



WILLIAM C. DEMENT
SLEEP AND CHRONOBIOLOGY BEHAVIORAL
SCIENCE RESEARCH APPRENTICESHIP

BRADLEY HOSPITAL COBRE CENTER FOR
SLEEP AND CIRCADIAN RHYTHMS IN CHILD
AND ADOLESCENT MENTAL HEALTH

Retreat Colloquium
Brown University
Providence, RI

16-18 August 2026

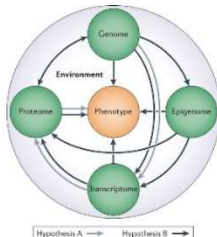
Summer Sleep & Circadian Rhythms Retreat, 2026

Sunday	Event		Topic
1730	Welcome Dinner	<u>Location</u> Center Place Patio 50 Park Row West Providence, RI	Food & Fun
Monday	Event	Speaker	Topic
0800	Breakfast Buffet & Poster Viewing	All	Room 107 (Foyer) Smith Buonanno Hall 95 Cushing St., Providence
0830		Mary A. Carskadon, PhD, Director	Welcome & Introductions All Talks in Smith Buonanno 106
0845		Allyson Hughes, MD, Visiting Investigator	Clinical Phenotype and Circadian Assessment of Non- 24-Hour Sleep–Wake Rhythm Disorder
0945		Sarah McCarthy, Dement Fellow	Sleep's Role in the Presentation and Treatment of Post Traumatic Stress Disorder in Veterans
1000		Daïdo Ouattara, Dement Fellow	Midnight Munchies: How Should Overnight Healthcare Workers Eat to Support Alertness Without Worsening Metabolic Health?

Summer Sleep & Circadian Rhythms Retreat, 2026

Monday	Event	Speaker	Topic
1015		Marshall Horton, Rosekind-Babcock Dement Fellow	Optimizing Athletic Competition within Process C
1030		Kate Batterson, Dement Fellow	Adult Narcolepsy and Idiopathic Hypersomnia: A Comparison of Symptoms, Diagnosis, and Treatment
1045	Morning Tea & Poster Viewing	All	Smith Buonanno 107 (Foyer)
1115		Anastacia Kudinova, PhD, COBRE Research Project Leader	Sleep and Circadian Timing and Suicidal Thoughts and Behavior in Youth: The Role of Self- Referential Processing and Its Neural Correlates
1130		Marie-Rachelle Narcisse, PhD, COBRE Research Project Leader	Sleep and Circadian Rhythm Phenotypes in Adolescent Depression: Exploring the Role of the Oral Microbiome
1145		Sarah A. Thomas, PhD, COBRE Research Project Leader	An Initial Test of Biobehavioral Mechanisms of Sleep Health Alterations in the Context of Adolescent Cannabis Using Objective Methods
1200	Lunch & Poster Viewing	ALL	Smith Buonanno 107 (Foyer)

Summer Sleep & Circadian Rhythms Retreat, 2026

Monday	Event	Speaker	Topic
1315		Michael Scullin, PhD, Visiting Investigator	Sleep and Learning: From the Lab to the Classroom to the Community
1415		Anastacia Kudinova, PhD, COBRE Pediatric Biopsychology Research Core Deputy Director	COBRE Mental Health Assessment and Technology Research Core [Pediatric Biopsychology Research Core]
1430		Jared M. Saletin, PhD, COBRE Sleep and Circadian Methods Research Core Director	COBRE Sleep and Circadian Methods Research Core
1445		John McGeary, PhD, COBRE Pediatric Biopsychology Research Core Biomarker Lab Director & Director of the Biospecimen Laboratory	COBRE Biomarker Research Core
1500		Christopher D. Houck, PhD, COBRE Admin Core Deputy Director & Pilot Project Program Director	COBRE Pilot Project Program
1515	Afternoon Tea & Poster Viewing	All	Smith Buonanno 107 (Foyer)
1540	Small Group Career Discussions	Dement Fellows & Research Assistants with Visiting Investigators, Center Researchers, and Colleagues	Various Locations
1740	Dinner followed by Games	All	Pembroke Green Rain: Smith Buonanno 107 (Foyer)

Summer Sleep & Circadian Rhythms Retreat, 2026

Tuesday	Event	Speaker	Topic
0830	Breakfast & Poster Viewing	All	Room 107 (Foyer) Smith Buonanno Hall 95 Cushing St., Providence
0900		Darlynn Rojo-Wissar, PhD, COBRE Pilot Project Leader	Effects of Domestic Violence on Relationships among Father Involvement and Child Sleep and Mental Health
0915		Shufang Sun, PhD, COBRE Pilot Project Leader	Thriving in a Digital World: A Mindfulness-Based Pilot to Support Sleep and Mental Health in Teens
0930		Grace Cushman, PhD, COBRE Pilot Project Leader	An Investigation of Sleep Health, Stress, and Disease Symptoms in Pediatric Functional Abdominal Pain Disorders
0945		Petya Radoeva, PhD, COBRE Pilot Project Leader	Neural Correlates of Sleep Problems in Adolescents with Autism Spectrum Disorder
1000		Catherine Dement Roos, Guest of Honor	William Dement, a legacy in Sleep
1045	Morning Tea & Poster Viewing <i>(Last chance to view!)</i>	All	Smith Buonanno 107 (Foyer)

Summer Sleep & Circadian Rhythms Retreat, 2026

Tuesday	Event	Speaker	Topic
1115		Katya Michkovskaia, Dement Fellow	How Does the Brain Construct Dream Reality?
1130		Ashna Akepati, Dement Fellow	Sleep Health During Infancy as a Predictor of Autism Development in Childhood
1145		Jane Poinsette, Dement Fellow	The Role of Slow-Wave Sleep in Alzheimer's Dementia
1200		Kathy Mitchell, Dement Fellow / Research Assistant	Sleep Deprivation, Inflammation, and Disease: The Role of Immune Dysregulation
1215		Mary A. Carskadon, PhD, Director	Conclusion & Thanks
1230	Box Lunch To Go	ALL	Smith Buonanno 107 (Foyer)
1245	COBRE EAC Meeting	EAC Committee Members	
		Adjourn	

Summer Sleep & Circadian Rhythms Retreat, 2026

Visiting Investigator Presentations

Allyson Hughes, MD

Assistant Professor in the department of Pulmonary, Critical Care, and Sleep Medicine, Warren Alpert Medical School, Brown University

Clinical Phenotype and Circadian Assessment of Non-24-Hour Sleep–Wake Rhythm Disorder

We performed systematic review of sighted patients with N24SWD at BIDMC between February 2007 and May 2026 utilizing baseline demographic and health features, sleep features, PSG data, melatonin profiles, and cloud-based PAP data when available. Our conclusions show that N24SWD represents a complex, heterogeneous phenotype extending beyond isolated rhythm drift. A multimodal longitudinal framework integrating behavioral records, endogenous biomarkers, and digital timing clues support the clinical assessment and management of complex N24SWD. Additionally, we review 4 patients with comorbid N24SWD, OSA, and central hypersomnia treated with sodium oxybate therapy.

Michael Scullin, PhD

Professor of Psychology and Neuroscience, Baylor University, Baylor, TX

Sleep and Learning: From the Lab to the Classroom to the Community

In this talk, Dr. Michael Scullin will share his career trajectory from his early stumblings as an undergraduate research assistant to his current position as Professor of Psychology and Neuroscience at Baylor University. Along the way, he will describe how both luck and intentional pursuit led him to develop a program of research on sleep and learning. The talk will highlight his team's efforts to move from ecologically plausible laboratory studies to classroom-based interventions designed to improve student learning and well-being. It will conclude with his work translating research for the broader public through the development of museum exhibits on sleep and brain health.

Catherine Dement Roos

William Dement, a legacy in Sleep

This talk will include personal stories of how Dr. Dement's vision, passion, and unique personality shaped the fields of sleep research and sleep medicine. By offering a daughter's perspective on highlights of his career, I hope to inspire the next generation of sleep researchers to build on his legacy.

Summer Sleep & Circadian Rhythms Retreat, 2026

EP Bradley Hospital Sleep Research Lab Apprenticeship Faculty, Trainees, & Staff

APPRENTICESHIP FACULTY

Alpert Medical School of Brown University,
Department of Psychiatry and Human
Behavior

Mary A. Carskadon, PhD, Director

Jared M. Saletin, PhD, Deputy Director

David H. Barker, PhD

Rachel S. Herz, PhD

John McGeary, PhD

Marie-Rachelle Narcisse, PhD

Darlynn Rojo-Wissar, PhD

DEMENT FELLOW GUEST MENTORS

Major Allison Brager, PhD, States Military Academy
West Point

Amanda Cremone-Caira, PhD, Merrimack College

Karen Konkoly, PhD, University of Cambridge, UK

Bryce Mander, Ph.D., University of California Irvine

Kiran Maski, MD, Harvard University

John McGeary, PhD, Brown University

Lindsay Stager, PhD, Brown University

RESEARCH COLLABORATION

DOD MTEC

Eliza Van Reen, PhD, CPS, Inc., Newport, RI

Jay Mulrooney, CPS, Inc., Newport, RI

NIDA MOUD

Chelsie Benca-Bachman, PhD, Alpert Medical
School of Brown University

John E. McGeary, PhD, Alpert Medical School of
Brown University

Ju Park, PhD, Alpert Medical School of Brown
University

Susan Ramsey, PhD, Alpert Medical School of
Brown University

Josiah D. Rich, MD, MPH, Alpert Medical School of
Brown University

SLEEP LAB TRAINEES, E.P.

BRADLEY HOSPITAL

Kathleen O'Hora, PhD, DPHB T32 Postdoctoral
Research Fellow in Child Mental Health,
Alpert Medical School of Brown University

Lindsay Stager, PhD, DPHB T32 Postdoctoral
Fellow in Cardiovascular Behavioral
Medicine, Alpert Medical School of Brown
University

SLEEP LAB RESEARCH STAFF, E.P.

BRADLEY HOSPITAL

Caroline Gredvig-Ardito, Data Manager & IRB
Coordinator

Sriya Chakravarty, Research Assistant

Jayne Clark, Senior Supervising Sleep
Technologist

Stephanie Manuli, Clinical Research Assistant

Kelly McCormick, Supervising Sleep Technologist

Kathy Mitchell, Research Assistant

Frances Moriniere, Super Tech

Sophia Nicogossian, Super Tech

Luna Sato, Research Assistant

Devon Ryan, Super Tech

Katherine Skordinski, Supervising Sleep
Technologist

Summer Sleep & Circadian Rhythms Retreat, 2026

EP Bradley Hospital COBRE Center for Sleep and Circadian Rhythms in Child and Adolescent Mental Health Faculty, Staff, & Colleagues

CENTER EXECUTIVE LEADERSHIP TEAM

Mary A. Carskadon, PhD, Director
Christopher Houck, PhD, Admin Core Deputy Director
Jennifer Wolff, PhD, Pediatric Biopsychology Research Core (PBRC) Director
John E. McGeary, PhD, Pediatric Biopsychology Research Core (PBRC) Biomarker Lab Director
Anastacia Y. Kudinova, PhD, PBRC Deputy Director, RPL
Jared M. Saletin, PhD, Sleep and Circadian Methods Research Core (SCMRC) Director
David Barker, PhD, SCMRC Deputy Director
Cristal L. Medeiros, COBRE Administrator and Department Research Administrator

CENTER PROJECT LEADERS

Anastacia Kudinova, PhD, Research Project Leader
Marie-Rachelle Narcisse, PhD, Research Project Leader
Sarah A. Thomas, PhD, Research Project Leader
Diana Grigsby-Toussaint, PhD, Research Project Grad
Jared M. Saletin, PhD, Research Project Grad
Giulia Righi, PhD, Research Project Grad
Grace Cushman, PhD, Pilot Project Leader
Petya Radoeva, MD, PhD, Pilot Project Leader
Shufang Sun, PhD, Pilot Project Leader
Darlynn Rojo-Wissar, PhD, Pilot Project Leader
Selby Conrad, PhD, Pilot Project Grad
Justin Parent, PhD, Pilot Project Grad
Elizabeth Thompson, PhD, Pilot Project Grad

CENTER EXTERNAL ADVISORY COMMITTEE

Candice A. Alfano, PhD, Stonybrook University, EAC Chair
Ruth M. Benca, MD, PhD, Wake Forest University
Girardin Jean-Louis, PhD, University of Miami
Lisa J. Meltzer, PhD, National Jewish Health, University of Colorado

CENTER LOCAL GUIDANCE GROUP

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Henry Sachs, MD (Ex officio), President, Bradley Hospital
Larry Brown, MD, Director, Child, and Family Psychiatry
Daphne Koinis-Mitchell, PhD, Hasbro Child Psychology
Diane Lipscombe, PhD, Director, Carney Institute for Brain Science
Elizabeth McQuaid, PhD, Director, Child & Adolescent Psychology
Sharon Rounds, PhD, Director, Advance CTR
Anthony Spirito, PhD, Professor of Psychiatry, Alpert Medical School of Brown University

