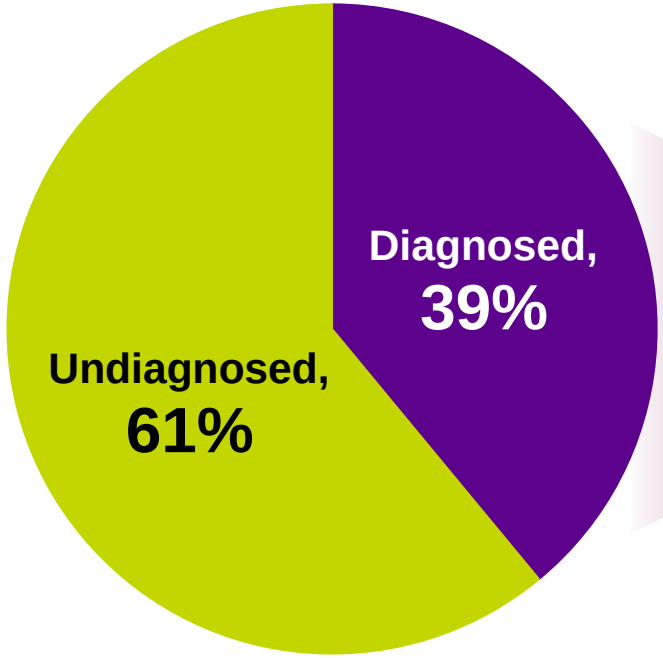
Narcolepsy Opportunity
Orphan Condition Remains Largely Under Diagnosed

165,000 patients with narcolepsy



Majority of narcolepsy patients diagnosed and drug-treated are treated with stimulants or WPAs

Source: SHA Claims Data Jul 2010 to Mar 2015

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OSA Opportunity

Larger Population But Very Low Drug Treatment Rate

~12M diagnosed; 6% drug treatment rate

Source: Won C, Hess G, Acquavella J, Bron M, Bujanover S, Villa KF. Poster presented at Annual Meeting of the Academy of Manage Care Pharmacy, October 2018.

