BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors. Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Savage, Lisa M.

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

POSITION TITLE: Chair and Professor of Psychology and Behavioral Neuroscience

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
University of Minnesota-Duluth	B.A.	1987	Psychology/Biology
University of Minnesota-Twin Cities	Ph.D.	1993	Psychology/Neuroscience
VA Medical Center-San Diego, CA	Post-doc	1995	Neurology Research

A. Personal Statement

I have trained undergraduate students, graduate students, and postdoctoral scholars, as well as directed the graduate program in Behavioral Neuroscience at Binghamton University for 10 years. I was co-chair of the Biological Science Department (2016-2019), and was Chair of the Psychology Department (2020-2023). My record demonstrates my success as an advocate for diversifying science. Presently, I am a MPI on a R25 training grant that provides opportunities for under-represented students from community colleges to come to Binghamton University for an intensive summer research program and assists these students in transferring to 4-yr colleges. As an under-represented minority scholar, I know the importance of programs to enhance diversity on university campuses and more broadly in science. Throughout my career. I have taken an active role in improving the scientific environment for non-traditional students to succeed. I have a commitment to scientific rigor, effective training, mentoring, as well as to promoting inclusive, safe, and supportive scientific environments for all trainees and colleagues. I am a member of the Neuroscience Scholars Program, an SFNsponsored program that builds community and careers of under-represented graduate students and postdoctoral scholars. I was recently a senior panelist at the Broadening the Representation of Academic Investigators in NeuroScience (BRAINS), a national program dedicated to advancing diversity and inclusion in Neuroscience. I have also held a leadership position within the APA as the president of Division 6 (Behavioral Neuroscience), and within my University (Co-Chair of Biological Sciences; Chair of Psychology). Beyond my administration expertise, I also have the leadership skills necessary to successfully mentor a diverse set of trainees.

In addition, I am an active research scientist. For the past 5 years, I have served on the executive committee of the Developmental Exposure Alcohol Research Center (DEARC). Currently, I am the scientific director for the DEARC. Furthermore. I am also a scientific member of the Neurobiology of Adolescent Drinking in Adulthood (NADIA) consortium, which spans multiple research institutions studying the consequences of adolescent alcohol exposure. My formal background is in psychology and neuroscience, with specific training in the neurobiology of learning and memory. For the past 28 years, I have used translational models of memory-related disorders to understand how cognitive dysfunction maps onto brain pathology associated with alcohol use disorders. Specifically, I am interested in the mechanisms by which alcohol-related brain damage can be recovered. I have investigated the changes in frontocortical, basal forebrain cholinergic, hippocampal, and thalamic pathology, as a consequence of alcohol-related brain damage. I use my expertise in animal behavior, neurochemistry, and

functional neuroanatomy to investigate neural adaptions following developmental alcohol exposure. Such studies will shed light on why developmental alcohol abuse is associated with increased risks of adult alcohol use disorders, cognitive impairment, and alcohol-related brain damage.

- Fecik MJ*, **Savage, LM**. (2023). Modes of Acetylcholine Signaling in the Prefrontal Cortex: Implications for Cholinergic Dysfunction and Disorders. In (Ed T. Heinbockel) Acetylcholine: Recent Advances and New Perspectives. IntechOpen, Rijeka, Croatia
- **Savage LM**, Nunes PT*, Gursky ZH*, Milbocker KA*, Klintsova AY. (2021). Midline thalamic damage associated with alcohol-use disorders: disruption of distinct thalamocortical pathways and function. *Neuropsychology Review*, 31:447-471. Doi:10.1007/s11065-020-09450 PMID: 32789537 PMCID: PMC7878584
- Nunes Toledo P*, Kipp BT*, Reitz NL*, Savage LM. (2019). Aging with alcohol-related brain damage: Critical brain circuits associated with cognitive dysfunction. Chapter 4. In (Eds Deak, T., Savage, LM) Late aging-associated changes in alcohol sensitivity, inflammation and cognitive decline. Elsevier, Amsterdam Netherlands. PMID: PMC7372724
- **Savage LM.** (2015). Alcohol-related brain damage and neuropathology. Chapter 7, In (Eds: Svanberg, J., Withall A, Draper B, Bowden S) *Alcohol and the Adult Brain*, Taylor & Francis, Psychological Press, East Sussex, UK. 108-125.