CENTER STAFF & COLLEAGUES

Stephanie Goldstein, PhD, PBRC Consultant
Judy Kimberly, PhD, Admin Core Evaluation Consultant
Bridget Berman, PBRC Lab Assistant
Christopher Breault, PBRC Consultant
Sarah Cunningham, PBRC Research Assistant
Catrina Elizardo, Research Assistant
Nikole Giovannone, Research Assistant
Isabella Gomez, PBRC Research Assistant
Kiara Medeiros, Research Assistant
Mehlika Ozgencil, PBRC Research Assistant
Leslie Ramirez, Research Assistant
Celia Roberto, Research Assistant
Aashritha Sadu, SCMRC Research Assistant

Summer Sleep & Circadian Rhythms Retreat, 2026

2026 WILLIAM C. DEMENT FELLOWS

Ashna Akepati

Rising Senior at University of Houston majoring in Psychology with minors in Biology and Medicine & Society

Kate Batterson

Rising Senior at University of South Carolina majoring in neuroscience and art history

Marshall Horton

Graduate of the University of Notre Dame with an honors B.A. degree in Psychology and Spanish

Sarah McCarthy

Rising Senior at University of Massachusetts Amherst obtaining dual degrees in Psychology and Mathematics with a concentration in Statistics

Katya Michkovskaia

Rising Junior at Brown University concentrating in Neuroscience and Literary Arts

Kathy Mitchell

Graduate of University of Rochester with a concentration in Microbiology and Immunology

Daïdo Ouattara

Rising Senior at Brown University concentrating in Public Health and Health & Human Biology

Jane Poinsette

Graduate of University of South Carolina with a concentration in Cognitive and Behavioral Neuroscience

ACKNOWLEDGEMENTS

The summer apprenticeship and research projects were funded by generous donations from Dr. Christine Acebo, Dr. Candice Alfano, Dr. Thomas F. Anders, Mr. Henry Arantes, Dr. Debra Babcock, Dr. Dean Beebe, Dr. Ruth Benca, Dr. Nathaniel Bowling, Major Allison Brager, Dr. Mary Carskadon, Brig. Gen. Bruce M. Carskadon, Ms. Amy Carskadon, Dr. Valerie Crabtree, Dr. Leisha Cuddihy, Dr. Namni Goel, Dr. Jennifer Goldschmied, Dr. Diana Grigsby-Toussaint, Dr. Allison Harvey, Dr. Brant Hasler, Ms. Lois Hely, Dr. Rachel Herz, Mr. Thomas Kazlauskis, Dr. Elizabeth Klerman, Dr. Anastacia Y. Kudinova, Ms. Lynne Lamberg, Ms. Chanliny Lim, Dr. Stephanie Crowley McWilliam, Dr. Lisa J. Meltzer, Mr. Kenneth Miller, Dr. Richard Millman, Dr. Jodi A. Mindell, Ms. Alexandria Muench, Dr. Janet M. Mullington, Dr. Marie-Rachelle Narcisse, Dr. Edward F. Pace-Schott, Ms. Elaine Poole, Dr. Donn Posner, Dr. Darlynn Rojo-Wissar, Ms. Catherine Roos, Dr. Mark Rosekind, Dr. Tracy R. Rupp, Dr. Michael Scullin, Dr. Katherine Sharkey, Celso E. Teixeira, Dr. Michelle G. Thompson, Ms. Julie C. Thurman, Dr. Fred Turek, Dr. Joyce A. Walsleben, Dr. Amy Wolfson, Dr. Lora Wu, Dr. James Wyatt, Dr. Phyllis C. Zee. This work was also supported by the EP Bradley Hospital COBRE Center for Sleep and Circadian Rhythms in Child and Adolescent Mental Health funded by the NIGMS grant number P20GM139743. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Summer Sleep & Circadian Rhythms Retreat, 2026

DEMENT FELLOW POSTER PRESENTATIONS

Ashna Akepati

Sleep Health During Infancy as a Predictor of Autism Development in Childhood

Kate Batterson

Adult Narcolepsy and Idiopathic Hypersomnia: Comparison of Symptoms, Diagnosis, and Treatment

Marshall Horton

Optimizing Athletic Competition through the Lens of Process C

Sarah McCarthy

Sleep's Role in the Presentation and Treatment of PTSD in Veterans

Katya Michkovskaia

How Does the Brain Construct Dream Reality?

Kathy Mitchell

Sleep Deprivation and Immune Dysregulation: Mechanisms Linking Sleep Loss to Chronic Inflammation

Daïdo Ouattara

Midnight Munchies: Can Chrononutrition Support Alertness and Metabolic Health During Overnight Work?

Jane Poinsette

The Role of Slow-Wave Sleep in Alzheimer's Dementia Risk

Sleep Health During Infancy as a Predictor of Autism Development in Childhood

Ashna Akepati^{1, 2}

1. Sleep for Science Laboratory, Brown University Health
2. Department of Psychology, University of Houston



Introduction

- During infancy, the circadian rhythm is rapidly developing.^{7, 8}
- Infant sleep is crucial for neurodevelopment. Sleep spindles and slow waves were linked to various neurodevelopmental outcomes.^{9, 10}
- Assessing how sleep during infancy affects neurodevelopmental trajectories, particularly Autism Spectrum Disorder (ASD) risk, is essential

Introduction

- In one retrospective study, infants were categorized as either an “irritable” or “apathetic” type of neonate. There were significantly more irritable-type neonates in the ASD group. They also had more nighttime awakenings and delayed sleep-onset latency (SOL).⁷
- Another longitudinal study found that toddlers diagnosed with ASD at 3 years old had a shorter sleep duration and more night awakenings during infancy (Figure 1).¹

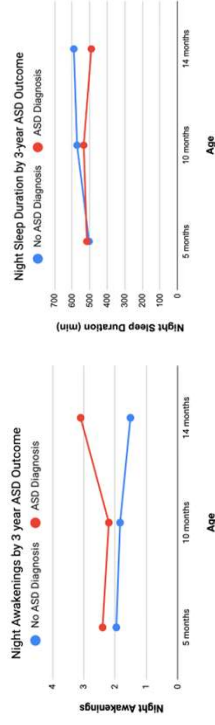


Figure 1

References

1. Begum-Al-J, Gossell, K., Mason, L., Pasco, G., Charnan, T., Johnson, M. H., Jones, E. J., & The STARKS Team. (2023). Infant sleep predicts trajectories of neurodevelopmental outcomes at 3 years. *Journal of Child Psychology and Psychiatry*, 64(1), 1-10. <https://doi.org/10.1111/jcpp.15000>
2. Doi, L. A., Pincus, A. L., & Johnson, M. H. (2023). Sleep and neurodevelopmental outcomes at 3 years. *Journal of Child Psychology and Psychiatry*, 64(1), 1-10. <https://doi.org/10.1111/jcpp.15000>
3. O'Brien, C. M., & Johnson, M. H. (2023). Sleep and neurodevelopmental outcomes at 3 years. *Journal of Child Psychology and Psychiatry*, 64(1), 1-10. <https://doi.org/10.1111/jcpp.15000>
4. Grunzire, M., Jackson, K., Spurner, N. J., Gibson, J., Whitham, J., Williams, A., S., Doherty, R., & Kenworthy, D. J. (2016). Behavioral interventions for infant sleep problems: A randomized controlled trial (PEDIATRICS). *PEDIATRICS*, 137(6), <https://doi.org/10.1542/peds.2015-1486>
5. Research, 19(11), 2231-2239. <https://doi.org/10.1002/aur.70724>
6. MacDuffie, K. E., Shen, M. D., Doger, S. R., Styer, M. A., Kim, S. H., Peterson, S., Parley, J. J., St. John, J. T., Wolff, J. J., Swanson, M. R., Botteron, K. N., Zsigmond, L. L., Piven, J., & Estes, A. M. (2020). Sleep onset problems and neurodevelopment in infants later diagnosed with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 61(12), 1611-1620. <https://doi.org/10.1111/jcpp.15000>
7. Make, T., Toyooka, M., Tanaka, S., Koriishi, Y., Oniki, K., Sawashita, J., Tajima, S., Koriishi, J., Naka, A., & Kikuchi, K. (2020). Neonatal infant sleep wake rhythm as a predictor of autism spectrum disorders. *Neurobiology of Sleep and Circadian Rhythms*, 9, 100053. <https://doi.org/10.1016/j.neurobi.2020.100053>
8. O'Brien, C. M., & Johnson, M. H. (2023). Sleep and neurodevelopmental outcomes at 3 years. *Journal of Child Psychology and Psychiatry*, 64(1), 1-10. <https://doi.org/10.1111/jcpp.15000>
9. Thim, E., Schneider, N., & Broekman, B. (2017). Infant sleep and its relation with cognition and growth: a narrative review. *Nature and Science of Sleep*, Volume 9, 1-10. <https://doi.org/10.1016/j.nsc.2017.09.006>
10. Ventura, S., Matheson, S. R., O'Toole, J. M., Livingston, V., Murray, D. M., & Boylan, G. B. (2025). Infant sleep EEG features at 4 months as biomarkers of neurodevelopment at 18 months. *Pediatric Research*, 98(4), 1474-1485. <https://doi.org/10.1038/s41390-025-03893-6>
11. White-Traut, R. C., Nelson, M. N., Shewitt, J. M., Cunningham, N., & Pineda, M. (1997). Responses of preterm infants to unimodal and multimodal sensory intervention. *Pediatric Nursing*, 23(2), 105-112. <https://pubmed.ncbi.nlm.nih.gov/1653507/>

Sleep Onset and Hippocampal Development

- In infants at elevated likelihood (EL) for ASD who were later diagnosed, high infant sleep onset problems (ISOP) predicted steeper gains in hippocampal volume (Figure 2).⁶
- Hippocampal connectivity may contribute to core ASD symptoms such as social awareness and restricted/repetitive behaviors.⁵
- Further research is needed to establish whether sleep onset problems are a cause or symptom of ASD-related neurodevelopmental differences.

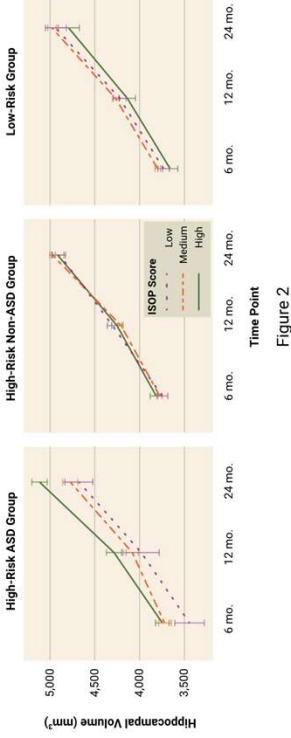


Figure 2

Sensory Gating and Sleep Problems

- EL and typical likelihood (TL) infants aged 8-11 months old napped with no noise and then with 60dB pure tones. Infants with greater sensory sensitivity had decreased slow-wave and sleep spindle densities during sleep.³

- In another study, EL infants were more likely to have a lower tactile suppression index (TSI). Reduced sensory gating was then found to predict delayed SOL across EL and TL infants (Figure 3).²

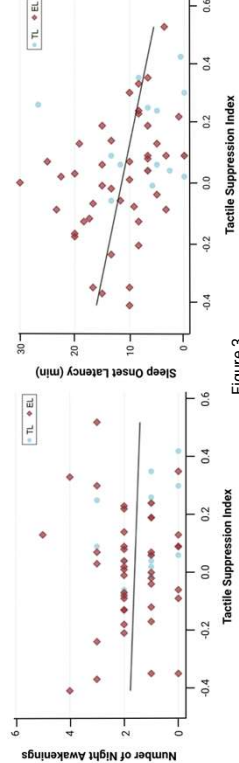


Figure 3

Autobiography

- Incoming senior at the University of Houston, majoring in Psychology with minors in Biology and Medicine & Society
- Researching at the Sleep and Anxiety Center of Houston and the Neuroscience of Development & Evidence-based Interventions Lab at UH, as well as the Moreno Lab at Baylor College of Medicine
- I chose this topic because I want to improve early detection and intervention for neurodivergent youth to support success in school, work, and relationships.
- After graduation, I plan on taking a gap year and working full-time in research before applying to PhD programs in Clinical Psychology.

