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Genetic and neurophysiological approaches to tackle neurodevelopmental disorders

Rett syndrome is a postnatal childhood disorder that causes a broad range of severe neuropsychiatric disabilities. The Zoghbi lab discovered that mutations in *MECP2* cause Rett syndrome and before long it became clear that mutations in *MECP2* can also cause autism and other neuropsychiatric phenotypes. Using genetically-engineered mice, the Zoghbi lab learned that the brain is acutely sensitive to MeCP2 levels and that both decreases and increases in MeCP2 levels can lead to neurological problems that are also observed in humans. They showed that normalizing MeCP2 levels can reverse disease-like features in a mouse model of the human *MECP2* duplication syndrome. Insight gained from studies of *MECP2* disorders, inspired the Zoghbi lab to extend their studies to other dosage sensitive proteins that cause a broad spectrum of neuropsychiatric disorders. Zoghbi and collaborators have been gradually pinpointing the neurons and circuit abnormalities that mediate various symptoms. Building on this understanding of neural substrate-phenotype relationships, they showed that deep brain stimulation of a specific neural network improved learning and memory in a Rett syndrome mouse model.



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Keck Seminar
Friday, Oct 12, 4pm

BioScience Research Collaborative

BRC Auditorium