Current projects that I would like to highlight include:

- 1. U01AA028710: Consortium on the Neurobiology of Adolescent Drinking in Adulthood (NADIA) NIH-NIAAA, Center PI: Crews FT; Component 7, PI: Savage, LM; Co-I Vetreno RP Recovery of Adolescent Alcohol Disruption of Basal Forebrain-Cortical Projection Circuits.
- 2. U01AA028710-04S1: Research Supplement to Promote Diversity in Health-Related Research
- 3. P50AA017823: Developmental Exposure Alcohol Research Center (DEARC)
 NIH-NIAAA, Center PI: Deak, T; Main 2 PI: Savage, LM
 Binge-type alcohol exposure during adolescence alters the septohippocampal circuit during advanced
- 4. R25GM056637: SUNY Upstate Bridges to the Baccalaureate Program NIH-NIGM: MPI: Savage LM, DiLorenzo, P

B. Positions, Scientific Appointments, and Honors

Positions and Employment

2019-present	Scientific Director, Developmental Exposure Alcohol Research Center
2020-2023	Chair of Psychology, Binghamton University
2016-2019	Co-Chair of Biological Sciences, Binghamton University
2016-2021	Editorial Board of Neuropsychological Review
2013-2014	President of Division 6 (Behavioral Neuroscience) for American Psychological
	Association
•	Professor, Department of Psychology, Binghamton University
2005-2016	Behavioral Neuroscience Area Head, Department of Psychology, Binghamton University
2002-2008	Associate Professor, Department of Psychology, Binghamton University
2001-2005	Director of Graduate Studies, Department of Psychology, Binghamton University
1995-2001	Assistant Professor, Department of Psychology, Binghamton University (SUNY)
1992-1994	Research Associate, Veterans Affairs Medical Center, San Diego California
	Department of Neurology Research
1989-1992	Instructor, University of Minnesota, Department of Psychology
1988-1989	Teaching Assistant, University of Minnesota, Department of Psychology
1984-1987	Research Trainee, University of Minnesota-Duluth School of Medicine

Honors and Awards

2015	SUNY Chancellor's Award for Excellence in Scholarship and Creative Activities
2014	Provost's Award for Faculty Excellence in Undergraduate Research Mentoring
2002	APA Award for Distinguished Scientific Early Career Contribution to Psychology-
	Animal Learning and Behavior, Comparative
1995	American College of Neuropsychopharmacology Minority Travel Fellowship

1992	Society for Neuroscience Minority Travel Fellowship
1991-92	NIH Psychopharmacology trainee – UMN
1988-91	US Department of Education, Indian Fellowship Program
1987	Graduate Fellowship for Minorities and Disadvantaged Students-U of Minnesota

Professional Affiliations

American Psychological Association: Fellow, Divisions 3 and 6

Society for Neuroscience: member

Research Society for Alcoholism: member

C. Contributions to Science

- 1. **Alcohol-related brain pathology.** Thiamine deficiency is a key factor in alcohol-related brain damage. My work across 20 years has shed light on the fact that damage to specific diencephalic regions is critical to alcohol-related cognitive dysfunction. Furthermore, there is cholinergic dysfunction associated with both thiamine deficiency and alcohol toxicity, and this alteration in forebrain cholinergic neurons, as well as contaminant loss of innervation to the frontal cortex and hippocampus, which modulates both dysfunction and recovery of cognitive performance.
 - Reitz NL,* Nunes PT,* Savage LM. (2021). Adolescent binge-type ethanol exposure in rats mirrors age-related cognitive decline by suppressing cholinergic tone and Hippocampal Neurogenesis.
 Frontiers in Behavioral Neuroscience, 15:772857. doi: 10.3389/fnbeh.2021.772857.