Significance

- Identifying early factors that play a role in ASD development can lead to an earlier diagnosis & treatment.
- Doing so can improve quality of life and long-term neurodevelopmental/behavioral outcomes for children and parents.¹

Future Directions

- Sleep interventions for families/infants to improve sleep quality and decrease ASD symptom severity⁴
- Pilot studies for sensory interventions to help EL infants¹¹
- More studies with objective sleep measures like polysomnography and actigraphy

Background

Narcolepsy and idiopathic hypersomnia (IH) are disorders of hypersomnolence characterized by excessive daytime sleepiness (EDS), issues with nocturnal sleep, and markedly reduced quality of life.^{1,2}

- Narcolepsy → type 1 (NT1) & type 2 (NT2), often involves disrupted nighttime sleep (DNS) and cataplexy in NT1.³
- IH is 1 of 3 types of hypersomnia.³

Symptoms & Signs

Table 1: Comparison of Narcolepsy & IH Symptoms

Narcolepsy	Both	IH
More abbreviated, disrupted nighttime sleep ³	EDS: sleep attacks, involuntary napping, excessive sleep need, difficulty holding attention, & cognitive dysfunction ⁶	Extensive sleep duration
“Irresistible” daytime sleep tendency ⁵	Additional comorbidities: fatigue, obesity, psychiatric disorders (depression, anxiety), ⁶ sleep disorders (sleep apnea, RBD, RLS, PLMS) ⁵	More resistible daytime sleep tendency
DNS: sleep paralysis, hypnagogic/hypnopompic hallucinations, & vivid dreams/nightmares ⁶	Heterogeneous disorders with no known causes (NT2, IH) ⁶	Severe sleep inertia
Cataplexy, loss & consequential deficiency of orexin neurons (NT1) ⁷	Possibly immune-mediated conditions that develop in genetically predisposed individuals ⁶	Long, unrefreshing naps

Pathways of Diagnosis

Two genes are overrepresented in these disorders:

- Narcolepsy: HLA-DQB106:02 (especially overrepresented in NT1)⁸
- Idiopathic hypersomnia: HLA-DQB102⁸
Although insufficient for diagnosis, genes can indicate the presence of these disorders.⁸ This HLA class II genotyping aligns with the potential of narcolepsy and IH to be autoimmune disorders.⁸

Clinical diagnosis of narcolepsy or IH is based on a patient's sleep history and their thorough neurologic, general medical, and psychiatric assessment.³

- Only use sleep laboratory recordings if auxiliary symptoms are absent or other comorbid sleep disorders are suspected³
- Multiple questionnaires and scales test for the conditions, including the ESS,¹⁰ MSLT,¹¹ MWT,¹² NSS-CT,¹³ HIS,¹⁴ IHSS,¹⁵ and SIQ,¹⁶ — measuring subjective daytime sleepiness, sleep tendency, mean sleep latency, ability to stay awake, sleep inertia, and symptom severity, frequency, and impact.

Adult Narcolepsy and Idiopathic Hypersomnia: Comparison of Symptoms, Diagnosis, and Treatment

Kate P. Batterson
1. Sleep for Science – Bradley Hospital Sleep Lab at Brown Health 2. University of South Carolina

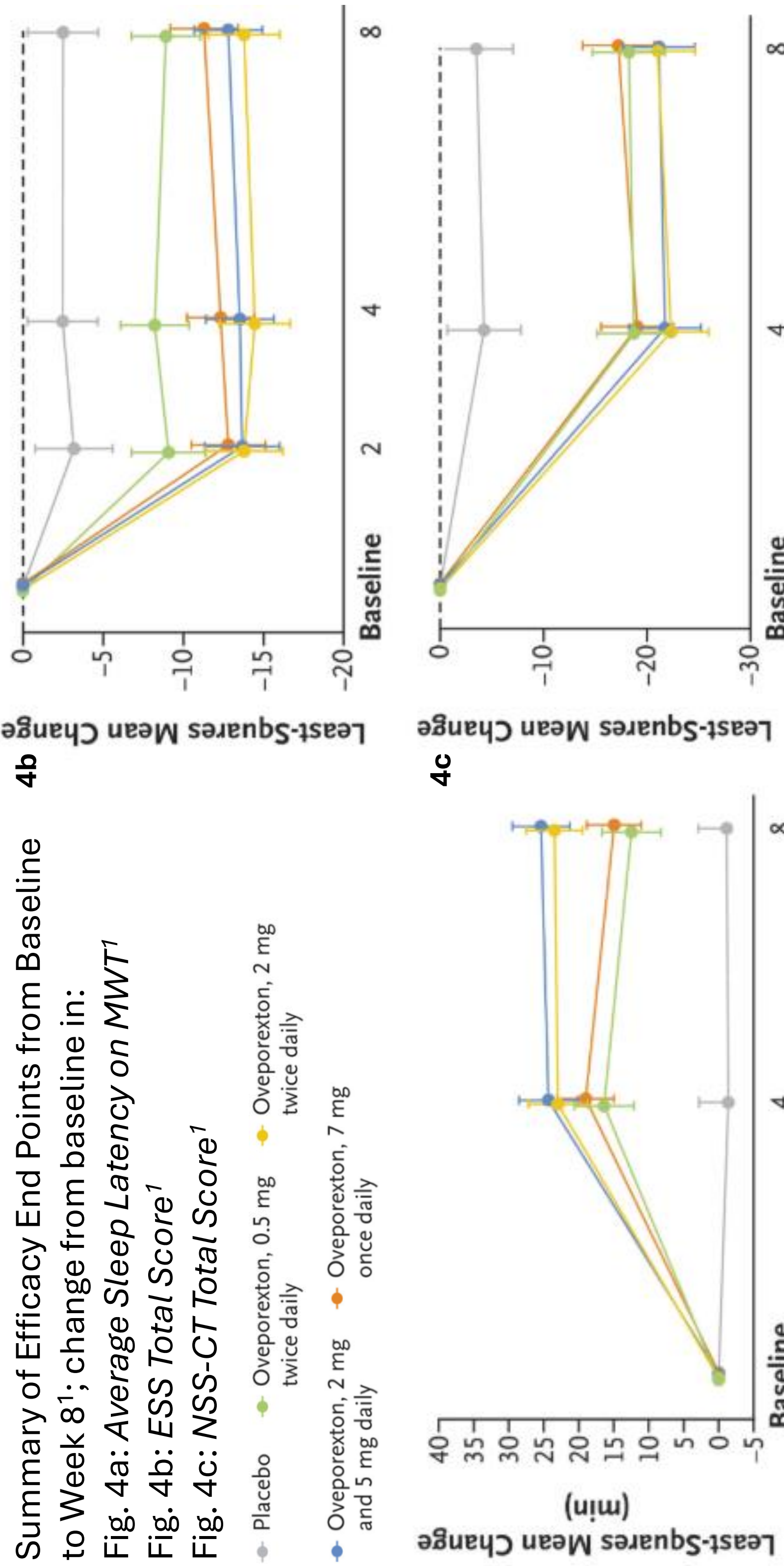
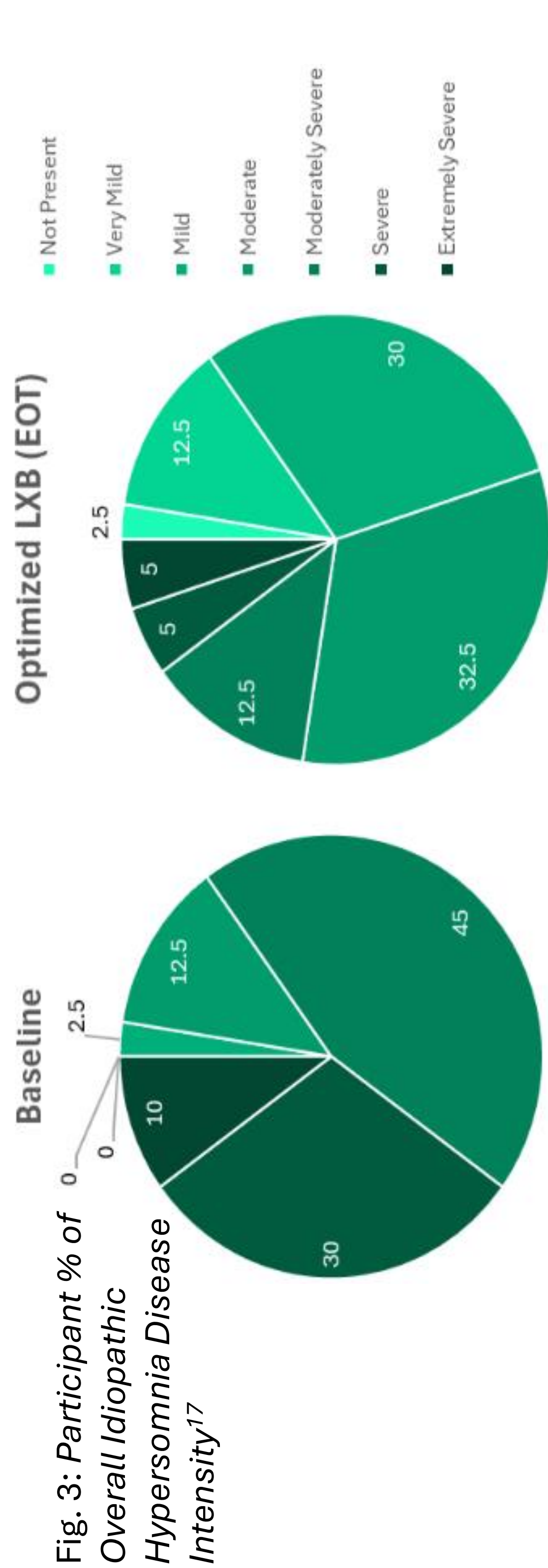
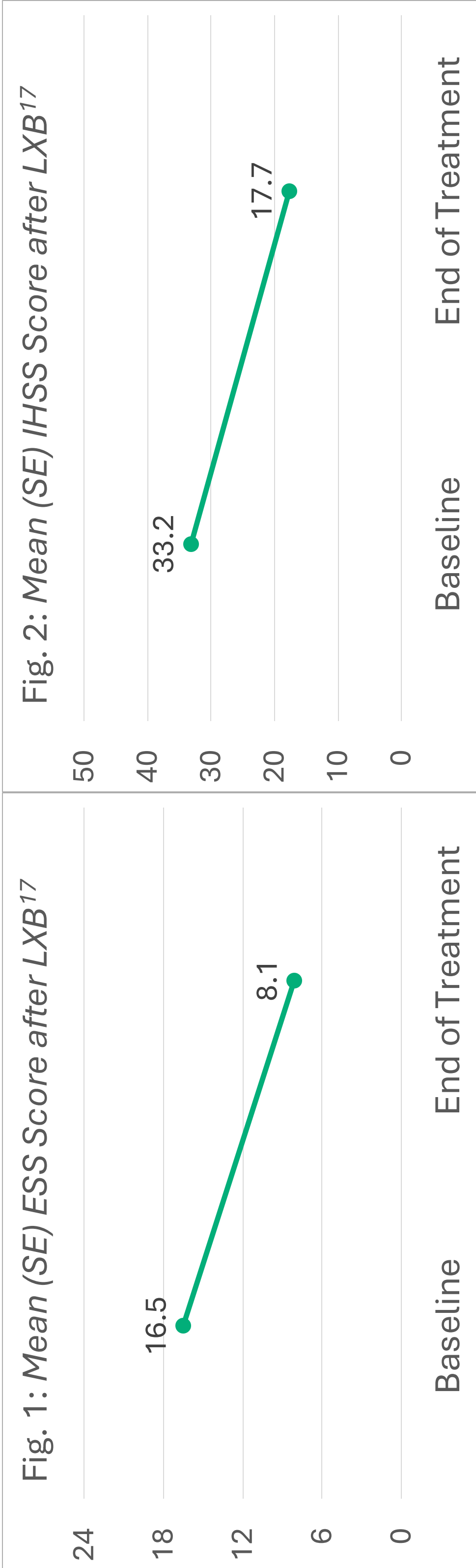
Pharmacological Treatments

Medications & therapies for narcolepsy and IH are often necessary in moderate to severe cases, and they have substantially stronger evidence than behavioral interventions.^{6,2}

- Evidence for many medications and traditional stimulants is limited²
- Some data shows wake-promoting agents improve EDS⁶
- Weak/no data exist concerning sleep paralysis and hallucination treatment⁶

Table 2: Three Different Pharmacological Treatments for Narcolepsy & IH

Modafinil	Low-Sodium Oxybate (LXB)	Ovoporexton (TAK-861)
• Available, has strongest evidence for improving IH symptoms ⁶	• Available, has strongest evidence for improving IH symptoms and cataplexy ^{6,2}	• Preclinical stage, a highly selective oral OX2R agonist ¹
• Most extensively studied, considered the first-line clinical treatment ²	• Recent advancement in IH treatment ²	• Significantly improved measures of wakefulness, sleepiness, and cataplexy over an 8-week period in NT1 (Fig. 4) ¹
• Consistently improves EDS ²	• Significant improvements in EDS, sleep inertia, and overall functioning ² (Fig. 1, Fig. 2)	• Yielded greater dose-dependent improvements in MWT and ESS than available narcolepsy medications ¹



Non-Pharmacological Treatments

Examples of non-pharmacological treatments include:

- Scheduled naps* and bed/rise times to improve subjective & objective EDS^{2,6}
- Structured daytime activities²
- Cognitive behavioral therapy for hypersomnia (CBT-H)²
- Sleep hygiene education & implementation²
- Maintenance of general health (e.g. regular physical activity, dieting)⁶

These may be more feasible than medications due to youth, pregnancy, or other limiting factors.⁶

- Limited scientific support & efficacy, yet strongly recommended with or without medication to promote compliance and acceptance^{6,2}
- Most patients still need pharmacological treatment due to severe symptoms.⁴
- *Naps generally provide little/no benefit in IH due to their prolonged, unrefreshing nature.²

Significance & Next Steps

Despite shared symptoms, narcolepsy and IH are different disorders that warrant different diagnoses & treatments. Both patients report severe impacts on their daily lives, affecting work, academics, social life, and personal relationships.⁴

- Often struggle to receive the correct diagnosis due to various similar and/or overlapping conditions; many wait years (even 10+) for a diagnosis.⁴

Further Research:

- Distinguishing phenotypes of narcolepsy & IH via genetic characterization → possibility of disparate biological mechanisms⁸
- Orexin replacement therapy, orexin selective receptor agonists, & histamine 3 receptor inverse agonists to treat cataplexy & EDS^{6,1}
- More randomized clinical trials assessing DNS in narcolepsy⁶
- More targeted IH research, therapies, & long-term management strategies²

Autobiography

About Me

- Originally from Orlando, FL
- Neuroscience & art history major in the University of South Carolina Honors College; graduating May 2027

- Began sleep research in fall 2024 under Dr. Alexandria Reynolds
- Career goal: clinical counselor in sleep, fertility, and/or genetics

Why research narcolepsy & IH?

