

Genetic Control of Neural Circuit Formation in the Basal Ganglia: Implications for Childhood Neurological Disorders

April 2

Tuesday, 12:30 pm

Weekly Colloquium

Billings Building
Rosedale Conference Room



Speaker: Kenneth Campbell, Ph.D.

Regional Professor, UC Department of Pediatrics
Divisions of Developmental Biology and Neurosurgery
Cincinnati Children's Hospital Medical Center
Cincinnati, OH

Host: Yutaka Yoshida, Ph.D.

For more information, please contact

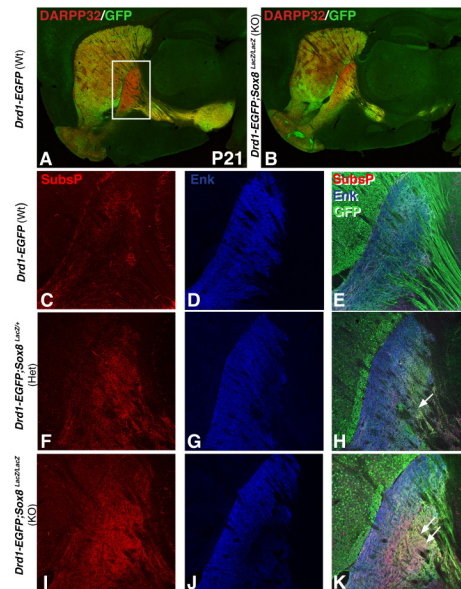
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Abstract

Our lab studies the molecular genetic mechanisms that control region specific neuronal differentiation in the mammalian ventral telencephalon and the subsequent assembly into circuitry within the basal ganglia and related brain structures. I will present work examining the molecular mechanisms that control the balance between neural progenitor maintenance and neuronal differentiation in the lateral ganglionic eminence (LGE) which gives rise to neurons of the basal ganglia. In the second half of the talk, I will discuss our work on the mechanisms controlling subtype-specific generation of striatal projection neurons. Our research has implications for understanding potential circuit abnormalities in childhood neurological disorders such as ADHD.



Ehrman L.A., Mu X., Waclaw R.R., Yoshida Y., Vorhees C., Klein W. and Campbell K. (2013) The LIM homeodomain protein *Islet1* is required for the correct development of the striatonigral pathway in the mouse, *Proceedings of the National Academy of Science USA*, 110: E4026-E4035.

Waclaw R.R., Ehrman L.A., Merchan-Sala P., Kohli V., Nardini D. and Campbell K. (2017) *Foxo1* is a downstream effector of *Isl1* in direct pathway striatal projection neuron development in the embryonic mouse telencephalon, *Molecular and Cellular Neuroscience*, 80: 44-51.

Kuerbitz J., Arnett M., Ehrman S., Fischer S.E., Garratt A.N., Williams M.T., Vorhees C.V., Muglia L., Waclaw R.R. and Campbell K. (2018) Loss of intercalated cells (ITCs) in the mouse amygdala of *Tshz1* mutants correlates with fear, depression and social interaction phenotypes, *Journal of Neuroscience*, 38: 1160-1177.



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