BIOGRAPHICAL SKETCH

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NAME: Plante, David Thomas

eRA COMMONS USER NAME (credential, e.g., agency login): dplante

POSITION TITLE: Associate Professor of Psychiatry

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Swarthmore College, PA	B.A.	05/2000	Biology and Women's Studies
University of North Carolina School of Medicine, NC	M.D.	05/2004	Medicine
Massachusetts General Hospital & McLean Hospital, MA		06/2008	Adult Psychiatry (Residency)
Brigham & Women's Hospital, MA		06/2010	Sleep Medicine (Fellowship)
University of Wisconsin-Madison, WI	Ph.D.	05/2018	Clinical Investigation

A. Personal Statement

I am an Associate Professor of Psychiatry and Sleep Medicine. I am currently the Medical Director of Wisconsin Sleep and the Program Director of the UW-Madison clinical Sleep Medicine fellowship program. My research focus has been at the intersections of sleep and neuropsychiatric disorders, with specific emphasis on hypersomnolence in non-cataplectic disorders of central hypersomnolence. As a clinician-scientist in sleep medicine, I have expertise in polysomnography, advanced EEG analysis techniques, wearable technologies, and objective measures used to quantify various aspects of sleep phenotypes. I have served on the American Academy of Sleep Medicine Scoring Manual Editorial Board starting in 2017, most recently as Vice Chair.

B. Positions and Honors

Positions and Employment

2004-2008 Clinical Fellow in Psychiatry, Harvard Medical School, Boston, MA
2008-2010 Clinical Fellow in Sleep Medicine, Brigham and Women's Hospital, Boston, MA
2010-2014 Assistant Professor (CHS), Department of Psychiatry, University of Wisconsin School of Medicine and Public Health, Madison, WI
2014-2021 Assistant Professor (Tenure Track), Department of Psychiatry, University of Wisconsin School of Medicine and Public Health, Madison, WI
2017- Medical Director, Wisconsin Institute for Sleep and Consciousness
2021- Associate Professor (Tenure Track), Department of Psychiatry, University of Wisconsin School of Medicine and Public Health, Madison, WI

Other Experience and Professional Memberships

2004-2022 Member, American Psychiatric Association
2004-2010 Member, Massachusetts Psychiatric Society
2008-2010 Member, Massachusetts Sleep Medicine Society
2011-2022 Member, Wisconsin Psychiatric Society
2004- Member, American Academy of Sleep Medicine
2011- Member, Sleep Research Society

Honors

1996	Tandy Technology Scholar
2000	Phi Beta Kappa, Swarthmore College
2002	Academic Merit Award, UNC School of Medicine
2004	Fordham Leadership Award, UNC School of Medicine
2004	Alpha Omega Alpha, UNC School of Medicine
2008	Laughlin Foundation Merit Award, Massachusetts General Hospital Department of Psychiatry
2009	American Sleep Medicine Foundation Young Investigator Travel Award
2012	Sleep Research Society Young Investigator Award
2014	Chairman's Choice Travel Award, Society of Biological Psychiatry
2015	Biomarker Trainee Travel Award, Sleep Research Society
2015	Travel Award, U13 Conference: Sleep, Circadian Rhythms, and Aging: New Avenues for Improving Brain Health, Physical Health, and Functioning
2018	Phi Kappa Phi, University of Wisconsin-Madison
2020	Sigma Xi, University of Wisconsin-Madison

C. Contribution to Science

1. *Hypersomnolence in Mood Disorders*

My most prominent research efforts have focused on the evaluation of excessive daytime sleepiness and sleep duration in depressive illness. Work from my lab, as well as research collaborations conducted with the Wisconsin Sleep Cohort Study, have underscored the complex and often paradoxical relationships between subjective and objective measures of hypersomnolence in mood disorders. In addition, results from this work suggest that physiological changes in the brain associated with excessive sleepiness cut across traditional diagnostic boundaries, and may be widely applicable to a number of central nervous system disorders.

a. **Plante DT**, Finn LA, Hagen EW, Mignot E, Peppard PE. Subjective and objective measures of hypersomnolence demonstrate divergent associations with depression among participants in the Wisconsin Sleep Cohort Study. J Clin Sleep Med. 2016; 12(4):571-8. PMCID: PMC 4795285.

b. **Plante DT**, Cook JD, Goldstein MR. Objective measures of sleep duration and continuity in major depressive disorder with comorbid hypersomnolence: a primary investigation with contiguous systematic review and meta-analysis. J Sleep Res. 2017; 26(3):255-65. PMCID: PMC5435536.

c **Plante DT**, Birn RM, Walsh EC, Hoks RM, Cornejo MD, Abercrombie HC. Reduced resting-state thalamostriatal functional connectivity is associated with excessive daytime sleepiness in persons with and without depressive disorders. J Affect Disord. 2018; 227:517-20. PMCID: PMC5805569.

d. **Plante DT**, Cook JD, Barbosa LS, Goldstein MR, Prairie ML, Smith R, Riedner BA. Establishing the objective sleep phenotype in hypersomnolence disorder with and without comorbid major depression. SLEEP. 2019; 42(6): zsz060. PMCID: PMC6559176.