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Launch Initiatives

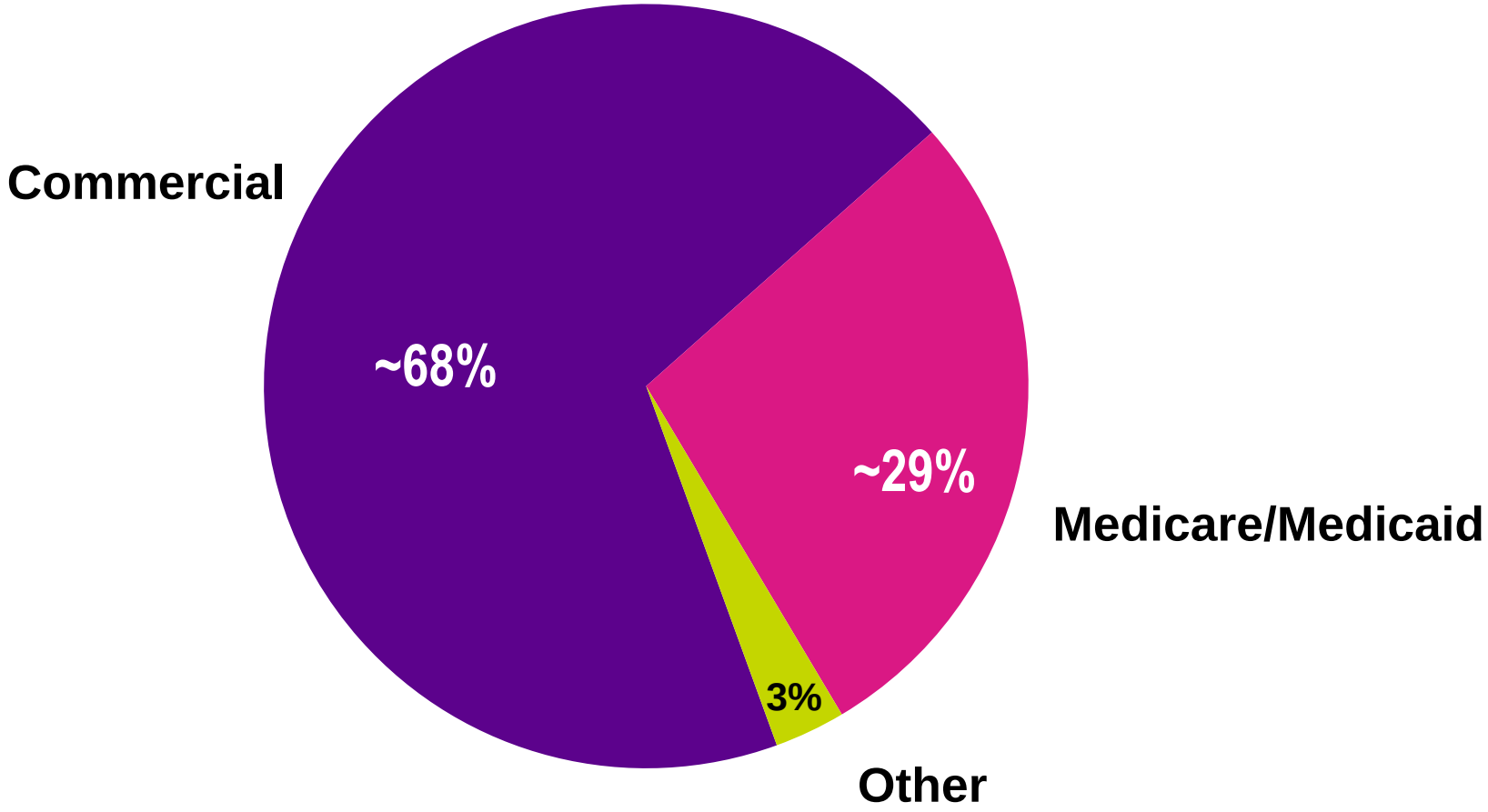
Educate Healthcare Providers

Empower Patients

Enable Patient Access

Stimulant/Wake-Promoting Agent Payer Landscape

Dominant Payer Type is Commercial



Source: SHA Diagnosis and Pharmacy claims for 1Q16-4Q17

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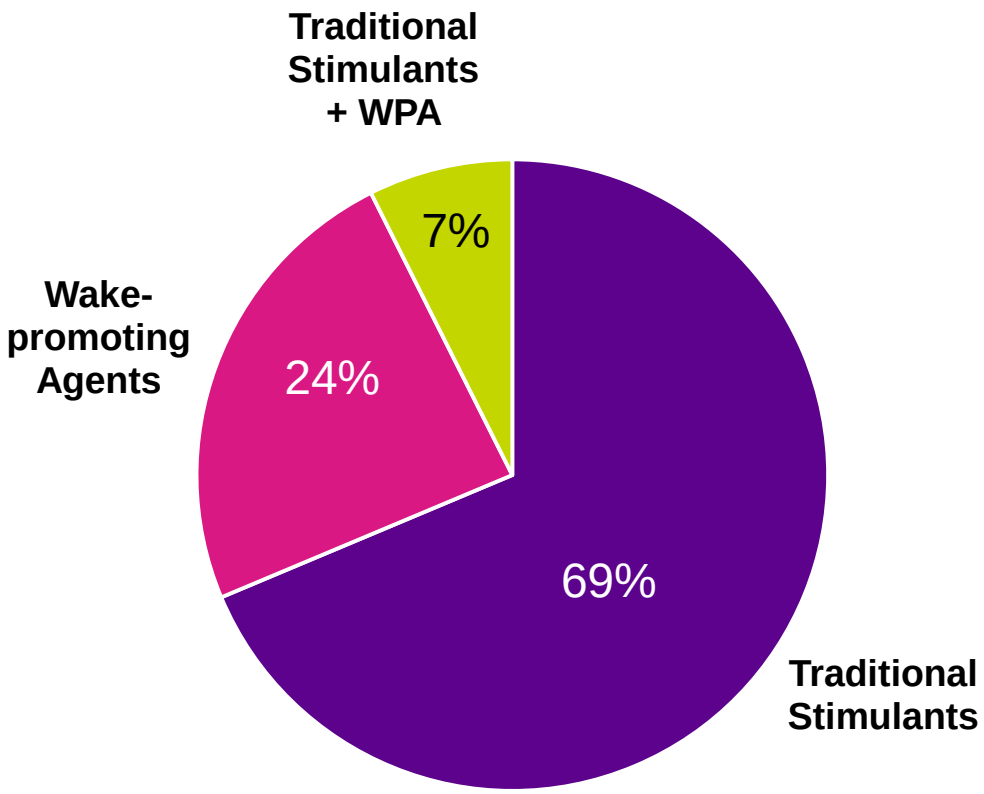
Traditional Stimulants Are the Most Commonly Prescribed OSA Medication

Percentage of OSA Patients on Class of TS/WPA Treatment



Generic Name	FDA Approval Status	Schedule
Wake Promoting Agents		
Modafinil	Approved for ES in OSA, Narcolepsy, Shift Work Disorder	IV
Armodafinil		
Traditional Stimulants		
Amphetamine salts/ Dextroamphetamine	Approved for Narcolepsy without any study data (grandfathered)	II
Methylphenidate		II
Methamphetamine	Approved for ADHD & Obesity	II
Dexmethylphenidate	Approved for ADHD	II
Lisdexamfetamine	Approved for ADHD & Binge Eating Disorder	II

Source: SHA claims data for 60 months from Jan 2014 to Dec 2018. Patients assigned a disease category if they have two diagnosis claims for those diseases in the 60 months of data.
Total WPA / TS patients N = 670,683

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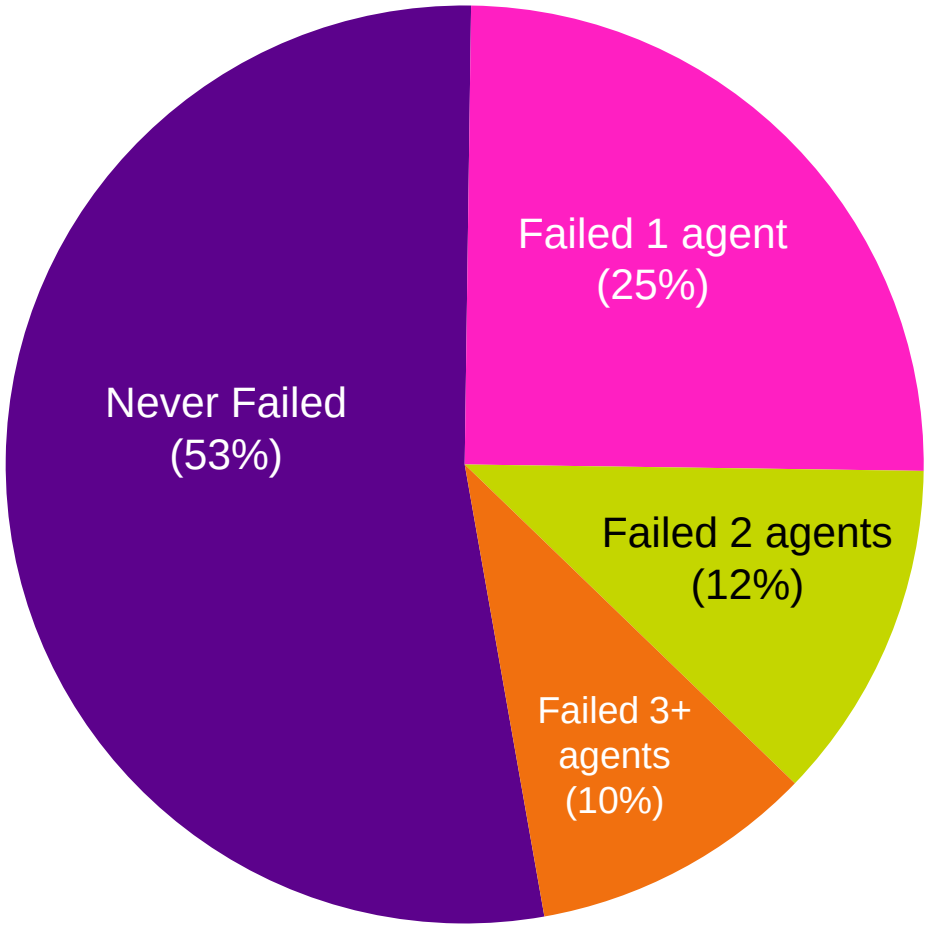
Nearly 50% of OSA Patients with EDS Fail One or More Traditional Stimulants or Wake-Promoting Agents



