

Special Lectures

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Theme A: Development

Probing Neural Circuits with Genetic Mosaics in Flies and Mice **CME**

Speaker: Liqun Luo, PhD
Stanford University
Saturday, Oct. 17, 2–3:10 p.m.
McCormick Place: Hall B1



A century ago, Ramón y Cajal used Golgi staining to systematically label single neurons and describe their morphology and connection patterns.

This lecture summarizes recent genetic mosaic methods not only to label but also to genetically manipulate singly labeled neurons. The discussion will include findings on fly Mosaic Analysis with a Repressible Cell Marker (MARCM) analysis to explore the organization and development of wiring specificity in the olfactory circuit, and mouse Mosaic Analysis with Double Markers (MADM) to study the role of neuronal activity in wiring the mammalian brain.

How Do Astrocytes Promote CNS Synaptogenesis? **CME**

Speaker: Ben A. Barres, MD, PhD
Stanford University School of Medicine
Tuesday, Oct. 20, 10–11:10 a.m.
McCormick Place: Hall B1



Astrocytes are a major cell type in the brain. Until recently, they were believed to have only passive, supporting roles. This lecture summarizes recent studies demonstrating that astro-

cytes powerfully stimulate the formation and function of synapses. Using findings from recent studies, this lecture describes efforts to identify the glial-secreted proteins that induce excitatory synapse formation and postsynaptic glutamate receptivity, as well as the neuronal receptors through which these proteins act.

Theme B: Neural Excitability, Synapses, and Glia: Cellular Mechanisms

Receptors, Synapses, and Memories **CME**

Speaker: Richard L. Huganir, PhD
Johns Hopkins University School of Medicine, Howard Hughes Medical Institute
Sunday, Oct. 18, 8:30–9:40 a.m.
McCormick Place: Hall B1



Neurotransmitter receptors mediate signal transduction at synaptic connections between neurons in the nervous system. Ongoing investigations are studying the

molecular mechanisms in the regulation of neurotransmitter receptor function and synaptic transmission, specifically through the modulation of receptor function by protein phosphorylation and the regulation of synaptic targeting of receptors. These studies have demonstrated that the modulation of receptors is a major mechanism for the regulation of synaptic transmission and plasticity in the brain.

From Synapses to Autism — Neurexins, Neuroligins, and More **CME**

Speaker: Thomas C. Südhof, MD
Stanford University
Sunday, Oct. 18, 1–2:10 p.m.
McCormick Place: Hall B1



Neural circuits consist of networks of synapses that mediate human perception, thought, and behavior. These are impaired in cognitive diseases such as autism. The formation and

plasticity of synaptic networks depend on synaptic cell-adhesion molecules, such as neurexins and neuroligins, which interact with each other at synapses. This lecture discusses neurexins and neuroligins as central determinants of the properties and plasticity of neural circuits and the dysfunction of neurexins and neuroligins in autism and other neural circuit disorders.

Neuronal Activity and Circuit Assembly **CME**

Speaker: Rachel O.L. Wong, PhD
University of Washington
Tuesday, Oct. 20, 11:30 a.m.–12:40 p.m.
McCormick Place: Hall B1



The diversity and complexity of neural circuits have made it challenging to fully understand how synaptic connectivity patterns are organized during develop-

ment. Studies across many model systems have implicated neurotransmission in patterning neuronal processes and their connections. However, imaging approaches have enabled researchers to uncover unexpected effects of early neurotransmission on circuit assembly in the vertebrate retina. Together with past findings, recent observations raise further debate on how activity might act to locally and globally shape neuronal networks *in vivo*.

Theme C: Disorders of the Nervous System

Prophylaxis of Posttraumatic Epilepsy: Waiting for Translation **CME**

Speaker: David A. Prince, MD
Stanford University Medical Center
Monday, Oct. 19, 8:30–9:40 a.m.
McCormick Place: Hall B1



Traumatic brain injury can initiate both adaptive and maladaptive processes leading to recovery and/or epileptogenesis. The latent period between trauma and seizures provides an oppor-

tunity for prophylactic intervention, once the critical underlying pathophysiological processes are identified. This lecture reviews anatomical and electrophysiological data focused on two key epileptogenic mechanisms following cortical injury. Promising experimental results will be described suggesting these aberrant processes may be modified to prevent posttraumatic epilepsy.

Understanding Neurogenetic Mechanisms in Neuropsychiatric Conditions: Lessons from Williams Syndrome CME

Speaker: Karen F. Berman, MD
National Institute of Mental Health,
National Institutes of Health
Tuesday, Oct. 20, 1–2:10 p.m.
McCormick Place: Hall B1



Williams Syndrome (WS), caused by hemizygous microdeletion of some 1.6 megabases on chromosome 7q11.23, has a unique profile of striking behavioral features: hypersociability combined with differential impact on cognitive functions — some mildly affected while others, particularly visuospatial construction, are severely impaired. Because the genes involved are known, WS affords a privileged setting for investigating how genes are translated in the brain to produce cognitive and behavioral features.