2. *EEG Alterations in Mood Disorders*

When I joined the faculty at the University of Wisconsin-Madison (UW) in 2010, my research initially focused on incorporating hdEEG methodologies in the study of sleep pressure in healthy and disordered populations. Initial studies focused on evaluation of slow waves and overnight changes in spontaneous waking EEG in unipolar major depressive disorder as markers of sleep homeostatic function. These studies demonstrated topographic and sex-related changes in slow wave activity, with women with depression having significantly higher absolute slow wave activity than other comparison groups. In addition, patients with depression did not demonstrate overnight decline in spontaneous waking EEG or changes in auditory evoked potentials, nor correlations of frontal waking EEG activity with slow wave activity, which were evident in healthy controls. These data extend prior work that suggests sleep homeostasis is aberrant in depression. Moreover, because mood disorders consist of heterogeneous sleep-related complaints, these investigations led to a pilot study that examined hdEEG in patients with depression who complained of hypersomnia. Preliminary findings suggested that these patients may have blunted slow wave activity and increased waking EEG slowing, which were originally posited to serve as a biomarker for sleepiness and target for treatment in the disorder. These findings were the cornerstone for a Mentored Career Development Award from NIMH.

a. **Plante DT,** Landsness EC, Peterson MJ, Goldstein MR, Riedner BA, Wanger T, Guokas JJ, Tononi G, Benca RM. Sex-related differences in sleep slow wave activity in major depressive disorder: A high-density EEG investigation. BMC Psychiatry, 2012; 12(1): 146. PMCID: PMC3507703

b. **Plante DT,** Landsness EC, Peterson MJ, Goldstein MR, Wanger T, Guokas JJ, Tononi G, Benca RM. Altered slow wave activity in major depressive disorder with hypersomnia: a high density EEG pilot study. Psychiatry Research: Neuroimaging. 2012; 201(3):240-4. PMCID: PMC3361575
c. Goldstein MR, **Plante DT**, Hulse BK, Sarasso S, Landsness EC, Tononi G, Benca RM. Overnight changes in waking auditory evoked potential amplitude reflect altered sleep homeostasis in major depression. Acta Psychiatrica Scand. 2012; 125(6):468-77. PMCID: PMC3303968
d. **Plante DT,** Goldstein MR, Landsness EC, Riedner BA, Guokas JJ, Wanger T, Tononi G, Benca RM. Altered overnight modulation of spontaneous waking EEG reflects altered sleep homeostasis in major depressive disorder: a high-density EEG investigation. J Affective Disord, 2013; 150(3):1167-73. PMCID: PMC3760229

3. *Effects of GABA-A Agonists on Sleep EEG*

In the process of developing workflows and processes in my laboratory at UW to analyze sleep and wake EEG data, I explored specific hypotheses regarding the effects of endogenous and exogenous agonists of the GABA-A receptor on sleep EEG. Utilizing EEG from clinical polysomnography collected at Wisconsin Sleep, the sleep lab affiliated with UW, we demonstrated medroxyprogesterone was associated with increased sleep spindle activity and number in women. However, finasteride, which inhibits 5α-reductase, a key enzyme in the conversion of central neurosteroids into compounds with GABA-A activity, was not associated with changes in sleep spindles in men, suggesting this pathway is not likely involved in the known psychiatric effects of this medication. Beyond analysis of standard EEG montages in these clinical datasets, we evaluated changes in slow waves and sleep spindles resulting from temazepam using high-density EEG. These results extended the prior literature by demonstrating that the effects of benzodiazepines on sleep EEG waveforms vary markedly depending on topographic location as well as method of analysis used to analyze sleep EEG.