Source: SHA medical (Dx) claims for 60 months from Jan 2014 to Dec 2018; A maximum grace period of 90 days between successive claims was used to define a patient restart; Regimens of low duration, i.e. < 25 days in length, are not included in the analysis

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Healthcare Provider Disease Education

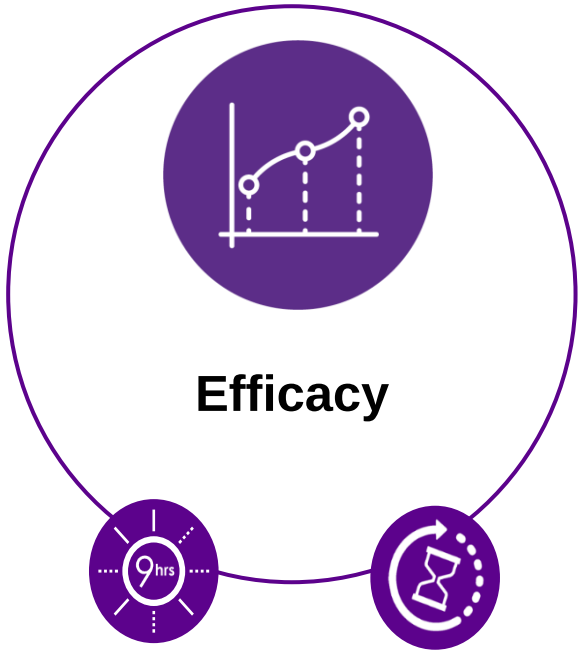
- Educate healthcare providers that EDS in OSA is a serious neurological condition
  - EDS in OSA pathophysiology
  - Burden of EDS on patients
  - Need for Rx therapy for EDS
- EDS Screening Toolkit
- Robust Congress Presence
  - ATS, SLEEP, U.S. Psych Congress, ANA, CHEST, AAN
- Live National Disease Awareness Broadcasts
- Have reached more than 1,200 clinicians who treat OSA patients through live educational programs at key congresses and virtual broadcasts in 1H19

Consumer Disease Awareness

A DIFFERENT KIND OF tired™

- Raise awareness and urgency for patients suffering from EDS due to OSA
  - EDS related to OSA is a real medical condition
  - Encourage people to self-identify
  - Empower people to take action and speak to their doctor
- In 1Q19, the campaign reached more than 5 million unique individuals

Sunosi Core Messages



Onset of Action, Duration of Effect and Meaningful Improvement In Wakefulness

Long Term Efficacy

EDS in OSA pathophysiology and Sunosi MOA (Inhibits the reuptake of dopamine and norepinephrine)