I've struggled with daytime sleepiness, low energy, and excessive sleep duration since high school. SLEEP 2026 made me consider the possibility of having a hypersomnolence disorder, so I wanted to further investigate.

References

1. Dauvilliers Y, Rutenfranz J, Kales A, et al. (2023). Narcolepsy and hypersomnia: a review of the evidence on pharmacological and nonpharmacological interventions for idiopathic hypersomnia. *Journal of Clinical Sleep Medicine*. / *JCSM* : official publication of the American Academy of Sleep Medicine. 2023;23(12):2023-2034. <https://doi.org/10.1093/sleep/zsac087>
2. Sani, V., & Sani, S. (2024). A scoping review of the evidence on pharmacological and nonpharmacological interventions for idiopathic hypersomnia. *Seminars in Neurology*, 73(3), 250-258. <https://doi.org/10.1055/s-008-1041425>
3. Martinelli, R. L., Brennan, R. W., & Cadeau, R. J. (1987). Disorders of excessive daytime sleepiness: narcolepsy and hypersomnia. *Seminars in Neurology*, 7(3), 250-258. <https://doi.org/10.1055/s-008-1041425>
4. Ortiz, L. E., Morse, A. M., Kram, L., Lavender, M., Horsnell, M., Cronin, D., Schneider, B., & Gudeman, J. (2025). A Survey of People Living with Narcolepsy in the USA: Path to Diagnosis, Quality of Life, and Treatment Landscape from the Patient's Perspective. *CNS drugs*, 39(Suppl 1), 23-36. <https://doi.org/10.1007/s00263-024-01142-8>
5. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
6. Basareo, V., & Dauvilliers, Y. (2023). Narcolepsy and hypersomnia: a review of the evidence on pharmacological and nonpharmacological interventions for idiopathic hypersomnia. *Journal of Clinical Sleep Medicine*. / *JCSM* : official publication of the American Academy of Sleep Medicine. 2023;23(12):2023-2034. <https://doi.org/10.1093/sleep/zsac087>
7. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
8. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
9. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
10. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
11. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
12. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
13. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
14. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
15. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
16. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>
17. Dauvilliers, Y., & Lammien, G. J. (2017). European guidelines and expert statements on the management of narcolepsy in adults and children. *European Journal of Neurology*, 28(15), 2830-2839. <https://doi.org/10.1002/ajmg.b.32322>

Background

- Wanted to be equipped to advise athletes on how to optimize their workouts and competitions.
- Even within sports, competitions vary widely in their start times.
- A growing sports culture sees athletes routinely crossing national and international borders without time to acclimate.

Literature Review

- Consisted of 3 domains:
1. An overview of the circadian rhythm of peak athleticism.
 2. The circadian rhythm of athleticism with respect to chronotype.
 3. Countermeasures to circadian misalignment in a new age of competition.

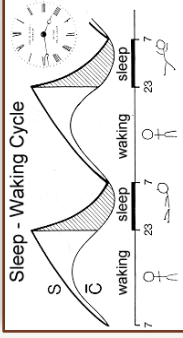
1 – Circadian Rhythm of AP

- A large body of evidence supports optimal athletic performance (AP) when body temperature peaks around 1700.
- Body temperature is related to several physical variables, such as anaerobic power and muscle strength, so sports that involve a lot of contact, sprinting, or lifting will benefit most from an afternoon session.
- Over time, performance improves during a certain time of day if one repeatedly trains at that time, which is important particularly for athletes whose sports typically happen in the morning such as swimming, endurance running and triathlons, golf, and crew.

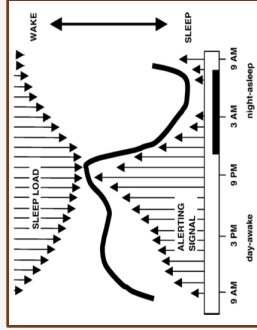
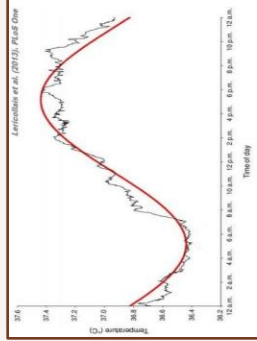
Optimal athletic conditions occur between 1600 and 2000 because core body temperature peaks during this window, however, performance is also enhanced if it takes place during the time at which it is trained. Additionally, morning-type individuals may be uniquely equipped for success shortly after waking.

Key Left to Right:

Borbély 2-process, CBT through the day. Alertness as a function of S and C.



Dijk, Homeostatic and Gender Regulation of the Sleep-Wake Cycle, 2016



Baker & Cajochen, Physiology and Behavior, 2007

2 – Considering Chronotype

- Larks and Owls have offset Process C arcs, creating different athletic preferences that go beyond just shifting earlier or later.
- Larks are better in the morning than their evening counterparts, but do not always show a deficit in the evening, perhaps benefitting from both their morning alertness and afternoon body temperature peak.
- Owls need significantly more time after waking to perform at their best and show reduced HRV recovery in the morning.

Thank you to Dr. Allison Brager and Dr. Saeitin for their mentorship throughout his project. A special thank you, as well, to Dr. Carskadon and the members of Bradley Sleep Lab -- particularly my fellow larks -- for engineering a strong research community and enjoyable summer!

3 – A New Age of Athletics

- A 21st century surge in sports culture and marketing means athletes are travelling across time zones more than ever.
- This is also true for college athletes who are simultaneously balancing intense schedules on top of getting an education.
- It is crucial in this new age for colleges and organizations to consider the effects of circadian misalignment not only on performance but on the quality of life of their athletes. Caffeine may offer a short-term aid.

Future directions

- Integrating Process S. Though sleep deprivation (SD) does not derail strength, successive nights of SD impede training. Furthermore, SD is known to cause severe cognitive deficits that may affect competition. Collegiate athletes are chronically sleep-deprived due to intense schedules and may be at greater risk for sleep disorders. Pro athletes are likely both misaligned and sleep-deprived from extensive travel.
- Integrating mindset. Larks endorse less perceived exertion in the morning despite no difference between owls in HR and distance walked. Larks' earlier circadian phase coupled with low sleep pressure makes them more alert in the mornings, which could be why they endorse more vigor and feel fresher at this time.

About Me

- I recently graduated from the University of Notre Dame with a BA in Psychology, spending two years in Dr. Jess Payne's Sleep, Stress, and Memory lab. I will be returning there for a gap year in the fall.
- I am deeply passionate about the many ways we can optimize our lives (particularly through our mental health), and I naturally gravitate to athletics.
- I want to give research-informed advice to my clients, so I saw the importance of obtaining a foundational understanding the fluctuations of AP throughout the human circadian cycle.

References

- Blatter, K. & Cajochen, C. (2007). Circadian rhythms in cognitive performance: Methodological constraints, protocols theoretical underpinnings. *Physiology & Behavior*, 90(2-3), 196-208.
- Dijk, D.-J. (2016). *Homeostatic and circadian regulation of the sleep-wake cycle* (PowerPoint presentation).
- Lericiollas, R., et al. (2013). Morning anaerobic performance is not altered by vigilance impairment. *PLOS ONE*, 8(3), e58638.

Sleep's Role in the Presentation and Treatment of PTSD in Veterans

Sarah McCarthy^{1,2}

- 1- Bradley Sleep Lab, Brown University
- 2- Department of Psychological and Brain Sciences, University of Massachusetts Amherst

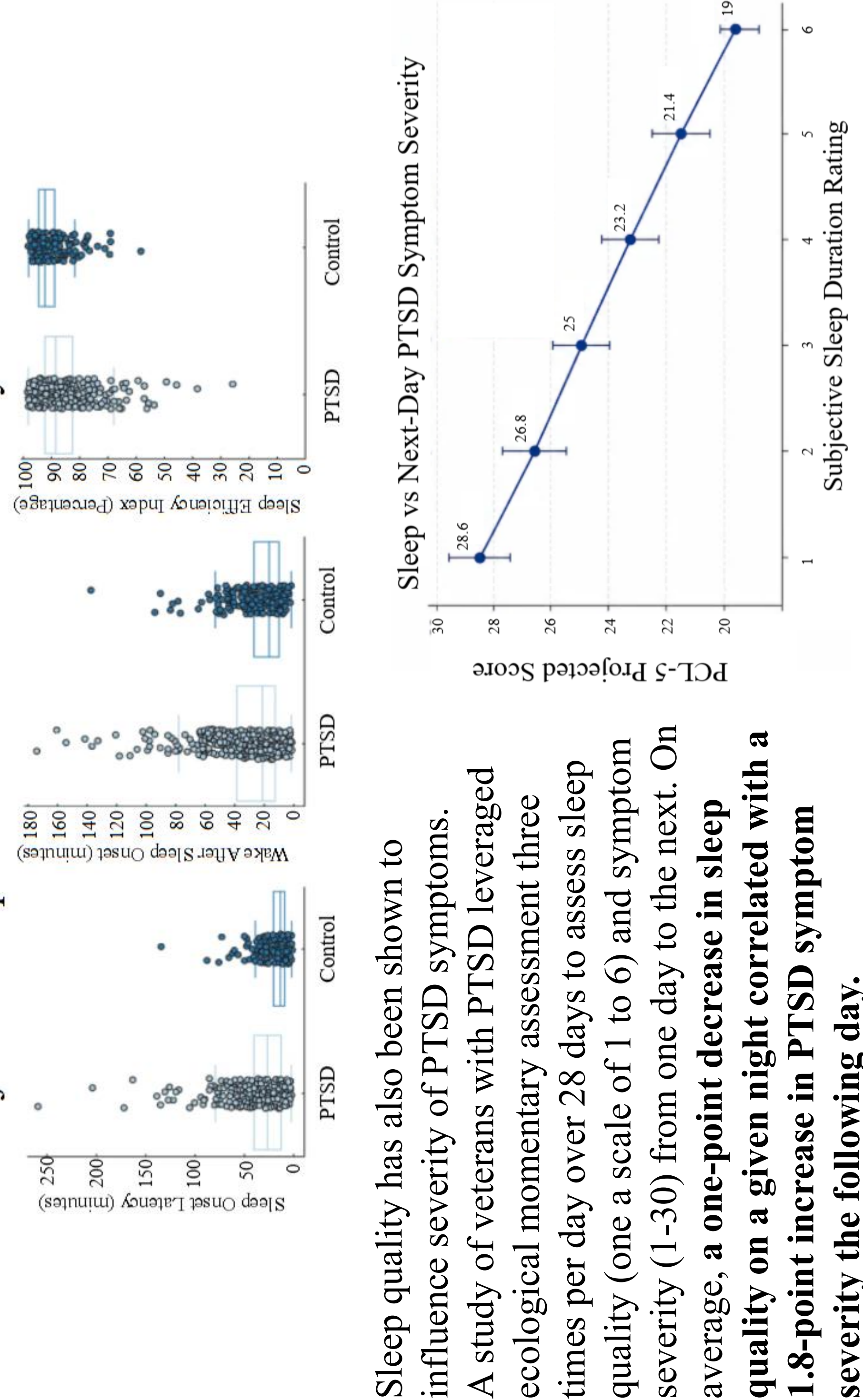
Introduction

- Post traumatic stress disorder (PTSD) is a condition that develops in response to witnessing a traumatic event and affects approximately 7% of veterans and over 20% of veterans who have deployed
- An estimated 27-54% of military personnel and veterans already struggle with insomnia, regardless of PTSD status
- Sleep issues are a core feature of PTSD; 70-91% of individuals with PTSD struggle to fall or stay asleep, and an estimated **90-100% of veterans with PTSD report sleep issues.**

Sleep and PTSD Bidirectionality

Subjective and objective sleep quality demonstrate an influence of PTSD on sleep quality. One study found objective measures and subjective reports of sleep to positively correlate across participants, and participants with PTSD had, on average, **+14 minutes sleep onset latency, +9 minutes wake after sleep onset, 6.6% lower sleep efficiency, significantly more N2 sleep, and more fragmented REM sleep.**

Objective Sleep Differences: PTSD vs Healthy Control



Sleep quality has also been shown to influence severity of PTSD symptoms. A study of veterans with PTSD leveraged ecological momentary assessment three times per day over 28 days to assess sleep quality (one a scale of 1 to 6) and symptom severity (1-30) from one day to the next. On average, **a one-point decrease in sleep quality on a given night correlated with a 1.8-point increase in PTSD symptom severity the following day.**

Sleep and PTSD Development

Sleep not only influences the severity of PTSD symptoms, but the development of PTSD itself. A 2021 study followed veterans without PTSD for 7 years and tracked their sleep and PTSD symptoms at 4 intervals. Those who reported fair or poor sleep at baseline were associated with a **60% increased risk for developing PTSD** compared to veterans rating their sleep as fairly good or good at baseline.

