Lisa Marie Savage, Ph.D. 2018

I. University Address

Department of Psychology
SUNY-Binghamton
Phone: 1-607-777-4383
Fax: 1-607-777-4890
E-mail: lsavage@binghamton.edu

II. EDUCATION

Undergraduate
University of Minnesota-Duluth
B.A. August 1987
Major: Psychology
Minor: Biology

Graduate
University of Minnesota, Twin Cities
Ph.D. October 1993
Major: Experimental Psychology
Minor: Neuroscience
Advisor: Dr. J. Bruce Overmier

III. PROFESSIONAL POSITIONS

1. Research Trainee, University of Minnesota-Duluth School of Medicine,
Department of Physiology 1984-1987
Duties: Tissue culture maintenance, electrophysiological recording
Supervisor: Robert Pozos, Ph.D., M.D., Head of Physiology Department

2. Teaching Assistant, University of Minnesota, Department of Psychology,
Introduction to Statistics, 1988
Analysis of Complex Behavior, 1989
Supervisor: Travis Thompson, Ph.D. Professor of Psychology

3. Instructor, University of Minnesota, Department of Psychology.
Undergraduate courses taught:
Introduction to Laboratory Psychology; Psychophysics 1989, 1990
Introduction to Laboratory Psychology; Learning & Memory 1991, 1992
Supervisor: Paul Fox, Ph.D. Professor of Psychology

4. Post Doctorial Research Associate, Veterans Affairs Medical Center, San Diego California.
Department of Neurology Research. 1992-1995
Supervisor: Philip Langlais, Ph.D. Professor of Psychology (SDSU) and Neuroscience (UCSD)

5. Assistant Professor, Department of Psychology, Behavioral Neuroscience Program
State University of New York-Binghamton. 1995-2000

6. Associate Professor: Department of Psychology, Behavioral Neuroscience Program
State University of New York-Binghamton. 2001-2008

7. Professor: Department of Psychology, Behavioral Neuroscience Program
State University of New York-Binghamton. 2009-current
Courses taught:

Undergraduate		***Graduate***	
Comparative Neuropsychology	n=25	Alcohol Induced Brain Damage	n=8
Drugs & Behavior	n=250	Conditioning and Learning	n=15
Alcohol Effects on Brain & Behavior	n=25	Physiological Psychology	n=8
Learning Laboratory	n=125	Behavioral Neuroscience	n=10

Research in Behavioral Neuroscience	n=6	Neurobiology of Learning and Memory	n=11
Behavioral and Brain plasticity	n=21	Neurobiology of Reward	n=7
		Neuroplasticity and Behavior	n=11
		Neurobiology of Disease	n=9

8. Director of Graduate Studies: Department of Psychology,
State University of New York-Binghamton. 2001-2005

9. Behavioral Neuroscience Area head
State University of New York-Binghamton 2005-2016

10. President of Division 6 (Behavioral Neuroscience) for APA 2013-2014

11. Co-Chair of Biological Sciences: 2016-2019
State University of New York-Binghamton

IV. HONORS AND AWARDS:

NIH trainee; Minority Biomedical Research Services,
University of Minnesota-Duluth School of Medicine 1984-1985

NIH fellowship; Minority Access to Research Careers,
University of Minnesota-Duluth School of Medicine 1985-1987

Graduate Fellowship for Minorities and Disadvantaged Students;
University of Minnesota 1987-1988

Fellowship, Department of Psychology; University of Minnesota 1988-1989

Fellowship; US Department of Education, Indian Fellowship Program 1988-1991

NIH Trainee, Neurobehavioral Pharmacology Trainee Grant,
University of Minnesota, 1991-1992

Society for Neuroscience Minority Travel Fellowship 1992

American College of Neuropsychopharmacology Travel Fellowship
Awards for Minorities 1995

APA Award for Distinguished Scientific Early Career Contribution to Psychology
Animal Learning and Behavior, Comparative 2002

Provost's Award for Faculty Excellence in Undergraduate Research Mentoring 2014

Chancellor's Award for Excellence in Scholarship and Creative Activities 2015

Career Champion: Fleishman Center for Career and Professional Development 2015

V. CURRENT GRANTS:

R01AA021775 NIAAA
Cortical Biobehavioral Disruption after Thiamine Deficiency and Chronic Alcohol
In this proposal, we use our recently developed translational animal model of chronic ethanol treatment (CET) combined with thiamine deficiency (TD) to determine both the independent

actions of CET and TD as well as how these factors synergistically interact to affect neurotrophin adaptation, cognitive functioning and activation of the fronto-cortico-limbic network (AIM 1). We will determine whether basal forebrain cholinergic cell loss, altered cortical cellular structure and dysfunctional acetylcholine (ACh) release are critical mediators of alcohol-related cognitive impairment. Furthermore, we will determine whether exercise can restore behavior, cholinergic innervation, and behaviorally stimulated ACh efflux across the hippocampus and frontal cortex (AIM 2). The final AIM (3) will determine whether a moderate TD episode during CET leads to greater disruption of cytogenesis (neurogenesis in the hippocampus and gliogenesis in the frontal cortex).