Schedule IV—low abuse potential

Long-term safety

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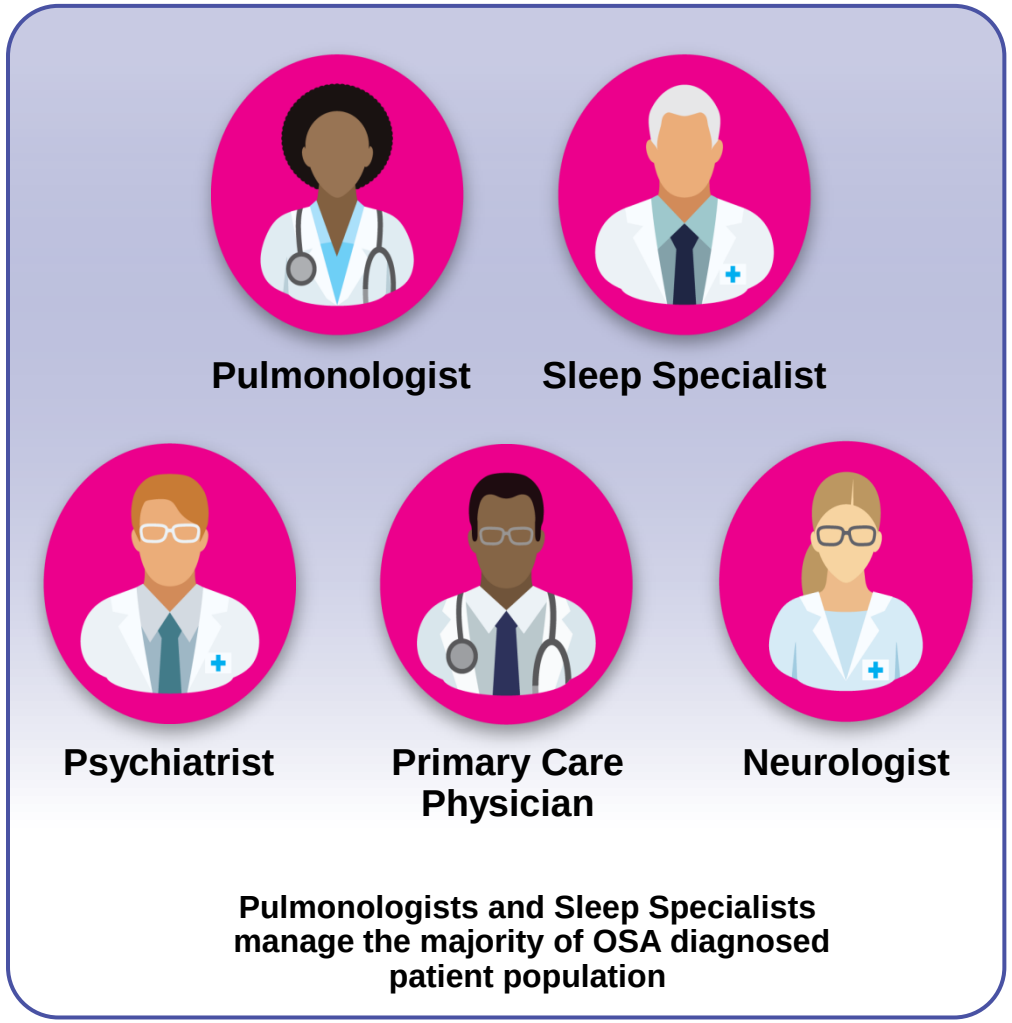
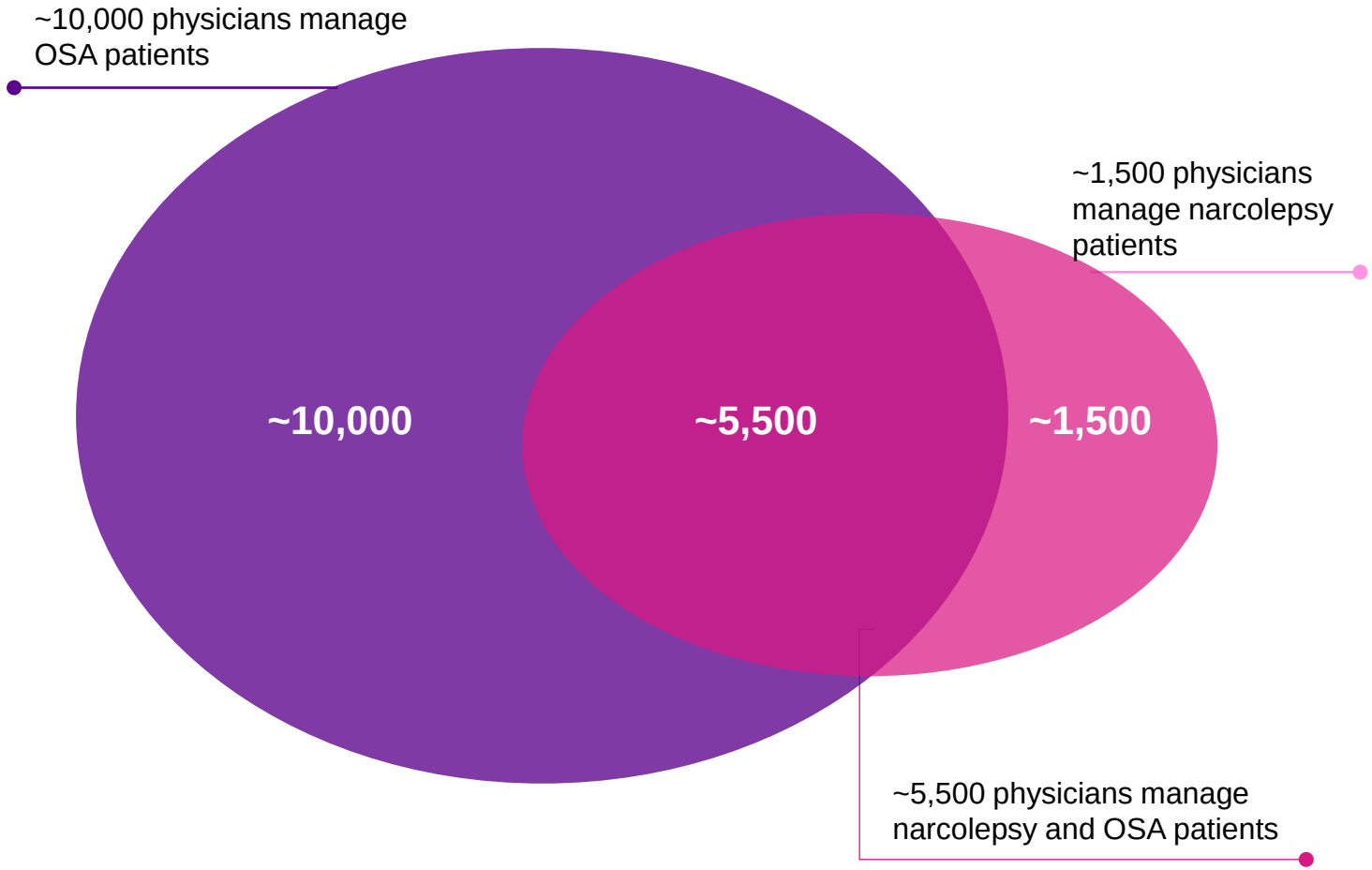
Targeted Prescriber Audience

Opportunity to Leverage Existing Relationships; Sleep Salesforce Expanded from 93 to 143 Territories



Pulmonologists and Sleep Specialists manage the majority of OSA diagnosed patient population