a. **Plante DT**, Goldstein MR. Medroxyprogesterone acetate is associated with increased sleep spindles during non-rapid eye movement sleep in women referred for polysomnography. Psychoneuroendocrinology, 2013; 38(12):3160-6. PMCID: PMC3844048
b. Goldstein MD, Cook JD, **Plante DT**. The 5α-reductase inhibitor finasteride is not associated with alterations in sleep spindles in men referred for polysomnography. Hum Psychopharmacol 2016; 31(1):70-4. PMCID: PMC4718775
c. **Plante DT**, Goldstein MR, Cook JD, Smith R, Riedner BA, Rumble ME, Jelenchick L, Roth A, Tononi G, Benca RM, Peterson MJ. Effects of oral temazepam on sleep spindles during nonrapid eye movement sleep: a high density EEG investigation. Eur Neuropsychopharmacol. 2015; 25(10): 1600-10. PMCID: PMC4600644
d. **Plante DT**, Goldstein MR, Cook JD, Smith R, Riedner BA, Rumble ME, Jelenchick L, Roth A, Tononi G, Benca RM, Peterson MJ. Effects of oral temazepam on slow waves during non-rapid eye movement sleep in healthy young adults: a high density EEG investigation. Int J Psychophysiol. 2016; 101:25-32. PMCID: PMC4766048

4. *Neurochemical Alterations in Sleep Disorders*

An early focus of my work was the identification of neurochemical alterations in primary insomnia (PI), a disorder characterized by chronic partial sleep loss. I was the principal investigator on a research effort sponsored by a mentored physician-scientist training award during which I examined brain γ-aminobutyric acid (GABA) via single-voxel ^{1}H-MRS at high field (4 Tesla) utilizing J-difference editing using Point Resolved Spectroscopy with MEGA suppression (MEGAPRESS). This work confirmed reductions in GABA in the occipital and anterior cingulate cortices in patients with PI, paralleling findings of other investigative groups that had applied similar methods in the study of major depressive disorder, which has been linked with insomnia in multiple epidemiological investigations. In addition, we applied novel linear mixed effects models to ^{31}P-MRS data collected in a separate group of patients with PI, demonstrating tissue type specific reductions of phosphocreatine in gray matter, suggestive of increased neuronal energy demand in the disorder. To further explore changes in brain bioenergetics associated with sleep loss, using a transdiagnostic approach, I applied similar methods of analysis in an acute sleep deprivation model, demonstrating gray matter specific increases in phosphocreatine resultant after recovery sleep. These in vivo findings illustrate specific metabolic effects in gray matter related to sleep loss in normal and pathologic processes, and will likely inform the identification of neurochemical biomarkers of acute and chronic sleep loss.

a. **Plante DT**, Jensen JE, Schoerning L, Winkelman JW. Reduced γ-aminobutyric acid in occipital and anterior cingulate cortices in primary insomnia: a link to major depressive disorder? Neuropsychopharmacology, 2012; 37(6):1548-57. PMCID: PMC3327859
b. Harper DH, **Plante DT**, Jensen JE, Ravichandran C, Buxton OM, Benson KL, O'Connor SP, Renshaw PF, Winkelman JW. Energetic and cell membrane metabolic products in patients with primary insomnia: a 31P MRS study at 4 Tesla. SLEEP, 2013; 36(4): 493-500. PMCID: PMC3612248
c. **Plante DT**, Trksak GH, Jensen JE, Penetar DM, Ravichandran C, Riedner BA, Tartarini WL, Dorsey CM, Renshaw PF, Lukas SE, Harper DG. Gray matter specific changes in brain bioenergetics after acute sleep deprivation: a 31P MRS study at 4 Tesla. SLEEP, 2014; 37(12):1919-1927. PMCID: PMC4548516

5. *Sleep Disturbance in Borderline Personality Disorder*

Sleep disturbance plays an important role in the presentation and course of a number of neuropsychiatric disorders, but scant research has examined the role of sleep in borderline personality disorder (BPD). To begin to address this shortcoming in the literature, while a resident, I developed collaborative investigations with Dr. Mary Zanarini, examining sleep-related pathology in patients with BPD who were participating in the ongoing McLean Study of Adult Development. We have demonstrated that patients with BPD use more sedative-hypnotics than comparison participants, and that maladaptive cognitions about sleep and subjective sleep disturbance are associated with absence of recovery from BPD. These studies demonstrate the importance of sleep in BPD, and the need to develop targeted sleep-related therapies in the disorder.

a. **Plante DT**, Zanarini MC, Frankenburg FR, Fitzmaurice GM. Sedative-hypnotic use in patients with borderline personality disorder and axis II comparison subjects. J Pers Disord. 2009; 23(6):563-71. PMCID: PMC3222941
b. **Plante DT**, Frankenburg FR, Fitzmaurice GM, Zanarini MC. Relationship between sleep disturbance and recovery in patients with borderline personality disorder. J Psychosom Res, 2013; 74(4):278-82. PMCID: PMC3603271
c. **Plante DT**, Frankenburg FR, Fitzmaurice GM, Zanarini MC. Relationship between maladaptive cognitions about sleep and recovery in patients with borderline personality disorder. Psychiatry Res, 2013 2010(3):975-9. PMCID PMC3840073.

Complete List of Published Work in MyBibliography:
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