 PMCID: PMC8569390
 - Kipp BT*, Nunes PT*, Savage LM. (2021). Sex differences in cholinergic circuits and behavioral disruptions following chronic ethanol exposure with and without thiamine deficiency. Alcoholism, Clinical and Experimental Research, 45:1013-1027. doi:10.1111/acer.14594. PMCID: PMC8131257
 - Chatterton BJ*, Nunes PT*, Savage LM. (2020). The effect of chronic ethanol exposure and thiamine deficiency on myelin-related genes in the cortex and the cerebellum. Alcoholism, Clinical and Experimental Research, 44:2481-2493. doi: 10.1111/acer.14484. PMCID: PMC7725981
 - Nunes Toledo P,* Vedder LC,* Deak T, Savage, LM (2019). A pivotal role for thiamine deficiency in the expression of neuroinflammation markers in models of alcohol-related brain damage. *Alcoholism*, Clinical and Experimental Research. 43, 425-438. doi: 10.1111/acer.13946.PMCID: PMC6397077
- 2. **Cognitive thalamus.** Related to my primary line of research on alcohol-related brain pathology, we have demonstrated that the anterior and midline thalamus are critical for spatial cognition in their own right. Furthermore, several midline thalamic nuclei direct the interactions between the hippocampus and frontal cortex and this circuit is critical for restoration of behavioral function.
 - Gursky ZH*, Savage LM, Klintsova AY. (2021). Executive functioning-specific behavioral impairments in a rat model of human third trimester binge drinking implicate prefrontal-thalamo-hippocampal circuitry in Fetal Alcohol Spectrum Disorders. *Behavioral Brain Research*, 405:113208. doi: 10.1016/j.bbr.2021.113208. PMCID: PMC8005484.
 - Gursky ZH*, Savage LM, Klintsova AY. (2019). Nucleus reuniens of the midline thalamus of a rat is specifically damaged after postnatal alcohol exposure, *NeuroReport*, 30, 748-752. doi: 10.1097/WNR.00000000001270. PMCID: PMC6599629
 - Vedder LC,* Savage LM (2017). BDNF regains function in hippocampal long-term potentiation deficits caused by diencephalic damage. *Learning & Memory* 24: 81-85. doi:10.1101/lm.043927. PMCID: PMC5238722
 - Bobal M,* Savage LM (2015). The role of the ventral midline thalamus in cholinergic-based recovery in the amnestic rat. Neuroscience 285: 260-268. doi:10.1016/j.neuroscience.2014.11.015 PMCID: PMC 4282987
- 3. The role of acetylcholine in modulating cognitive function. We are one of the few labs that measure behavioral-relevant changes in acetylcholine efflux, in several brain regions, in normal rodents as well as

neuropathological models. We have demonstrated that different task demands evoke different profiles of ACh efflux as a function of brain region and neuropathological state.

- Kirshenbaum GS, Chang CY, Bompolaki M, Bradford VR, Bell J, Kosmidis S, Shansky RM, Orlandi J*, Savage LM, Harris AZ*, David Leonardo E, Dranovsky A (2023). Adult-born neurons maintain hippocampal cholinergic inputs and support working memory during aging. Mol Psychiatry. doi: 10.1038/s41380-023-02167-z.
- Kipp BT,* Nunes PT, Galaj E,* Hitchcock B, Nasra T*, Poynor KR*, Heide SK*, Reitz NL*, Savage LM. (2021). Adolescent Ethanol Exposure Alters Cholinergic Function and Apical Dendritic Branching Within the Orbital Frontal Cortex. Neuroscience. 473:52-65. PMCI: PMC9168833
- Hall J.* Gomez-Pinilla F, Savage LM (2018). Nerve growth factor is responsible for exercise-induced recovery of septohippocampal cholinergic structure and function. Frontiers in Neuroscience, doi.org/10.3389/fnins 2018.00773, PMCID: PMC6222249
- Hall J,* Savage LM (2016). Exercise leads to the re-emergence of the cholinergic/nestin neuronal phenotype within the medial septum/diagonal band and subsequent rescue of both hippocampal AcCh efflux and spatial behavior. Experimental Neurology 278: 62-75. doi:10.1016/j.expneurol.2016.01.018 PMCID: PMC4794758
- *= Trainee author
- 4. <u>Training of underrepresented scholars</u>: I have been a contributor and supporter of the training underrepresented (UR) students in STEM at all academic levels. Presently, I co-direct an undergraduate training grant (R25) that prepares UR community college students for transfer to 4-year colleges and universities to earn degrees in the sciences. I am a member of the Neuroscience Scholars Program, an SFN sponsored program that builds community and careers of underrepresented graduate students and post-doctoral scholars. I also have had two Diversity Supplements awarded to my parent grants to facilitate the career of UR postdoctoral scholars. I was a senior panelist at the Broadening the Representation of Academic Investigators in NeuroScience (BRAINS), a national program dedicated to advancing diversity and inclusion in Neuroscience. As a minority scholar, I believe that diversifying science is critical to reducing and eventually eliminating mental and other health disparities.

Complete List of Published Work in MyBibliography:

http://www.ncbi.nlm.nih.gov/sites/myncbi/lisa.savage.1/bibliography/41150060/public/?sort=date&direction=asc ending