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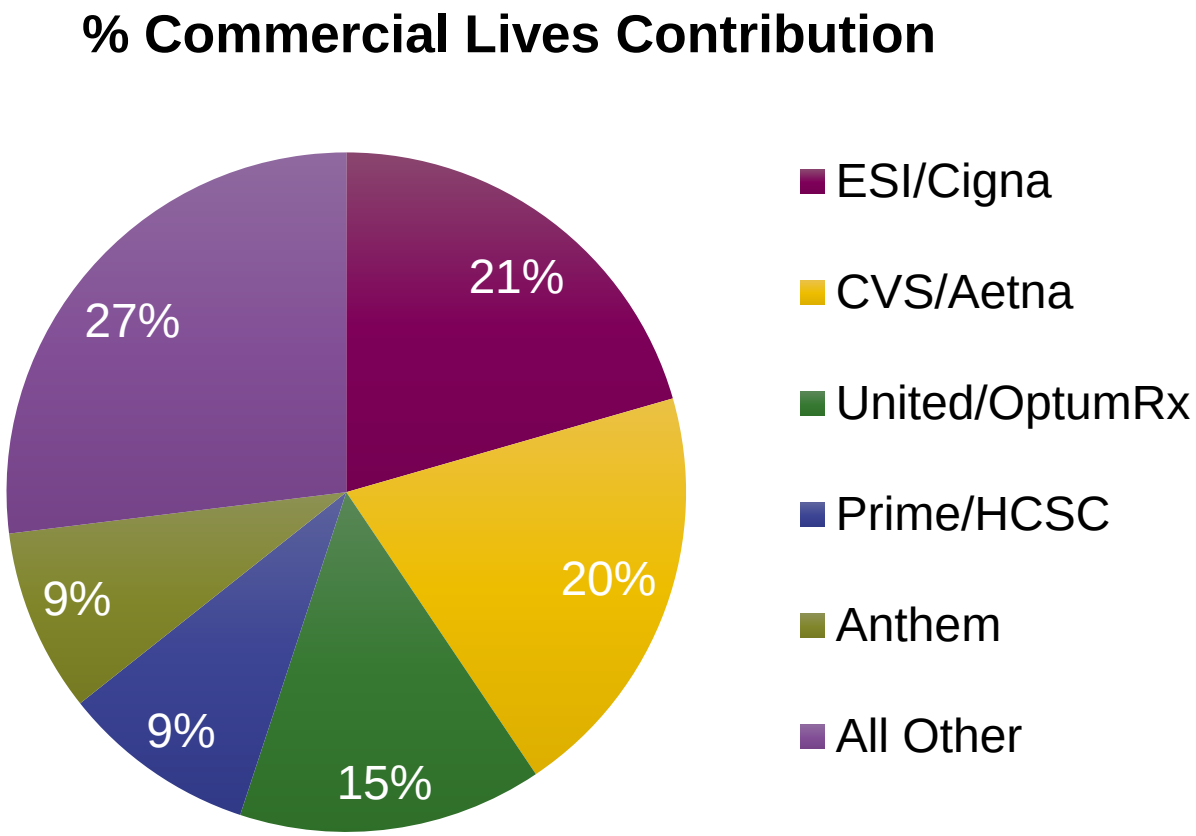
The Top 5 Commercial PBMs and Payers Cover Approximately 75% of Lives



- Marketplace consolidation has resulted in the top 5 commercial PBMs and payers covering approximately 75% of lives
- Most payers and PBMs exclude new retail Rx products from formulary at launch
- Few commercial plans have ‘open’ formularies, limiting initial access to new products

Source: Managed Markets Insights and Technology, LLC (MMIT), data current as of 2Q19

Robust Patient Services Programs at Launch

Focus on Ensuring Commercial Patient Access

Sample Programs

Prior Authorization Support

Savings Programs

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Sunosi Key Performance Indicators
Measurement of Launch Success

Performance

Customer Engagement

Market Access

Source of Business

Customer Awareness

Market Pulse

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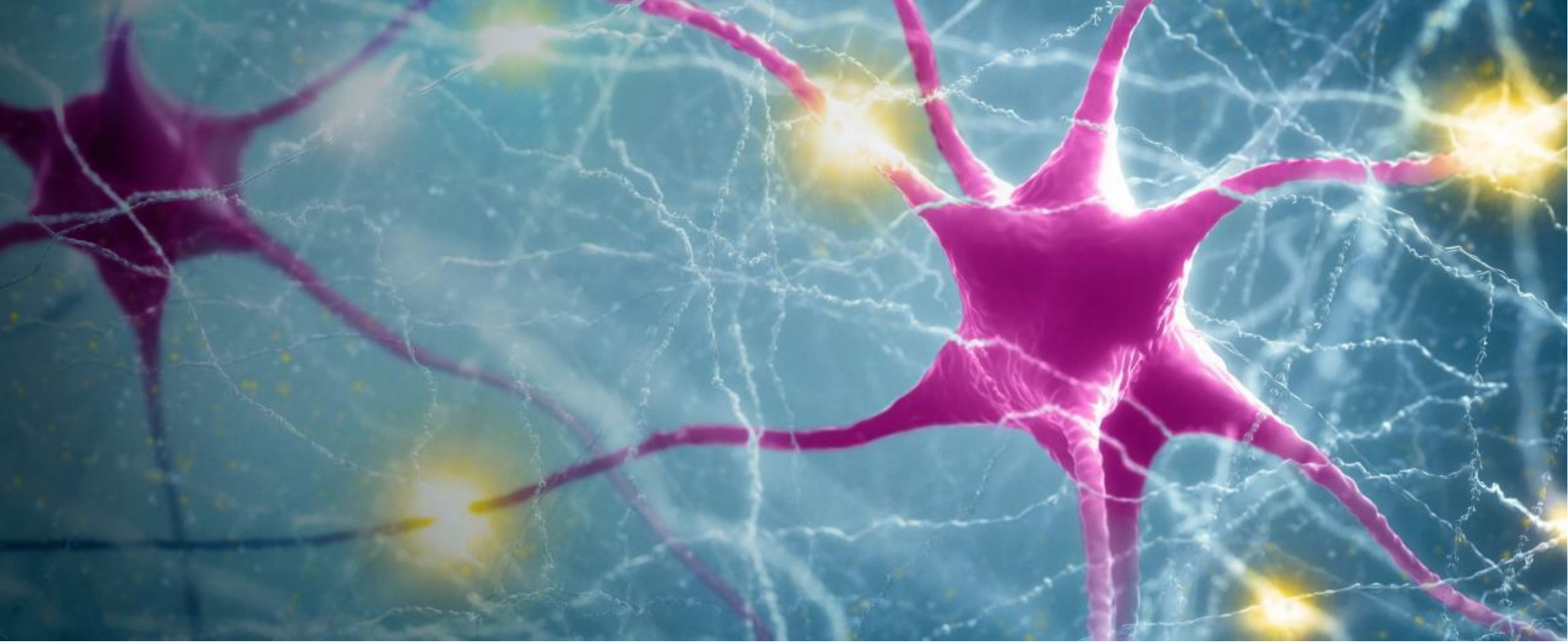