Poor sleep **prior to deployment** has also been shown to be a risk factor for PTSD development. One study found that sleep disturbances prior to deployment significantly predicted re-experiencing symptoms at 3 and 6 months post deployment.

Notable Complicating Comorbidities

Substance use disorder and depression commonly co-occur in individuals with PTSD and insomnia, with an estimated **68% of veterans with PTSD presenting with depression and 20% with substance use disorder.**

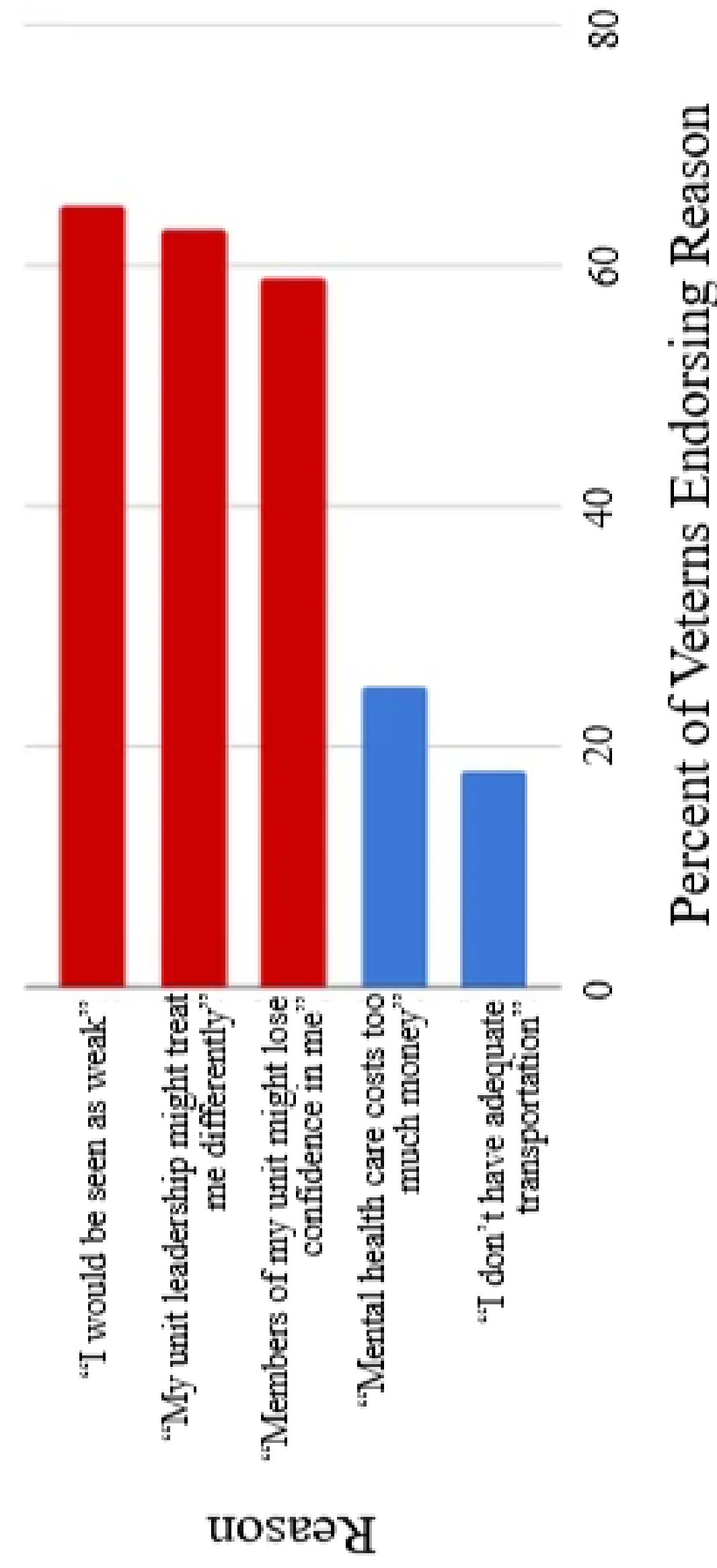
Major Depressive Disorder (MDD) has a bidirectional relationship with sleep similar to sleep and PTSD. Symptoms of MDD and PTSD can be similar and complicate one another by exacerbating symptoms that overlap, such as focus issues, reduced pleasure from daily activities, and irritability.

Substance use disorder often develops in veterans with PTSD when alcohol is used as a 'sleep aid' to reduce sleep onset latency, which individuals with PTSD often struggle with. There is a tradeoff, however, as alcohol prior to sleep has been shown to result in greater arousal later in the night which exacerbates REM fragmentation. This contributes to further emotional dysregulation in MDD and PTSD as well as more severe nightmares, another symptom of PTSD.

Stigma and Willingness to Seek Treatment

Stigma poses prominent barrier to mental health treatment in veterans. A 2004 study found stigma-related concerns among Army and Marine Corpsmen to be the **most commonly reported reasons for not seeking treatment**, even when veterans met clinical thresholds for treatment.

Reasons Veterans Did Not Seek Care



The Advantage of Sleep-Based Treatments

Sleep-based treatments are a promising solution to the stigma barrier. A 2018 study found that veterans were significantly more willing on a 4-point scale to seek treatment for insomnia than for depression or PTSD when presenting with comorbid insomnia and PTSD or comorbid insomnia and depression. Notably, veterans were also more willing to seek treatment for insomnia than for PTSD or depression **even when they did not have insomnia and did have PTSD or depression.**

Willingness to Seek Treatment by Condition

Positive Screen	Insomnia Treatment Willingness	PTSD or Depression Treatment Willingness
Insomnia + PTSD	2.89	2.68
Insomnia + Depression	2.87	2.62
PTSD	2.80	2.64
Depression	2.74	2.57

This finding is notable because a reduction in problematic sleep behaviors through sleep-based interventions not only improves sleep, but directly targets PTSD and MDD by reducing their textbook symptoms, and has the power to further reduce PTSD, MDD, and SUD severity by improving emotional regulation, memory, and ability to access coping skills with better sleep.

Sleep-Based Treatment Options

- Cognitive Behavioral Therapy for Insomnia: The gold standard for insomnia, improves PTSD symptoms directly and indirectly
- Cognitive Behavioral Therapy for Nightmares: Targets nightmares and improves PTSD symptoms.
- CBT-I and CBT-N with Other PTSD Treatments: Administering **both CBT-I/CBT-N and cognitive processing therapy (CPT)** has been shown to improve the outcomes of cognitive processing therapy for PTSD, regardless of order, with sleep therapies administered after CPT found as slightly more effective. CBT-I has also been shown to enhance **Prolonged Exposure (PE)** therapy.
- Targeted Memory Reactivation (TMR) to Enhance Encoding Consolidation
- PTSD Treatments: A study used auditory TMR to enhance Eye Movement Desensitization and Reprocessing (EMDR) Therapy. Therapeutic memories were cued during N2 sleep. The TMR group showed greater slow oscillation and spindle activity as well as reduced PTSD symptoms.

Significance

Better understanding the relationship between PTSD and sleep issues on both the objective and subjective scale, the common comorbid conditions occurring with PTSD and sleep issues, and primary barriers to treatment accessibility in veterans allow healthcare professionals to develop more effective intervention strategies and delivery methods for veterans with PTSD. Sleep offers a promising target for PTSD intervention in veterans for both its accessibility and its clinical relevance to the disorder.

Next Steps

Beyond further developing and disseminating sleep-based interventions for veteran with PTSD, I would suggest implementing prevention interventions in active military settings. Because poor sleep is not only a symptom but a driver of the severity and *development* of PTSD, equipping active-duty personnel with strategies to improve sleep their sleep in a hypervigilant and combat-readiness-heavy environment may not only directly improve performance, but prevent the later development of adverse mental health conditions.

About Me

I am a rising senior and member of the Commonwealth Honors College at the University of Massachusetts Amherst obtaining dual degrees in Psychology and Mathematics concentrated in Statistics.

My interest in this topic began growing up living between Vietnam veterans and learning about PTSD very young. I have always been intrigued by its ability to alter perception, memory, and emotion, and studying PTSD has grown more meaningful to me as I've gotten older and had numerous friends join the service.

I am graduating in December and plan to take a gap year before applying to PhD programs in clinical psychology. I plan to explore a career as an embedded clinical psychologist within the U.S. Navy, as the uniqueness of clinical care in that setting intrigues and excites me. During my gap year, I am interested in finding a research assistant position, completing a service mission related to providing healthcare, and putting more time into personal goals and hobbies including woodworking, yoga instructor certification, guitar, and driving cross country to see national parks.

References

Acknowledgements



This project would not have been possible without the insights and mentorship of Dr. John McCleary, and the guidance and feedback of Dr. Jared Saletin. Special thanks also to Dr. Mary A Carskadon for the opportunity to learn and grow throughout this fellowship.

How does the brain construct dream reality?

Ekaterina Michkovskaia

E.P. Bradley Sleep Research Lab, Mentors - Mary Carskadon, PhD, Jared Saletin, PhD, Karen Konkoly, PhD

Introduction

Dreams are subjective experiences that occur during sleep and require multidimensional neurobiological machinery. They perplex humans with their immersive, emotional, and bizarre nature; these traits can be attributed to various neural networks, ranging from visual processing and memory consolidation to emotional regulation and reality testing. Understanding the "dream anatomy" - how the brain makes up a dream – can offer insight into the underlying neurobiology and ultimately help discover the role of dreams.

Typical dream characteristics will be considered via the framework of "dream dimensions": **immersiveness**, **relevance**, and **creativity**

Visual and auditory aspects

Visual

Primary visual cortex V1 stays **deactivated** during REM, visual association cortex – **highly active**

Top-down activation: may be operating as a closed system (Braun, 1998); "virtual reality generator" (Hobson & Friston, 2012)

PGO waves from the brainstem may be interpreted as visual information

Auditory

Auditory impressions reported in 93.9% of collected dreams on average (Scarpelli, 2020)

Temporal cortex active during REM (Braun, 1997) – potentially the reason for hearing sounds in dreams

Vision and audition in wakefulness are stimulus-driven, in dreams - internally-driven

#immersiveness

Emotional aspects

75%-95% of dreams have emotional contexts (Hobson et al., 2000)

Maquet et al. (1996), Nature, PET study

- **Limbic hyperactivation** => emotional involvement
- **DLPFC hypofunction** => loss of working memory and reality-testing

Overactive limbic system and underactive dorsolateral prefrontal cortex make dreams emotional and their bizarreness goes unquestioned

#relevance #creativity

Memory sources

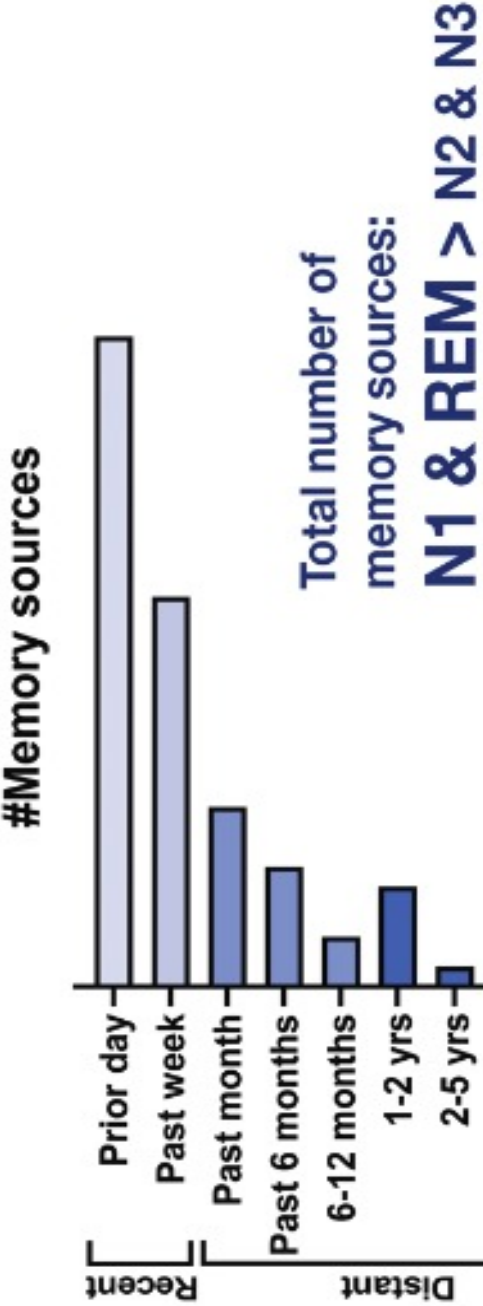
Role of the Hippocampus

- Damage to hippocampus – dreams **still present, but less coherent**
- Patients with **amnesia** - **fewer dreams** - less vivid, less detailed (Spanò, 2020)