5R25GM056637-15 (Savage, DiLorenzo, MPIs) NIH-NIGM
SUNY Upstate Bridges to the Baccalaureate Program
This program aims to identify the appropriate URM students in their first year at the community college; support the students in their science courses at the community college and provide a hands-on research experience in the university setting for these students.

P50AA017823 (renewal): Developmental Exposure Alcohol Research Center (DEARC)
NIH-NIAAA: Head Center PI: Spear, L.P.
1. Animal/Behavioral Core (ABC);
(Savage, PI)
The DEARC represents a comprehensive approach towards understanding functional and neural effects of developmental alcohol exposure at multiple neural levels and across the protracted span of brain development – including the important developmental transitions of adolescence. The “alcoholism generator” theme of our Center emphasizes the importance of developmental programming in the emergence of alcohol problems and alcoholism. The ABC is designed to: (a) to support production of animals needed for the DEARC; (b) provide oversight, standardization, and training for alcohol administration and measurement procedures; and (c) to function as a collaborative partnership with all DEARC PIs to develop and characterize the functional phenotypes that emerge as a result of ethanol exposure at various developmental stages.

2. NeuroCore
(Savage, Co-PI)
The NeuroCore is designed to support mechanistic approaches to assess consequences of develop-mental ethanol exposure, and how this exposure changes brain function. The NeuroCore consists of three subcomponents: The molecular subcomponent will provide nucleic acid purification, quantification and quality assessment, mRNA and microRNA expression profiling by sequencing as well as microarray, DNA profiling of transcription factor binding, modified histone binding, or CpG methylation by sequencing, multiplex validation of RNA or DNA-based data and real-time quantitative RT-PCR. Techniques available in the neuroanatomy subcomponent include immunofluorescence techniques to map regional alterations in neuronal and glial phenotypes, immunohistochemistry to determine functional anatomical circuit adaptations using expression of immediate early genes, neuropeptides, or signal transduction pathways, and other metrics of neuropathological consequences of alcohol exposure. The cellular subcomponent includes the necessary equipment, space and expertise to conduct *in vitro* experiments in primary neuronal, glial, and cell line specific cultures as well as embryonic and oocyte preparations to create genetically modified rodent models.

VI. PROFESSIONAL MEMBERSHIPS AND ACTIVITIES:

American Psychological Association: Member, Fellow Division 3 & 6
President Division 6, 2013
Treasurer, Division 6, 2015-2018