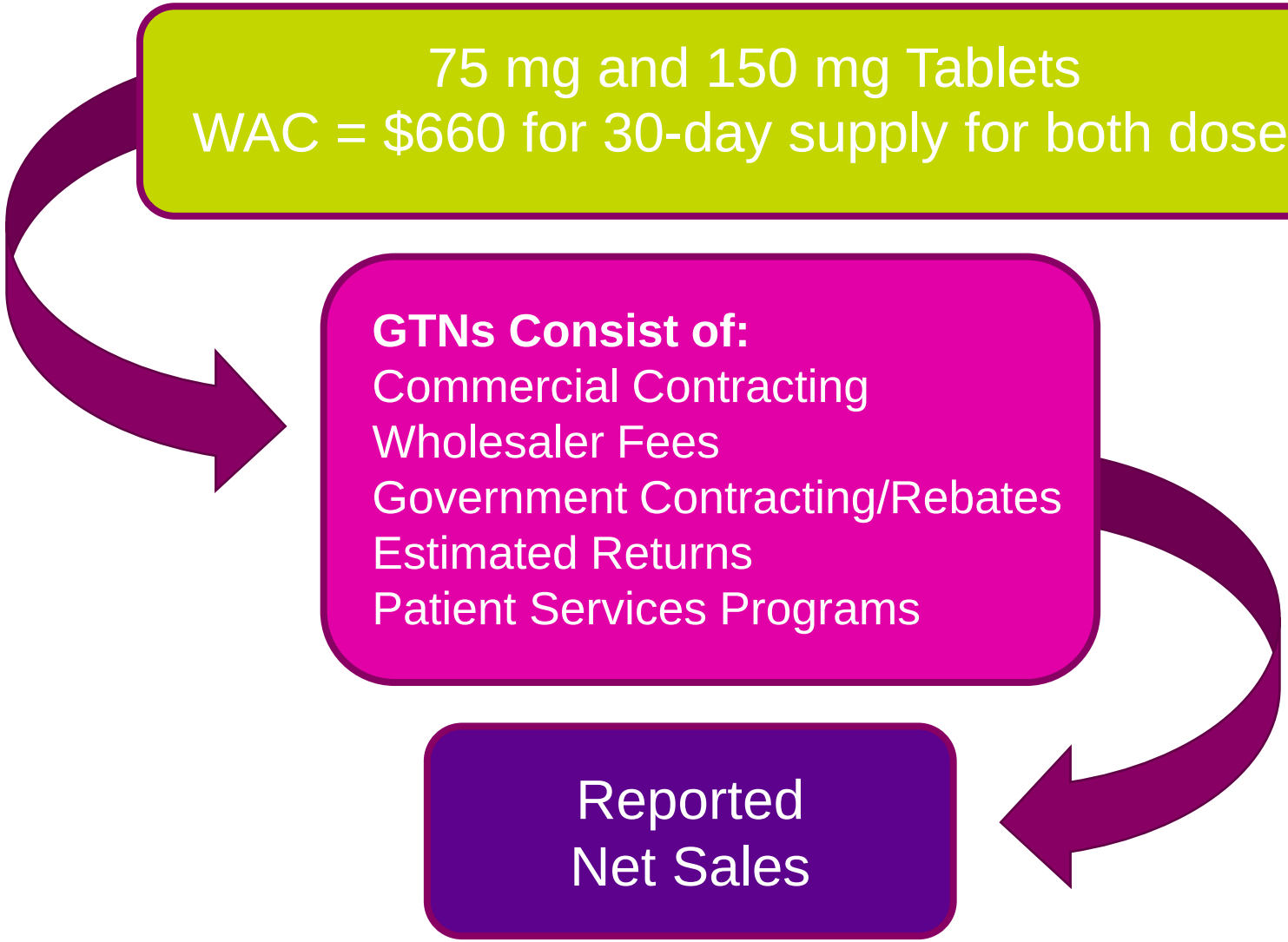
Financial Update

Matt Young
Executive Vice President and Chief Financial Officer

Jazz Pharmaceuticals

Sunosi Price and GTN Makeup

GTN Quarterly Variability Based on Payer Mix



GTNs are estimated to be higher in the near-term until steady-state commercial coverage obtained

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Projected U.S. Launch Curve - OSA & Narcolepsy Only