Memory sources

More **recent memories** for **early night**, more **remote memories** - **later at night** (Picard-Deland, Konkoly, 2023)

Hippocampal replay: memory consolidation during REM/NREM involves **selective reactivation** of **recently encoded memories** (Ólafsdóttir, 2018)



Associations: Wakefulness/NREM - strong associations, dreams – **searching for weak associations** (Stickgold, 1999) => **exploring new knowledge**

Memory sources shape dream content, potentially because dreams help process memories

#relevance #immersiveness

Imagination in a dream

Imagery in wakefulness/dreams

- Ichikawa's "Imaginative model of dreaming" – dreams are not hallucinations, but mental imagery
- Model NOT supported: patients with **aphantasia** still had **vivid dreams**
- => **process of dreaming ≠ process of imagination** (Whiteley, 2021)

Coherency

- Consistency in personality and interests between wakefulness and sleep (Nir & Tononi, 2010) → no matter how weird, **we recognize ourselves in our dreams**

Reality monitoring

- "Reality threshold" for vividness and perceptual visibility is crossed → assume as reality (Dijkstra, 2023)
- **Right inferior parietal cortex** (Brodmann's Area 40) is important for voluntary control of action and thought
- This area is **deactivated** during REM sleep → less "analysis" of the situation, reduced self-awareness, altered reflective thought (Nir & Tononi, 2010)

Reality threshold in dreams is lowered, which makes dreams appear real

#relevance #creativity

Significance

The multidimensional nature of dream anatomy offers us insight into its complex role and challenges a common view of dreams as "random neural signals". The intricacies of dream reality suggest that these experiences are **beyond being a useless byproduct of brain activity**.

Exploring the biological, cognitive, and psychosocial aspects of dreams can help us gain insight into their significance, as well as help **define the concept of human consciousness**.

Discussion and future directions

- Purposes of dreaming can be explained by the **NEXTUP model** (Stickgold & Zadra, 2021): the brain is searching and manufacturing distant and unusual connections between memories, exploring potential scenarios

Next steps

- Empirical **evidence for dreaming imagination** model
- Comparing imagery in wakefulness and sleep
- Understanding of **top-down activation** significance and benefits, as well the bigger **purpose of dreaming**
- Potential for intervention – **dream engineering**

About me

- Rising junior at Brown University studying **Neuroscience & Literary Arts**
- Volunteer at E.P. Bradley Sleep Research Lab

Why I chose this topic?

- Strong interest in **dream research**
- To help me understand the "anatomy of a dream"
- This question was raised in "When brains dream" by Dr. Stickgold & Dr. Zadra

My future

- PhD in Neuroscience with a specialization in dreams

References

1. Dijkstra, N., & Fleming, S. M. (2023). Subjective signal strength distinguishes reality from imagination. *Nature Communications*, 14(1). <https://doi.org/10.1038/s41467-023-37322-1>
2. Ruby, P. M. (2020). The neural correlates of Dreaming have not been identified yet. Commentary on "the neural correlates of Dreaming. *Nat neurosci*. 2017." *Frontiers in Neuroscience*, 14. <https://doi.org/10.3389/fnins.2020.5685470>
3. Siclari, F., Baird, B., Perogamvros, L., Bernardi, G., LaRoque, J. J., Riedner, B., ... Tononi, G. (2017). The neural correlates of Dreaming. *Nature Neuroscience*, 20(6), 872–878. <https://doi.org/10.1038/nrn.4545>
4. Tsunematsu, T. (2022). What are the neural mechanisms and physiological functions of dreams? *Neuroscience Research*, 169(169). <https://doi.org/10.1016/j.neures.2022.12.017>
5. Wamsley, E. J. (2020). How the brain constructs dreams. *eLife*, 9(9). <https://doi.org/10.7554/eLife.58874>

Sleep Deprivation and Immune Dysregulation: Mechanisms Linking Sleep Loss to Chronic Inflammation

Katherine Mitchell, Mary A. Carskadon, PhD., Jared M. Sadein, PhD. | E.P. Bradley Sleep For Science Research Laboratory, Brown University Health

INTRODUCTION

Sleep is increasingly recognized as a critical regulator of immune function. While acute sleep loss may temporarily activate protective immune responses, chronic sleep deprivation and fragmentation promote persistent low-grade inflammation through activation of the HPA axis, sympathetic nervous system, and pro-inflammatory pathways. These changes are associated with increased susceptibility to infection and the development of cardiovascular, autoimmune, metabolic, and neurodegenerative diseases. This review examines the mechanisms linking insufficient sleep to immune dysregulation and highlights sleep as an important determinant of long-term health.

SLEEP & IMMUNE HOMEOSTASIS

Sleep and the immune system communicate bidirectionally. Immune activation influences sleep, while adequate sleep supports immune homeostasis and insufficient sleep promotes immune dysregulation (Figure 1).

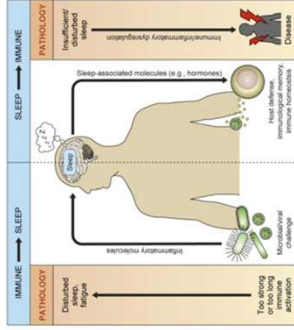


Figure 1.

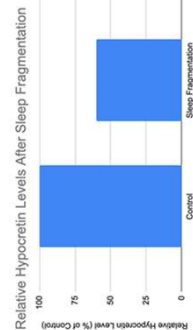


Figure 3a. Decreased hypocretin signaling.

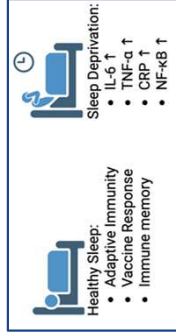


Figure 2. Healthy sleep promotes optimal immune mechanisms, whereas sleep deprivation increases pro-inflammatory signaling and inflammatory biomarkers.

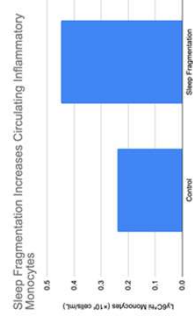


Figure 3b. Increased inflammatory monocytes.

SLEEP FRAGMENTATION & INFLAMMATION

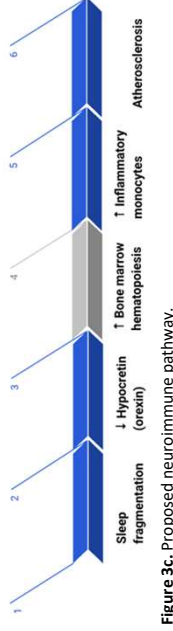


Figure 3c. Proposed neuroimmune pathway.

SLEEP DURATION & INFECTION RISK

Shorter Sleep Duration Is Associated With Greater Cold Susceptibility

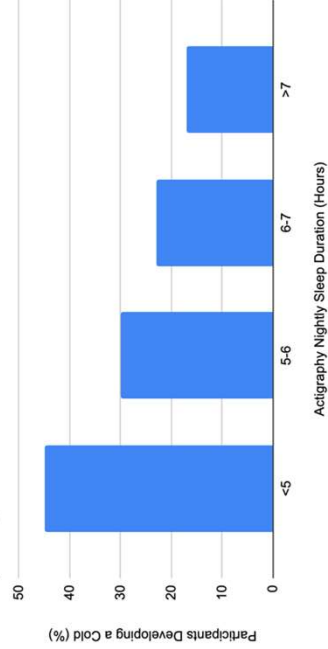


Figure 4. Human studies demonstrate that individuals sleeping <5 hrs/night are 2.6x more likely to develop symptomatic viral infections than those sleeping >7 hrs.

DISCUSSION & CONCLUSIONS

Growing evidence demonstrates that sleep is a fundamental regulator of immune function and is essential for maintaining effective immune defense. Chronic sleep deprivation promotes immune dysregulation through inflammatory signaling pathways, increasing susceptibility to infection and chronic disease. Improving sleep may therefore represent an accessible strategy for reducing inflammation and promoting long-term health.

CONCLUDING MODEL

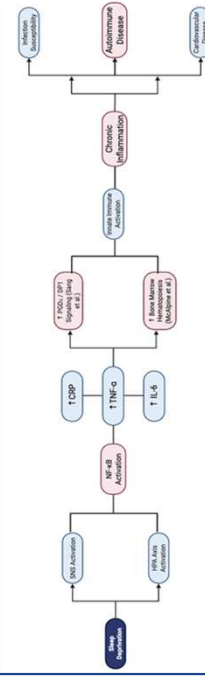


Figure 5. Proposed model summarizing mechanisms linking sleep deprivation, immune dysregulation, and disease. Made with Biorender.

FUTURE DIRECTIONS

- Define additional neuroimmune pathways linking sleep and inflammation.
- Determine whether improving sleep reverses chronic immune dysregulation.
- Identify biomarkers that predict disease risk associated with chronic sleep loss.
- Develop sleep-based interventions to reduce inflammatory disease burden.

AUTOBIOGRAPHY - ABOUT ME

- B.S. Microbiology and Immunology - University of Rochester
- Summer Dement Fellow
- Interested in how sleep affects immune regulation and chronic inflammation
- Sleep for Science Lab Research Assistant

REFERENCES

- 1) Besedovsky, L., Lange, T., & Haack, M. (2019). The Sleep-Immune Crosstalk in Health and Disease. *Physiological reviews*, 99(3), 1325–1380. <https://doi.org/10.1152/physrev.00010.2018>
- 2) Team, DDW. "Monitoring the Balance between Immune Repression and Stimulation in Cancer Immunotherapy." *Drug Discovery World (DDW)*, 13 Oct. 2020. www.ddw-online.com/maintaining-the-balance-between-immune-repression-and-stimulation-in-cancer-immunotherapy-1370202759/.
- 3) Sang et al. (2023). Prolonged sleep deprivation induces a cytokine-storm-like syndrome in mammals. *Cell*, 186(25), 4500–4516.e21. <https://doi.org/10.1016/j.cell.2023.10.025>
- 4) Colquhoun et al. (2019). Sleep modulates hematopoiesis and protects against atherosclerosis. *Nature*, 566(7744), 385–387. <https://doi.org/10.1038/s41586-019-0485-2>
- 5) Truller et al. (2015). Behaviorally Assessed Sleep and Susceptibility to the Common Cold. *Sleep*, 38(9), 1353–1359. <https://doi.org/10.5665/sleep.5400>
- 6) Urban M R. (2019). Sleep and inflammation: partners in sickness and in health. *Nature reviews. Immunology*, 19(11), 702–715. <https://doi.org/10.1038/s41585-019-0193-7>

PEDIATRIC SLEEP, RHYTHMS,
AND MENTAL HEALTH

Sleep for Science
RESEARCH LABORATORY
OF BROWN UNIVERSITY

- Female healthcare workers (N = 51) completed overnight shifts under five conditions: **no food, one or three low-GI meals, or one or three high-GI meals**. Overall vigilance did not significantly differ between the **no-food and meal conditions**. However, **three high-GI meals produced the most attentional lapses**:

- **4.6 more lapses** than one low-GI meal
- **3.4 more lapses** than three low-GI meals
- Not eating increased hunger, suggesting that **low-GI meals may offer a more practical compromise** than repeated high-GI eating.

● Healthy adults ($N = 19$) completed simulated night work with nighttime or daytime-only eating.

● Nighttime eating shifted the glucose rhythm **9.81 h** versus **0.57 h** with daytime eating ($P < .001$).

● Post-meal glucose increased **19.4%** with nighttime eating ($P = .002$)

● **Daytime-only eating preserved internal circadian alignment** and prevented worsening glucose tolerance.

Internal circadian misalignment

Internal circadian alignment

Night shift work
Night eating 8 PM - 1 AM
Day eating 8 AM - 11 AM

Night shift work
Night work 8 PM - 1 AM
Day eating 8 AM - 11 AM

- Physician trainees ($N = 61$) completed real overnight hospital shifts under usual-eating and structured nutrition conditions.
- With usual eating, vigilance declined **32.1 points**; with the lower carbohydrate-to-protein intervention, it improved **18.9 points**.
- **Difference: 51.02 points, $P = .011$.**
- Timing, meal composition, hydration, and nutrition guidance changed together, so the active component is unclear

- Healthy adults (**N = 10**) completed four simulated night shifts.
- One group consumed a meal providing **40% of daily energy**, while the other group did not eat overnight.

- **Timing matters most:** daytime-only eating preserved circadian alignment and glucose tolerance during simulated night work.
- **Amount also matters:** fasting best preserved glucose tolerance, while impairment increased from a small snack to a full nighttime meal.
- Large nighttime meals and repeated high-GI eating were least favorable for vigilance.
- When fasting is unrealistic, a **small, planned, lower-GI meal earlier in the shift** may offer a practical compromise.

