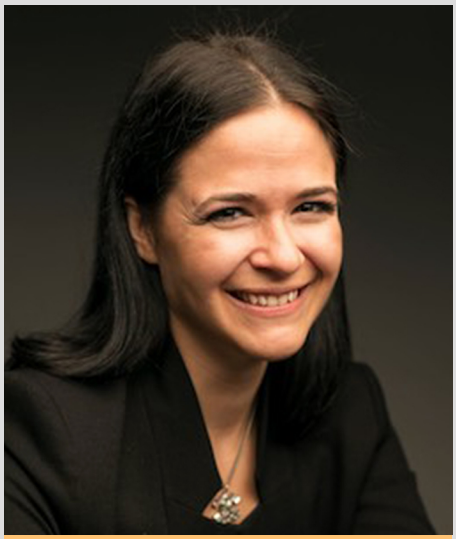

VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

Erin Calipari, PhD

Assistant Professor of Pharmacology
Assistant Professor of Psychiatry and Behavioral Sciences
Assistant Professor of Molecular Physiology & Biophysics

erin.calipari@vanderbilt.edu
615-343-5792
http://caliparilab.com/erin-s-calipari

Biosketch

Dr. Calipari received her PhD in Neuroscience in from Wake Forest University School of Medicine where she studied how self-administered drugs altered dopaminergic function to drive addictive behaviors. She then completed her postdoctoral training at Icahn School of Medicine at Mount Sinai, where she used circuit probing techniques to understand the temporally specific neural signals that underlie motivation and reward learning in behaving animals. Work in her lab integrates microscopy with molecular tools to record neural activity in the brain during behavior and determine the molecular targets that underlie changes in neural activity in response to environmental stimuli.

Key Publications

"Granulocyte colony stimulating factor enhances reward learning through potentiation of mesolimbic dopamine system function," *J. Neuroscience*, In Press, 2018

"Dopaminergic dynamics underlying sex-specific cocaine reward," *Nature Communications*, 8:13877, 2017

"In vivo imaging identifies temporal signature of D1 and D2 medium spiny neurons in cocaine reward," *Proceedings of the National Academy of Sciences U.S.A.*,113(10):2726-31, 2016

How does the brain encode information on a cellular level, and how does dysregulation in this process underlie psychiatric disease?

Our research is guided by two overarching questions:

1. How do **neural circuits integrate experiences** with positive and negative stimuli to guide behavior?

2. How does **dysfunction** in this process **precipitate psychiatric disease**?

One of the most fundamental forms of learning is the ability to associate **positive and negative stimuli** with cues that predict their occurrence. The ability to seek out rewarding, and avoid negative, stimuli is critical to survival and is evolutionarily conserved across species. However, **dysregulation** of these processes can precipitate a number of psychiatric disease states.

Addiction, depression, and anxiety are all examples of syndromes characterized in part by dysregulation of associative learning. These are among the most prevalent neuropsychiatric disorders and are highly comorbid. Therefore, **understanding the neural mechanisms governing associative learning** has widespread implications for developing treatment interventions for psychiatric disease.

Our work aims to combine cutting-edge technology with comprehensive models of psychiatric disease to **understand the circuit and molecular dysregulation** that underlies these disorders.

Together, our work uses and develops cutting-edge techniques to **outline the precise cells in the brain that encode information** and push the boundaries of how we understand learning, memory, and disease.