Transcriptional and Epigenetic Mechanisms of Drug Addiction CME

Speaker: Eric J. Nestler, MD, PhD
Mount Sinai School of Medicine
Wednesday, Oct. 21, 1–2:10 p.m.
McCormick Place: Hall B1



This lecture focuses on the role played by changes in gene expression, and related changes in chromatin remodeling in the brain's reward circuits in mediating the long-lasting alterations induced by chronic exposure to drugs of abuse that underlie aspects of drug addiction. Particular attention will be given to CREB and DeltaFosB, two transcription factors of interest, their numerous target genes, and downstream functional consequences as important mediators of drug action.

Theme D: Sensory and Motor Systems

Finding Your Way: The Brain's Mechanisms for Mapping External Space CME

Speaker: May-Britt Moser, PhD
Kavli Institute for Systems Neuroscience and
Centre for the Biology of Memory, Norwegian
University of Science and Technology
Monday, Oct. 19, 11:30 a.m.–12:40 p.m.
McCormick Place: Hall B1



A major goal of neuroscience is to understand neural circuit operations that guide complex behavior. Decades of work have shown how sensory systems detect and record features from the external world, but how the brain generates its own codes in the

nonsensory parts of the cortex remains a mystery. This lecture shows how a representation of an animal's current and past location is constructed in the entorhinal cortex and the hippocampus — many synapses away from the sensory cortices.

Theme E: Homeostatic and Neuroendocrine Systems

Biological Origins of Sex Differences in Brain Function and Disease CME

Speaker: Arthur P. Arnold, PhD
University of California-Los Angeles
Sunday, Oct. 18, 11:30 a.m.–12:40 p.m.
McCormick Place: Hall B1



Each of us has a sex, which profoundly influences our behavior, concept of self, and susceptibility to disease. In mammals, all sex differences stem originally from the genetic inequality of the sex chromosomes, which cause sex differences in gonadal secretions that in turn induce most sex differences in the brain. X and Y genes also have cell-autonomous sex-specific effects in nongonadal tissues, such as the brain, which lead to sex differences in function and disease.

Genetic, Molecular, and Physiological Mechanisms Involved in Human Obesity CME

Speaker: Sadaf Farooqi, PhD
University of Cambridge, United Kingdom
Wednesday, Oct. 21, 8:30 a.m.–9:40 a.m.
McCormick Place: Hall B1



Leptin acts by regulating a complex network of brain responses that can be studied using functional imaging to coordinate changes in nutritional state with changes in food intake and the “liking” of food. A downstream target of leptin action, the melanocortin 4 receptor, plays a key role in modulating sympathetic nervous system mediated changes in blood pressure. Genetic disruption of brain-derived neurotrophic factor and its receptor cause a complex neurobehavioral phenotype including hyperactivity and memory loss, as well as severe obesity.

Theme F: Cognition and Behavior

Complex Brain Networks: From Structural Connectivity to Functional Dynamics CME

Speaker: Olaf Sporns, PhD
Indiana University



Wednesday, Oct. 21,
11:30 a.m.–12:40 p.m.
McCormick Place: Hall B1
Nervous systems are complex networks of interconnected neural elements that engage in

spontaneous or evoked dynamics. Recent advances in the quantitative analysis and modeling of complex networks have provided new insights into the architecture of brain, structural, and functional connectivity — its small-world topology, efficient information flow, low wiring cost, modularity, and hubs. This talk provides an overview of this emerging field, with an emphasis on how complex network approaches can reveal structure/function relationships in the human brain.

Changing Fear CME

Speaker: Elizabeth A. Phelps, PhD
New York University
Wednesday, Oct. 21, 10–11:10 a.m.
McCormick Place: Hall B1



This lecture explores how animal models of fear learning extend to humans in a social context. Specifically, the lecture addresses how the neural circuitry of fear conditioning forms the basis to fears learned through social communication and how changing fears in humans through social and nonsocial means relies on overlapping neural mechanisms. Social means of fear learning and their alteration in humans take advantage of phylogenetically shared systems of simple fear conditioning. This flexibility of fear learning in humans may present unique challenges when trying to eliminate acquired fears.

Theme G: Novel Methods and Technology Development

Optogenetics: Development and Application CME

Speaker: Karl Deisseroth, MD, PhD
Stanford University
Tuesday, Oct. 20, 8:30–9:40 a.m.
McCormick Place: Hall B1



Optogenetics implements temporally precise control of defined electrical and biochemical events within specific cell types in living circuits, including those within freely behaving mammals. This lecture describes the development of optogenetic tools, along with applications to cells and circuits involved in synchrony, locomotion, awakening, and reward. Applications to disease questions also will be covered, where optogenetics has allowed establishment of causal relationships between precise activity patterns in defined cells and behavioral pathophysiology.