NEXT STEPS

- Measure metabolic health and performance together during actual overnight healthcare shifts
- Compare no food during the shift with one small, lower-GI meal earlier in the shift and a calorie-matched higher-GI meal later in the shift
- Measure glucose regulation, vigilance, hunger, gastrointestinal comfort, and adherence
- Control meal timing, meal size, caffeine, hydration, sleep, and workload to isolate the effect of meal composition

ABOUT ME

- Brown University student studying Public Health and Health and Human Biology
- Interested in how sleep and circadian rhythms shape health and behavior

- Overnight workers often cannot control when they work but food timing, meal size, and composition are potentially modifiable

- I wanted to understand whether chrononutrition could help preserve metabolic health while still supporting hunger control and alertness

- I plan to pursue medicine and research focused on improving care for underserved communities

- Chellappa SL, et al. *Science Advances*. 2021.
- Centofanti S, et al. *Diabetologia*. 2025.
- Grant CL, et al. *Industrial Health*. 2017.
- de Rijk MG, et al. *The Journal of Nutrition*. 2024.
- Makowski MS, et al. *Academic Medicine*. 2022.
- Cheng WJ, et al. *PLoS ONE*. 2021.
- Barrañán R, et al. *Journal of Clinical Sleep Medicine*. 2023.
- Marot LP, et al. *Nutrients*. 2023.
- Revior-González C, et al. *Nutrients*. 2025.
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Figure 1. Nighttime melatonin levels in the circadian decline in vigilance. A large nighttime melatonin adds to the circadian decline in vigilance. A large nighttime melatonin adds to the circadian decline in vigilance. A large nighttime melatonin adds to the circadian decline in vigilance.



The Role of Slow-Wave Sleep in Alzheimer's Dementia Risk

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INTRODUCTION

- Alzheimer's disease (AD) is the most common type of dementia and marked by a buildup of amyloid- β (A β) and tau tangles, leading to impaired cell signaling and cell death.
- Time spent in slow-wave sleep (SWS) decreases with age, and this decline correlates with rising AD pathology¹.
- The glymphatic system clears waste more efficiently during non-REM SWS and is compromised when this sleep stage is disrupted^{2,3,4}.
- This has led to a hypothesized mechanism linking SWS loss to AD pathology: reduced glymphatic clearance related to SWS loss allows amyloid- β and tau to accumulate over time, potentially accelerating disease progression^{5,6,7}.

RESEARCH QUESTIONS

- Do changes in sleep predict the future development of AD?
- What effect does SWS have on cognition and AD pathology?
- What role does the glymphatic system play in SWS and amyloid- β accumulation?

SLOW WAVE SLEEP AND AD PATHOLOGY

Ju et al. (2017)

- Disrupted SWS overnight using acoustic tones without shortening sleep duration⁵.
- CSF A β rose significantly after the SWS-disrupted night. Poor home sleep quality over the baseline period of 6 nights was associated with increased CSF tau levels⁵.

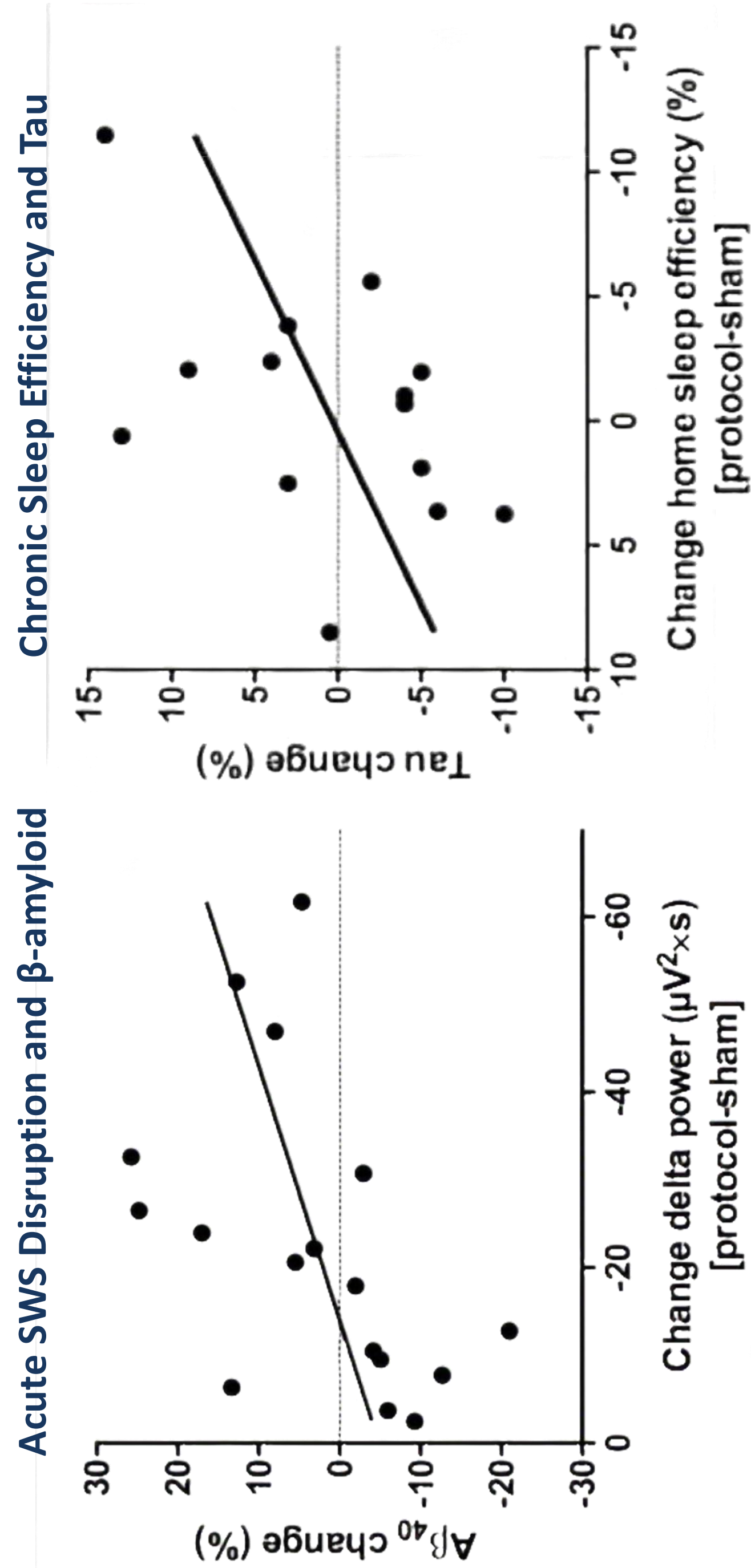


Fig. 1. Decreased slow-wave activity, measured via EEG delta spectral power, was correlated with increased amyloid- β ₄₀ ($r = 0.610$, $P = 0.009$). Adapted from Ju et al. (2017).

Fig. 2. Poorer sleep efficiency, measured actigraphically over 6 nights, predicted greater tau levels ($r = 0.543$, $P = 0.045$). Adapted from Ju et al. (2017).

Mander et al. (2015)

- Measured A β burden alongside SWS activity and memory recall of word pairs⁶.
- Higher A β was linked to decreased SWS activity and worse memory, pointing to a two-way relationship⁶.

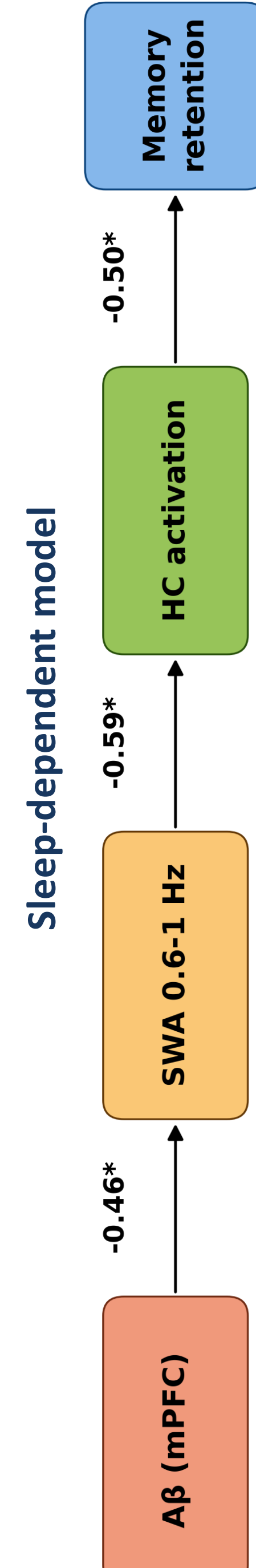


Fig. 3. Path analysis linking mPFC β -amyloid deposition (via PIB-PET), NREM slow-wave activity (0.6–1 Hz) in mPFC, and hippocampal activation during retrieval to overnight memory retention. Adapted from Mander et al. (2015).

SLOW WAVE SLEEP AND AD PATHOLOGY, CONTINUED

Himali et al. (2023)

- Measured SWS decline over 5 years and tracked dementia incidence over 17 years of follow-up in 346 adults (mean age 69 years, range 60-87 years)⁷.
- Each 1% annual decline in SWS was associated with a 27% increase in all-cause dementia and a 32% increase in AD risk**, independent of APOE ϵ 4, age, sex, and other cohort factors⁷.
- However, preclinical AD can begin up to 25 years before symptoms, so disrupted SWS may be a consequence of early pathology rather than a cause¹.

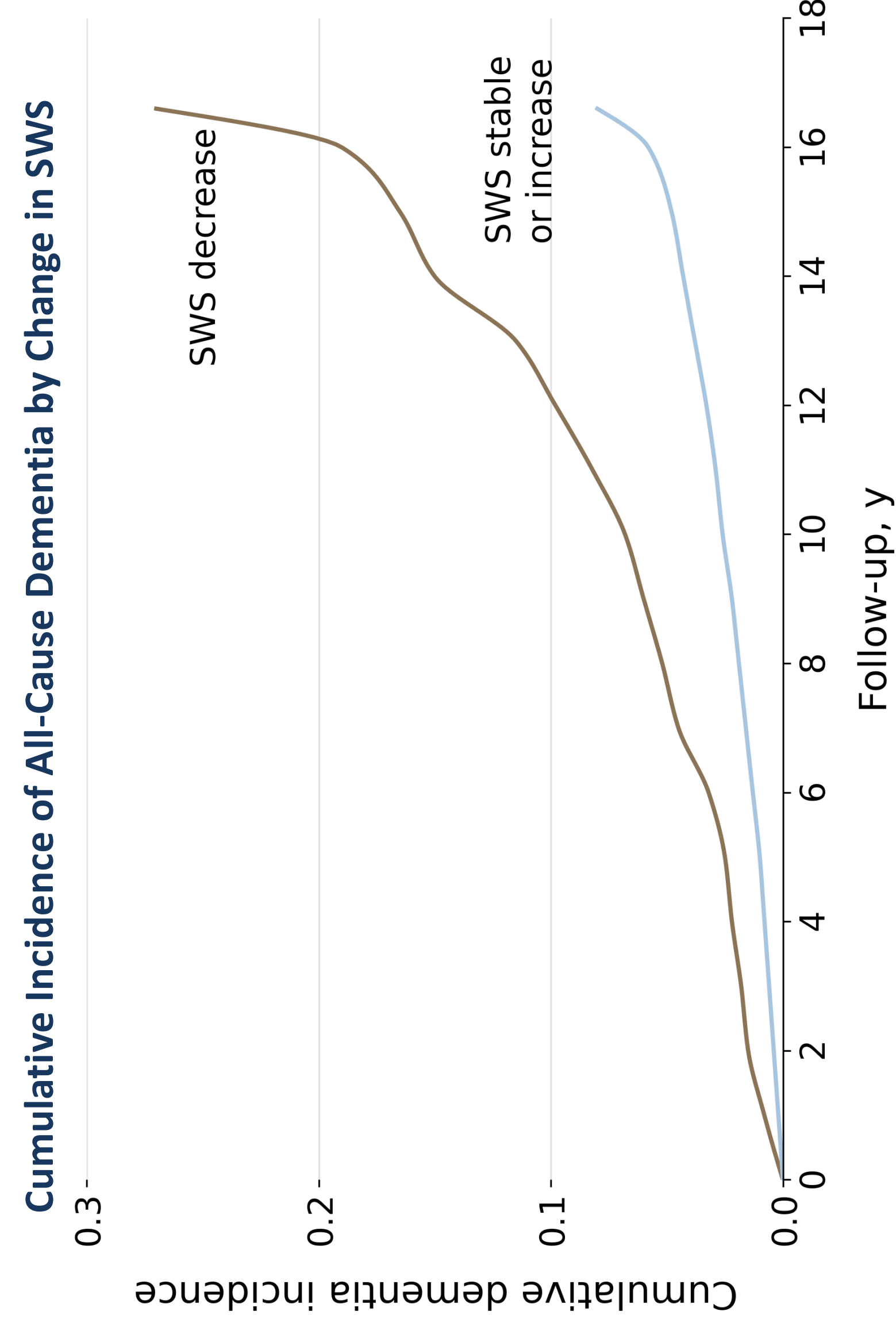


Fig. 4. Cumulative dementia incidence by SWS trajectory over 17-year follow-up. Adapted from Himali et al. (2023).