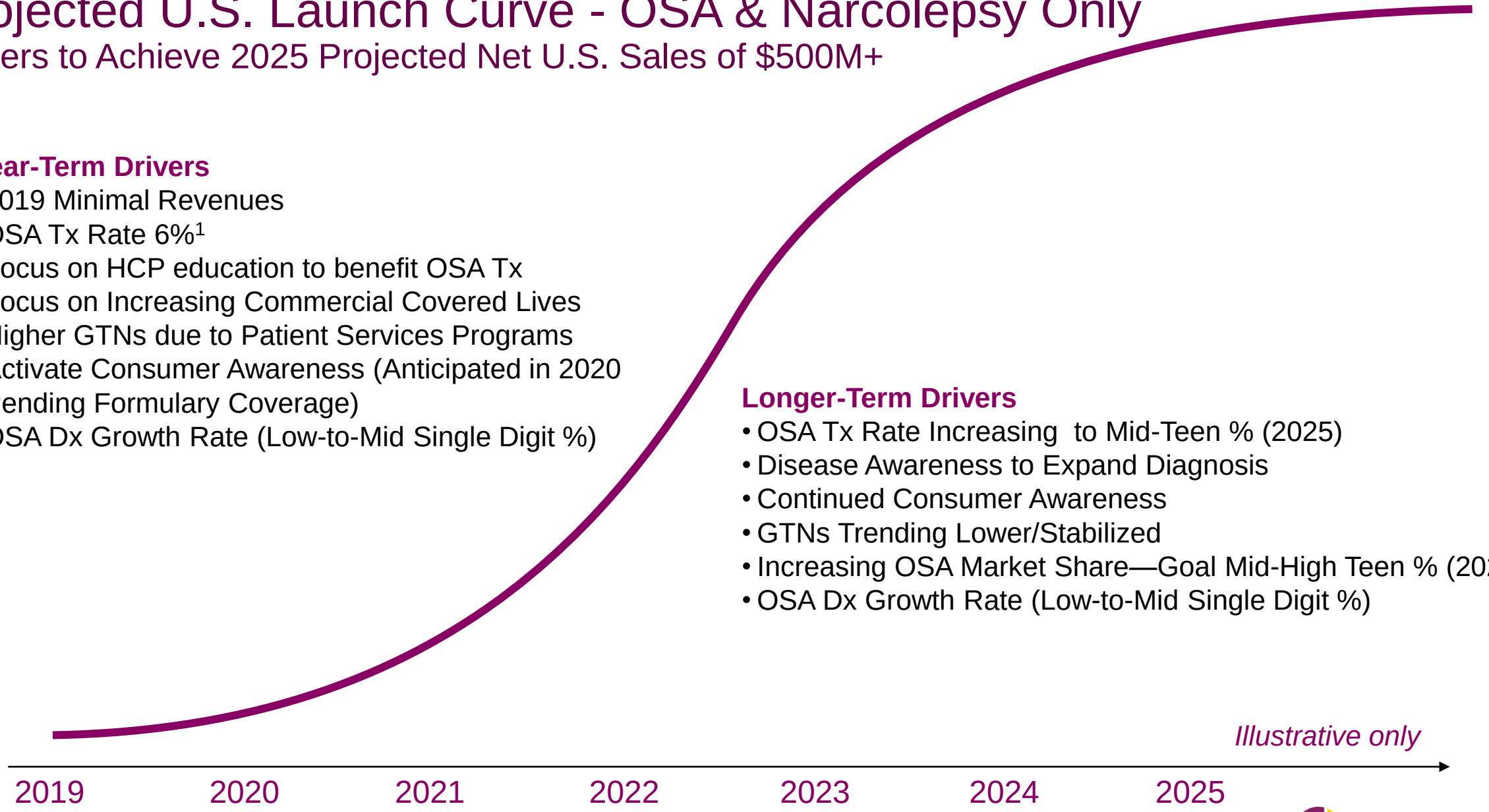
Drivers to Achieve 2025 Projected Net U.S. Sales of $500M+

Near-Term Drivers

- 2019 Minimal Revenues
- OSA Tx Rate 6%[1]
- Focus on HCP education to benefit OSA Tx
- Focus on Increasing Commercial Covered Lives
- Higher GTNs due to Patient Services Programs
- Activate Consumer Awareness (Anticipated in 2020 Pending Formulary Coverage)
- OSA Dx Growth Rate (Low-to-Mid Single Digit %)

Longer-Term Drivers

- OSA Tx Rate Increasing to Mid-Teen % (2025)
- Disease Awareness to Expand Diagnosis
- Continued Consumer Awareness
- GTNs Trending Lower/Stabilized
- Increasing OSA Market Share—Goal Mid-High Teen % (2025)
- OSA Dx Growth Rate (Low-to-Mid Single Digit %)



[1]Source: Diagnosed Prevalence; SHA claims data (2017), Projected 2019 data based on Jazz assumptions