Society for Neuroscience: Member

Research Society on Alcoholism: Member

VII. PUBLICATIONS:

1. **Savage, L.M**., Stanchfield, M.,° & Overmier, J.B. (1994). The Effect of Scopolamine, Diazepam and Lorazepam on Working Memory in Pigeons: An Analysis of Reinforcement Procedures and Sample Problem Type. Pharmacology, Biochemistry & Behavior, 48, 183-192.
2. **Savage, L.M.**, & Overmier, J. B. (1995). The Influence of Sequential Information in Rats: Learning, Memory and the Effects of Amnestic Drugs. Learning & Motivation, 26, 300-322.
3. Langlais, P.J., & **Savage, L.M**. (1995). Thiamine Deficiency in Rats Produces Cognitive and Memory Deficits on Spatial Tasks that Correlate with Tissue loss in Diencephalon, Cortex, and White Matter. Behavioural Brain Research, 68, 75-89.
4. **Savage, L.M**. & Langlais, P.J. (1995). Differential Outcomes Attenuates Spatial Memory Impairments Seen in Pyrithiamine-induced Thiamine Deficient Rats. Psychobiology, 23, 153-160.
5. Langlais, P.J., Zhang, S.X., & **Savage, L.M**. (1996). Neuropathology of Thiamine Deficiency: An Update on the Comparative Analysis Of Human Disorders and Experimental Models. Metabolic Brain Disease, 11 19-37.
6. **Savage, L.M**., Sweet, A.J.,° Castilo, R.,° & Langlais, P.J. (1997). The Role of Internal Medullary Lamina Nuclei and Posterior Thalamic Nuclei in Learning, Memory, and Habituation in the Rat. Behavioural Brain Research, 82, 133-147.
7. Linden, D.R., **Savage, L.M**., & Overmier, J.B. (1997). General Learned Irrelevance: A Pavlovian Analog to Learned Helplessness. Learning & Motivation, 28, 230-247.
8. **Savage, L.M**., & Parsons, J.P.* (1997). The Effects of Delay-Interval, Inter-trial-Interval, Amnestic Drugs, and Differential Outcomes on Matching-to-Position in Rats. Psychobiology, 25, 303-312.
9. **Savage, L.M**. (1998). Early Native American Uses of Animals for Medicine and Research. In: M. Bekoff. & C.A. Meaney (eds): Encyclopedia of Animal Rights and Animal Welfare. Greenwood Publishing Group. Westport, CT. pp. 252-253.
10. **Savage, L.M**., Castillo, R. °, & Langlais, P.J. (1998). Effects of thalamic intralaminar nuclei and internal meduallary lamina on spatial memory and object discrimination. Behavioral Neuroscience, 112, 1339-1352.
11. Overmier, J.B., **Savage, L.M**. & Sweeney, W.A.* (1999). Behavioral and Pharmacological Analysis of Memory Offer New Options for Remediation. pp. 231-246. In M. Haug & R. Whalen (Eds). Animal Models of Human Emotion and Cognition. APA publications: Washington, D.C.
12. **Savage, L.M**., Pitkin, S.*, & Knitowski. K* (1999). Rats exposed to pyrithiamine- induced thiamine deficiency are more sensitive to the amnestic effects of scopolamine and MK-801: Examination of working memory, response selection, and reinforcement contingencies. Behavioural Brain Research, 104, 13-26.
13. **Savage, L.M**., Pitkin, S.*, & Careri, J.° (1999). Memory enhancement in aged-rats: The differential outcomes effect. Developmental Psychobiology, 35, 318-327.

14. **Savage, L.M**., Candon, P.*, & Hohmann H.° (2000). Alcohol-induced brain damage and behavioral dysfunction: Using an animal model to examine sex differences. Alcoholism; Clinical and Experimental Research. 24, 4, 465-475.
15. Hochhalter, A.K. *, Sweeney, W.A. *, **Savage, L.M**., Bakke, B.L., & Overmier, J.B. (2001). Using animal models to address the memory deficits of Wernicke-Korsakoff syndrome. In Carroll, M.E., & Overmier, J.B, (Eds). Animal research and human health: Advancing human welfare through behavioral science. (pp. 281-292). Washington, DC, US, American Psychological Association.
16. Pitkin, S.* & **Savage, L**. (2001). Aging potentiates acute neurological symptoms and brain pathology in pyrithiamine-induced thiamine deficient rats. Behavioural Brain Research, 119, 167-177.
17. **Savage, L**. (2001). In search of the neurobiological correlates of the Differential Outcomes Effect. Integrative Physiological and Behavioral Science, 36, 182-195.
18. **Savage, L**. (2002). Biography for Distinguished Scientific Early Career Contribution to Psychology- Animal Learning, Comparative, American Psychologist, 57, 17-20.
19. Ramos, R.,* & **Savage, L.M.** (2003). The Differential Outcomes Procedure can enhance or interfere acquisition—dependent on when Pavlovian conditioning trial occur and the nature of the conditional discrimination rule. Integrative Physiological and Behavioral Science, 38, 17-35.
20. **Savage, L.,** Chang, Q., & Gold., P.E. (2003). Interactions between diencephalic and hippocampal processing. Learning and Memory, 10, 242-246.
21. Pitkin, S.R.* & **Savage, L.M**. (2004). Age-related vulnerability to diencephalic amnesia produced by thiamine deficiency: The role of time of insult. Behavioural Brain Research, 148, 93-105.
22. **Savage, L.M.** (2004). Animal models of memory disorders give insight into how psychological and neural systems interact. Psychological Science Agenda, 18, 7. Washington, DC, US, American Psychological Association. http://www.apa.org/science/psa/sb-savageprt.html
23. **Savage, L.M**., Buzzetti, R., & Ramirez, D. (2004). The effects of hippocampal lesions on learning, memory and reward expectancies. Neurobiology of Learning and Memory. 82, 109-119.
24. Ramirez, D*., Buzzetti, R*. & **Savage, L.M**. (2005). The role of the GABA-A agonist muscimol on memory performance: Reinforcement contingencies determines the nature of the deficit. Neurobiology of Learning and Memory, 84, 184-191.
25. Chang, Q, **Savage, L.M.,** & Gold, P.E. (2006). Microdialysis measures of functional increases and brain regional differences in ACh release: A requirement for acetylcholinesterase inhibitors in the perfusate. Journal of Neurochemistry. 97, 697-706.
26. Roland, J.J.* & **Savage, L.M**. (2007). Hippocampal and striatal acetylcholine efflux during learning in diencephalic-lesioned rats. Neurobiology of Learning and Memory, 87, 123-132.
27. **Savage, L.M**., Roland, J.J.*, Klintsova A. (2007). Selective septohippocampal – but not forebrain amygdalar – cholinergic dysfunction in diencephalic amnesia. Brain Research, 1139, 210-219.
28. Ramirez, D.* & **Savage, L.M**. (2007). Differential Involvement of the basolateral amygdala, orbitofrontal cortex, and nucleus accumbens core in the acquisition and use of reward expectancies. Behavioral Neuroscience, 121, 896-906.
29. **Savage, L.M**, Koch, A.* & Ramirez, D.* (2007). Amygdala inactivation by muscimol, but not ERK/MAPK inhibition, impairs the use of reward expectancies during short-term memory. European Journal of Neuroscience, 26, 3645-3651.
30. Vetreno, R. * Anzalone, S. *, & **Savage L.M**. (2008). Impaired, spared and enhanced ACh efflux across the hippocampus and striatum in diencephalic amnesia is dependent on task demands. Neurobiology of Learning and Memory, 90, 237-244.