GLYMPHATIC CLEARANCE: A HYPOTHESIZED MECHANISM

The glymphatic system is a CSF-driven pathway. CSF enters brain parenchyma via periaxial spaces, mixes with interstitial fluid (ISF), and exits through perivenous spaces, clearing metabolic waste, including A β and tau².

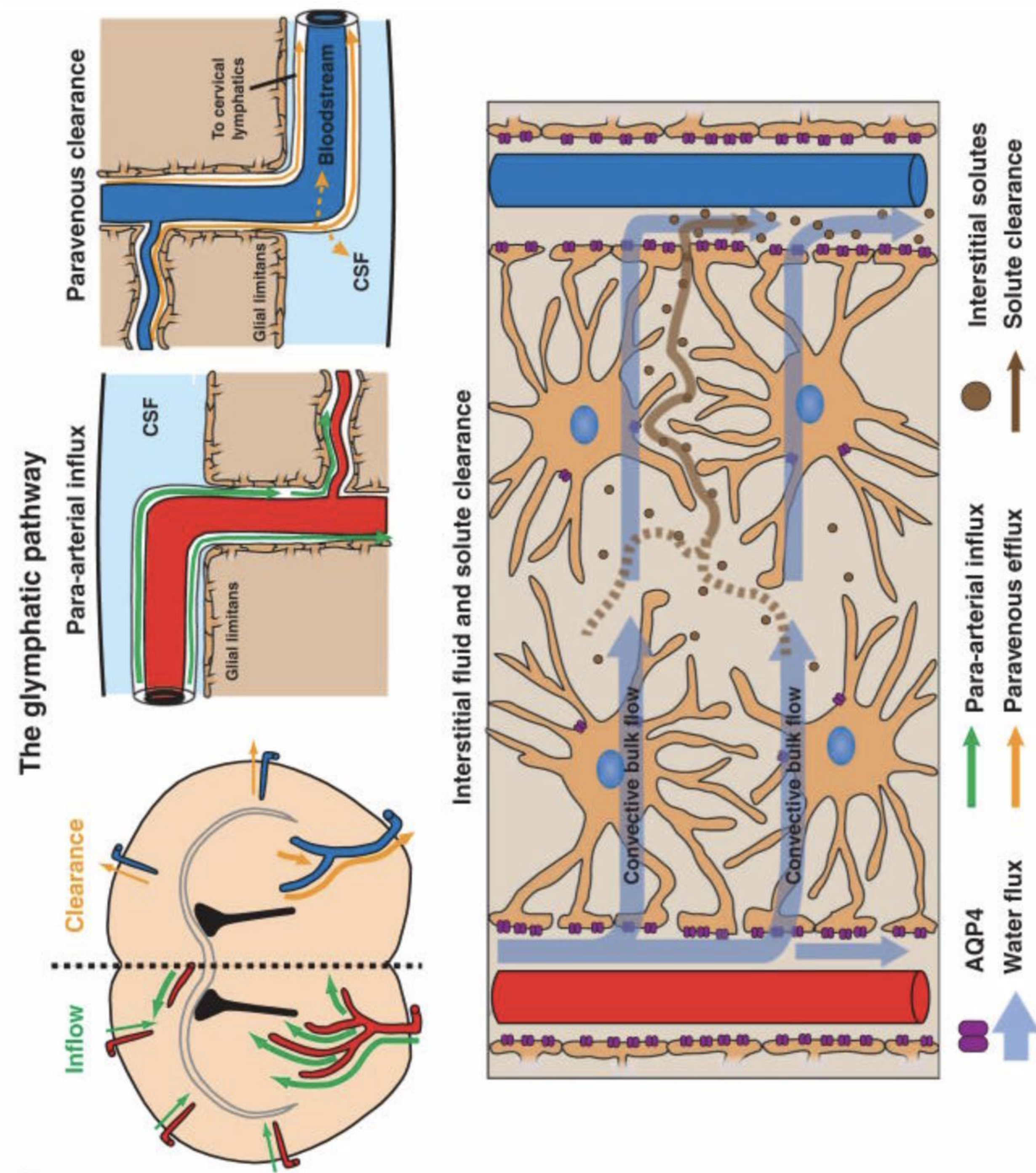


Fig. 5. The glymphatic pathway: CSF enters via para-arterial spaces and is driven across the parenchyma by AQP4-dependent convective flow at astrocytic end feet, clearing interstitial solutes along perivenous routes. Adapted from Iliff et al. (2012).

GLYMPHATIC CLEARANCE, CONTINUED

- The glymphatic system is far more active during sleep than wake. Interstitial space expands by 60%, increasing the exchange rate between CSF and ISF².
- NREM slow waves are followed by oscillations in cerebral blood volume and CSF flow, localizing clearance to this stage specifically³.
- When SWS is disrupted, this mechanism is compromised, allowing A β to accumulate⁴. This impairment may worsen future sleep and clearance, creating a self-reinforcing cycle⁹.
- Independent of sleep, glymphatic disruption, measured by MRI (DTI-ALPS), predicts early A β buildup and progression toward AD¹⁰. This underscores its role as a key link between SWS loss and dementia.

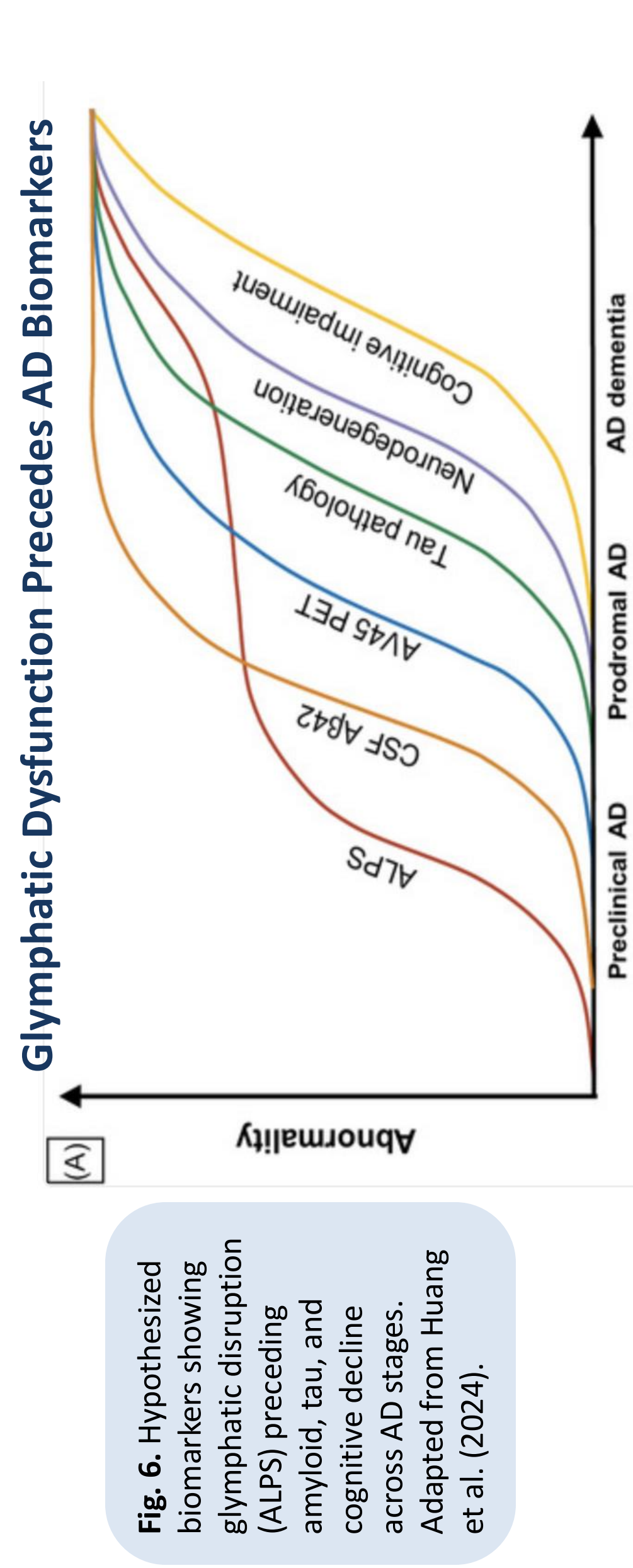


Fig. 6. Hypothesized biomarkers showing glymphatic disruption (ALPS) preceding amyloid, tau, and cognitive decline across AD stages. Adapted from Huang et al. (2024).

TAKEAWAYS

- As SWS declines with age, the glymphatic system clears waste less efficiently, contributing to a buildup of AD pathology.
- While SWS loss predicts dementia risk, AD pathology may also disrupt SWS, suggesting the relationship may be bidirectional.
- Glymphatic failure itself predicts AD progression, underlining it as a critical downstream link.

Limitations: No single factor explains dementia risk; the glymphatic pathway is one proposed mechanism. Age, vascular health, genetic risk, and other factors likely interact rather than forming a linear causal chain. Additionally, glymphatic clearance cannot be directly measured in humans. Rodent studies used to map the glymphatic system may not reflect a human model.

Future Directions: Large scale longitudinal studies paired with more direct measures of glymphatic clearance are needed to establish the causal link between SWS, glymphatic disruption, and AD progression.

AUTOBIOGRAPHY – WHY I CHOSE THIS TOPIC

During SLEEP 2026, I attended discussions on the relationship between sleep disruption, cognitive impairment, and neurodegenerative disorders. I was surprised to learn the many factors that can contribute to AD and how much research still has to answer.

- 2026 graduate from the University of South Carolina
- Majorored in Neuroscience and worked USC's Sleep Research Lab under Dr. Alex Reynolds.
- My Future:
 - Currently taking a gap year while applying to medical school, hoping to specialize in neurology or psychiatry.

REFERENCES

- Ferrini-Strambi, L., Liguori, C., Lucey, B. P., et al. Role of sleep in neurodegeneration: The consensus report of the 5th Think Tank World Sleep Forum. *Neurological Sciences*, 45, 749–759 (2024). <https://doi.org/10.1007/s00221-023-02327-7>
- Iliff, M. J., Wang, M., Xie, L., et al. A paravascular pathway facilitates CSF flow through the brain parenchyma and the clearance of interstitial solutes, including amyloid β . *Science Translational Medicine*, 4(147), 147ra111. <https://doi.org/10.1126/scitranslmed.3003748>
- Xie, L., Kang, H., Xu, Q., Chen, M. J., Liao, Y., Thiagarajan, M., O'Donnell, J., Christensen, D. J., Nicholson, C., Iliff, J. J., Takano, T., Deane, R., & Nedergaard, M. (2013). Sleep drives metabolite clearance from the adult brain. *Science*, 342(6150), 373–377. <https://doi.org/10.1126/science.1241224>
- Fultz, N. E., Bommasir, G., Selkoepp, K., Stickgold, R., Rosen, B. R., Pollmeier, J. R., & Lewis, L. D. (2019). Coupled electrophysiological, hemodynamic and cerebrospinal fluid oscillations in human sleep. *Science*, 366(6465), 628–631. <https://doi.org/10.1126/science.aax5440>
- Ju, Y.-S., Owens, S. J., Sudiparti, S., Mander, B. A., Ruppel, S. L., Ruppel, J. M., Glasson, J. A., R., & Holtzman, D. M. (2017). Slow wave sleep promotes glymphatic clearance of amyloid- β and tau in the mouse brain. *Journal of Neuroscience*, 37(48), 14081–14091. <https://doi.org/10.1523/JNEUROSCI.0648-17.2017>
- Mander, B. A., Marks, S. M., Vogel, J. W., et al. (2015). β -amyloid disrupts human NREM slow waves and related hippocampus-dependent memory consolidation. *Nature Neuroscience*, 18, 1051–1057. <https://doi.org/10.1038/nn.4035>
- Himali, J. J., Baril, A. A., Cavuto, M. G., Vialouros, S., Wiedner, C. D., Himali, D., DeCarli, C., Redline, S., Belser, A. S., Sachdev, S., & Pase, M. P. (2023). Association between slow wave sleep loss and incident dementia. *JAMA Neurology*, 80(12), 1326–1333. <https://doi.org/10.1001/jamaneuro.2023.3889>
- Fide, P. K., Vinje, V., Prippe, A. H., Mardai, K.-A., & Ringstad, G. (2021). Sleep deprivation impairs molecular clearance from the human brain. *Brain*, 144(3), 865–874. <https://doi.org/10.1093/brain/awaa443>
- Huang, S.-Y., Zhang, X.-R., Guo, Y., et al. (2024). Glymphatic failure as a final common pathway to dementia. *Science*, 370(6512), 59–56. <https://doi.org/10.1126/science.abb729>
- Huang, S.-Y., Zhang, X.-R., Guo, Y., et al. (2024). Glymphatic system dysfunction predicts amyloid deposition, neurodegeneration, and clinical progression in Alzheimer's disease. *Alzheimer's & Dementia*, 32(5), 3253–3269.