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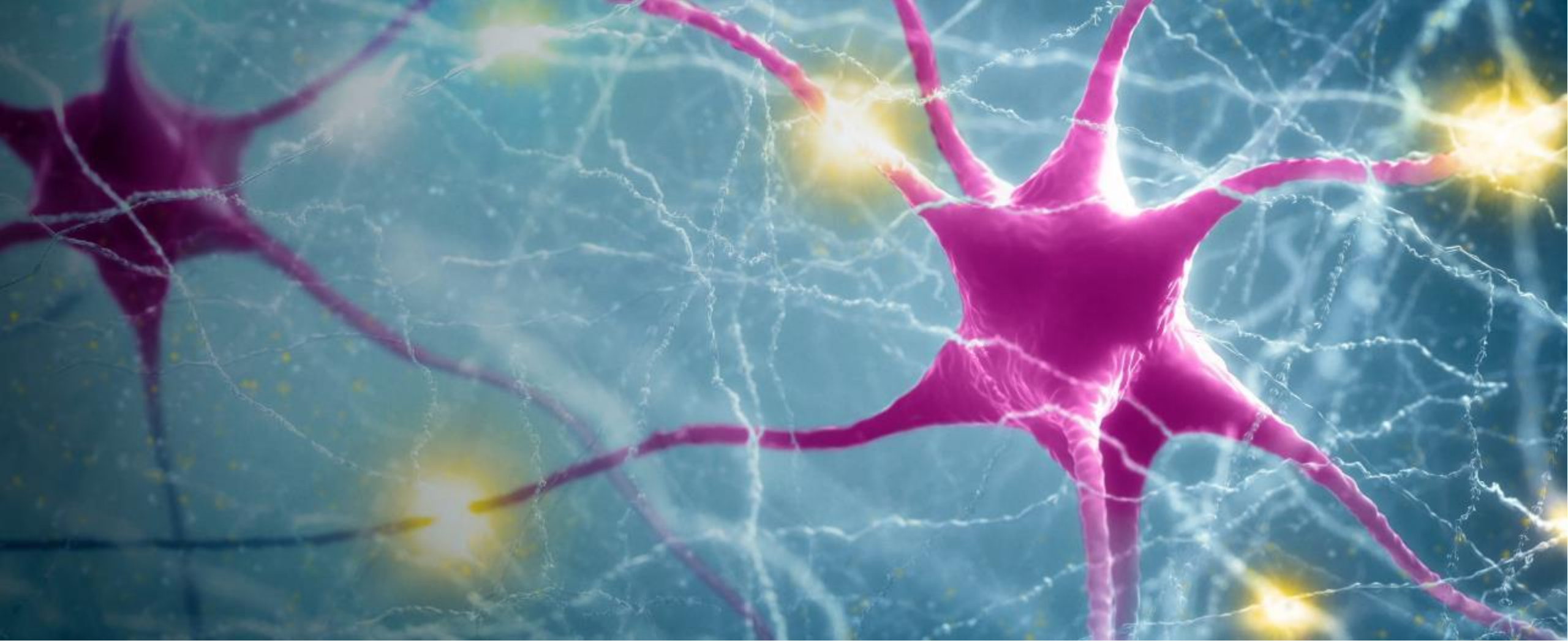

Appendix

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Metrics Summary

Metric	At Launch (2019)	2025 Goal
OSA Tx Rate	6%[1]	Mid-teen %
OSA Market Share	0%	Mid-to-high teen %
Patient Drug Consumption	~6 months/year	
Annual OSA Diagnosis Growth Rate	Low-to-mid single digit %	

[1]Source: Diagnosed Prevalence; SHA claims data (2017), Projected 2019 data based on Jazz assumptions

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CPAP Compliance vs Non-Compliance in Solriamfetol TONES 3 Study

Defined as:

- Compliant use of a primary OSA therapy will be defined as use of ≥ 4 hours per night on ≥ 70% of nights (≥ 5 of 7 nights/week) for subjects who use a device from which hourly usage data can be extracted.
- Compliant use of a primary OSA therapy will be defined as ≥ 70% of nights (≥ 5 of 7 nights/week) by historical report (with investigator concurrence) for subjects who use a device for which usage data cannot be retrieved.
- Receipt of a surgical intervention for OSA symptoms that is deemed to be effective will also be considered compliant use of a primary OSA therapy
- Non-compliant use of a primary OSA therapy will be defined as use at a frequency or duration less than that described above, or receipt of a surgical intervention that is no longer effective in the absence of compliant use of another primary OSA therapy.

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Glossary of Abbreviations

3PL = Third Party Logistics
AAN = American Academy of Neurology
ADHD = Attention Deficit Hyperactivity Disorder
AMCP = Academy of Managed Care Pharmacy
ANA = American Neurological Association
APAP = Auto-titrating Positive Airway Pressure
ATS = American Thoracic Society
BiPAP = Bi-level Positive Airway Pressure
BF = Basal Forebrain
BMI = Body Mass Index
BOND = Burden of Narcolepsy Disease Study
CHEST = American College of Chest Physicians
CI = Confidence Interval
CNS = Central Nervous System
CPAP = Continuous Positive Airway Pressure
CSF = Cerebrospinal Fluid
DEA = U.S. Drug Enforcement Agency
DNRI = Dopamine Norepinephrine Reuptake Inhibitor
DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, 5th Edition
Dx = Diagnosis
EDS = Excessive Daytime Sleepiness
ES = Excessive Sleepiness
ESS = Epworth Sleepiness Scale
EU = European Union
FAASM = Fellow of the American Academy of Sleep Medicine
FCCP = Fellow of the American College of Chest Physicians
FDA = U.S. Food and Drug Administration
GTN = Gross-To-Net
HCP = Healthcare Professional
ICSD-3 = International Classification of Sleep Disorders-Third Edition
IH = Intermittent Hypoxia
LC = Locus Coeruleus
LDT = Laterodorsal Tegmental Nucleus
LTIH = Long-term Intermittent Hypoxia
LS = Least Squares
MAA = Marketing Authorization Application
MACE = Major Adverse Cardiovascular Event
MAO = Monoamine Oxidase
MDD = Major Depressive Disorder
MITT = Modified Intention to Treat
MOA = Mechanism of Action
MSLT = Multiple Sleep Latency Test
MVA = Motor Vehicle Accidents
MWT = Maintenance of Wakefulness Test
NDA = New Drug Application
NDS = New Drug Submission
OSA = Obstructive Sleep Apnea
PAP = Positive Airway Pressure
PBM = Pharmacy Benefit Manager
PGI-C = Patient Global Impression of Change
PPT = Pedunculopontine Nucleus
PSG = Polysomnography
REM = Rapid Eye Movement
Rx = Prescription
SD = Standard Deviation
SE = Standard Error
SHA = Symphony Health
SLEEP = Sleep Research Society Annual Meeting
SOREMP = Sleep Onset REM Period
TONES = Treatment of Obstructive Sleep Apnea and Narcolepsy Excessive Sleepiness
TMN = Tuberomammillary Nucleus
TS = Traditional Stimulants
Tx = Treatment
VTA = Ventral Tegmental Area
WAC = Wholesale Acquisition Cost
WPA = Wake Promoting Agent

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