31. Roland, J.J.*, Mark, K.,* Vetreno, R.*, & **Savage, L.M**. (2008). Increasing hippocampal acetylcholine levels enhances behavioral performance in an animal model of diencephalic amnesia. Brain Research, 1234, 116-127.
32. **Savage, L.M**. & Ramos, R. (2009- invited review). Reward expectation alters learning and memory: The impact of the amygdala on appetitive-driven behaviors. Behavioural Brain Research, 198, 1-12.
33. Roland, J.J.* & **Savage, L.M**. (2009). Blocking GABA-A receptors in the medial septum enhances hippocampal acetylcholine release and maze behavior in a rat model of diencephalic amnesia, Pharmacology, Biochemistry & Behavior, 92, 480-487.
34. Roland, J.J.* & **Savage, L.M**. (2009). The Role of Cholinergic and GABAergic Medial Septal/Diagonal Band Cell Populations in the Emergence of Diencephalic Amnesia, Neuroscience, 160, 32-41.
35. Anzalone, S. *, Roland, J.J. *, Vogt, B. & **Savage, L.M.** (2009). Acetylcholine efflux from retrosplenial areas and hippocampal sectors during maze exploration. Behavioural Brain Research, 201, 272-278.
36. Vetreno, R. * Anzalone, S. *, & **Savage L.M**. (2010). Ethology, In I. Weiner (Ed.), Corsini Encyclopedia of Psychology, New York: John Wiley & Sons. Pp 602-603.
37. Roland, J.J.* Levinson, M. * Vetreno, R. *, & **Savage, L.M**. (2010). Differential effects of systemic and intraseptal administration of the acetylcholinesterase inhibitor tacrine on the recovery of spatial behavior in an animal model of diencephalic amnesia. European Journal of Pharmacology, 629, 31-39.
38. Anzalone, S.J. *, Vetreno, R.P. *, Ramos, R.R. & **Savage, L.M.** (2010). Cortical cholingeric abnormalities during maze exploration following diencephalic damage induced by thiamine deficiency. European Journal of Neuroscience, 32, 847-858.
39. **Savage, L.M.** & Guarino, S.* (2010). Memory for reward location is enhanced even though acetylcholine efflux within the amygdala is impaired in rats with damage to the diencephalon produced by thiamine deficiency. Neurobiology of Learning and Memory, 94, 554-560.
40. Vetreno, R.P. *, Hall, J. * & **Savage, L.M.** (2011). Alcohol-induced amnesia and dementia: What animal models have told us about the relationships between etiological factors, neuropathology and cognitive impairments. Neurobiology of Learning and Memory. 96, 596-608.
41. Dupre, K.B.*, Ostock, C.Y.*, Eskow-Jaunarajs, K.L.*, Button, T., Wolf, W., **Savage, L.M**., & Bishop, C. (2011). Local modulation of striatal glutamate efflux by serotonin 1A receptor stimulation in dyskinetic, hemiparkinsonian rats. Experimental Neurology, 229, 288-299.
42. Vetreno, R.P.*, Klintsova, A. & **Savage, L.M.** (2011). Stage dependent alterations in progenitor cell proliferation and neurogenesis in an animal model of Wernicke-Korsakoff syndrome. Brain Research, 1391, 132-146.
43. **Savage, L.M.** Hall, J.* & Vetreno, R.P.* (2011). Anterior thalamic lesion alter hippocampal dependent behavior and hippocampal acetylcholine release. Learning & Memory, 18, 751-758.
44. Vetreno, R.P.*, Ramos, R. Anzalone, S J.*, & .**Savage, L.M.** (2012). Brain and behavioral pathologies in an animal model of Wernicke's encephalopathy and Wernicke-Korsakoff syndrome. Brain Research, 1436, 178-192.
45. **Savage, L.M.** (2012). Sustaining high acetylcholine levels acetylcholine levels in the medial frontal cortex, but not retrosplenial cortex, recovers spatial alternation in the rodent PTD-model of amnesia. Behavioral Neuroscience, 126, 226-236.
46. Resende, L.S*., Ribeiro, A.M., Werner. D, Hall, J.* **Savage, LM**. (2012). Thiamine deficiency degrades the links between spatial behavior and hippocampal synapsin I and phosphorylated synapsin I protein levels. Behavioural Brain Research, 232, 421-425

47. **Savage, L.M**., Hall, J. * & Resende, L.S.* (2012, invited review). Translational rodent models of Korsakoff syndrome reveal the critical neuroanatomical substrates of memory dysfunction and recovery. Neuropsychology Review, 22,195-209.
48. Roland, J.J., Stewart, A.L., Janke, K.L., Gielow, M.R., Kostek, J.A., **Savage, L.M.**, Servatius, R.J. & Pang, K.C. (2014). Medial Septum-Diagonal Band of Broca (MSDB) GABAergic Regulation of Hippocampal Acetylcholine Efflux Is Dependent on Cognitive Demands. Journal of Neuroscience, 34, 506-514.
49. Hall, J.M.*, Vetreno, R.P.* & **Savage LM**. (2014). Differential cortical neurotrophin and cytogenetic adaptation after voluntary exercise in normal and amnestic rats. Neuroscience, 258, 131-146.
50. Bobal, M.* & **Savage, L.M.** (2015). The role of the ventral midline thalamus in cholinergic-based recovery in the amnestic rat. Neuroscience, 285, 260-268
51. Savage, L.M. (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.
52. Vedder, L.+, Hall, J.*, Jabrouin, K.R.* & **Savage, L.M.** (2015). Interactions between chronic ethanol consumption and thiamine deficiency on neural plasticity, spatial memory and cognitive flexibility. Alcoholism; Clinical and Experimental Research. 39:2143-2153.
53. Hall, J.* & **Savage, L.M**. (2016). The re-emergence of the Nestin-cholinergic phenotype within the medial septum and diagonal band after exercise that rescues hippocampal ACh efflux and spatial behavior in the amnestic rat. Experimental Neurology, 278, 62-75.
54. Fernandez, G.+, Stewart, W.* & **Savage, L.M**. (2016) Chronic drinking during adolescence predisposes the adult rat for continued heavy drinking: Neurotrophin and behavioral adaptation after long-term, continuous ethanol exposure. Plos 1: 1;11(3):e0149987
55. Vedder, L.C. + & Savage, L.M. (2017). BDNF regains function in hippocampal long-term potentiation deficits caused by diencephalic damage. Learning & Memory, 24, 81-85.
56. Fernandez, G.M.,+ Lew, B.,* Vedder, L.C.+ & **Savage, L.M.** (2017). Chronic intermittent ethanol exposure leads to alterations in brain-derived neurotrophic factor within the frontal cortex and impaired behavioral flexibility in both adolescent and adult rats. Neuroscience, 348, 324-334.
57. Fernandez, G.M.+ & **Savage, L.M.** (2017). Adolescent Binge Ethanol Exposure Alters Specific Forebrain Cholinergic Cell Populations and Leads to Selective Functional Deficits in the Prefrontal Cortex. Neuroscience, 361, 129-143.
58. Hall, J.* Gomez-Pinilla, F. & Savage, L.M. (submitted). Nerve growth factor is responsible for exercise-induced recovery of septohippocampal cholinergic structure and function.
59. Gursky, Z.,Savage, L.M. & Klintsova, A. (submitted) Selective damage of thalamic nucleus reuniens in a rat model of fetal alcohol spectrum disorders: alterations to hippocampus- and prefrontal cortex-dependent behaviors.
60. Kirshenbaum, G.S.+, Robson, V.*, Shansky, R., **Savage, L.M**., Leonardo., D.E. & Dranovsky, A. (submitted). Adult-born neurons maintain cholinergic tone in the hippocampus and support working memory during aging.

•= *undergraduate student co-author*
*= *graduate student co-author*
+= *post-doctoral scientist co-author*