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Special Lecture

008. Making, Breaking, and Linking Engrams

Location: Hall B1

Time: Sat, Oct. 17, 2015, 2:00 PM - 3:10 PM

Support: CIHR grant MOP-74650

NSERC grant

NARSAD grant

BRAIN Canada

Speakers: *S. A. JOSSELYN;
Neurosci. & Mental Hlth., Hosp. for Sick Children, Toronto, ON, Canada

Abstract: A fundamental goal of neuroscience is to understand how information is encoded, stored, linked and later used in the brain. The physical or functional representation of a memory (the memory trace or "engram") is thought to be sparsely encoded over a distributed memory network. However, identifying the precise neurons which make up an engram has challenged for scientists since Karl Lashley's conceded defeat in his "search for the engram" in 1950. This lecture will discuss new insights into how engrams are formed, linked and used.

Disclosures: S. A. Josselyn: None.

Presidential Special Lecture

009. Themes and Variations in Circuits and Behavior

Location: Hall B1

Time: Sat, Oct. 17, 2015, 5:15 PM - 6:25 PM

Speakers: *C. BARGMANN;
Dept Anat., Howard Hughes Med. Institute, The Rockefeller Univ., New York, NY

Abstract: Behavior is variable, both within and between individuals. The nematode worm *C. elegans* allows scientists to explore how genes, neurons, circuits, and the environment interact to give rise to flexible behaviors. Studies of *C. elegans* foraging behaviors have provided insights into three levels of behavioral variability: the gating of information flow by circuit state over

seconds, the extrasynaptic regulation of circuits by neuropeptides and neuromodulators over minutes, and natural genetic variation.

Disclosures: C. Bargmann: None.

Special Lecture

096. Genetic Dissection of Neocortical Circuits

Location: Hall B1

Time: Sun, Oct. 18, 2015, 8:30 AM - 9:40 AM

Speakers: *Z. J. HUANG;
Dept Neurosci, Cold Spring Harbor Lab., Cold Spring Harbor, NY

Abstract: The computational power of the neocortex emerges from a basic neural architectural plan rooted in the genome and conserved across species. Whereas a set of glutamatergic projection neurons constitute inter-areal processing streams and cortical output channels, diverse GABAergic interneuorns regulate the spatiotemporal configuration of neural ensembles. Systematic cell targeting and cell fate mapping provide entry points for integrating multiple approaches toward understanding the assembly and organization of cortical circuits. This lecture will discuss the progress and prospect on genetic targeting of glutamatergic and GABAergic neurons in the mouse, focusing on the construction and function of a chandelier cell-pyramidal cell module.

Disclosures: Z. J. Huang: None.

Special Lecture

102. From Spontaneous Neurotransmitter Release to Rapid Antidepressant Action

Location: Hall B1

Time: Sun, Oct. 18, 2015, 10:00 AM - 11:10 AM

Support: NIH Grant MH66198

Speakers: E. T. KAVALALI;
Ctr. Basic Neurosci, Univ. of Texas Southwestern Med. Ctr., Dallas, TX

Abstract: Fast synaptic communication in the brain requires synchronous vesicle fusion evoked by action potential-induced calcium influx. However, synaptic terminals also release

neurotransmitter via spontaneous vesicle fusion independent of presynaptic action potentials. Recent studies have reported a functional role for spontaneous neurotransmitter release events in the regulation of synaptic plasticity and homeostasis as well as certain behavioral outcomes such as rapid antidepressant responses. In parallel, there is evidence that the presynaptic mechanisms underlying spontaneous release of neurotransmitters and their postsynaptic targets are segregated from those of evoked neurotransmission. These findings indicate that spontaneous neurotransmission has an autonomous role in interneuronal communication that is distinct from that of evoked neurotransmitter release. This presentation will cover these recent advances in our understanding of spontaneous neurotransmitter release mediated neuronal signaling.

Disclosures: E. T. Kavalali: None.

Special Lecture

103. SfN Clinical Neuroscience Lecture: Neurotrophin Signaling and Epileptogenesis: Mechanistic and Therapeutic Insights

Location: Hall B1

Time: Sun, Oct. 18, 2015, 11:30 AM - 12:40 PM

Speakers: *J. O. MCNAMARA;
Dept Neurobio, Carl Deane Prof & Chair, Duke Univ. Med. Ctr., Durham, NC

Abstract: The lack of preventive or disease modifying treatments for common disorders of the nervous system is a glaring unmet medical need. The epilepsies are one of the most common serious neurological disorders. Temporal lobe epilepsy is a common form of human epilepsy that is often devastating, in part because many afflicted individuals experience recurrent seizures despite optimal use of anticonvulsant drugs. In addition to seizures, temporal lobe epilepsy is often accompanied by serious cognitive and emotional comorbidities including memory deficits, anxiety and depression. Among its diverse causes, an episode of prolonged seizures (status epilepticus) occurring in an otherwise healthy individual is thought to result in development of a severe form of temporal lobe epilepsy emerging many months or years later. Notably, animal models are available in which experimentally induced status epilepticus is followed by recovery, a seizure free latent period, and the subsequent development of temporal lobe epilepsy and comorbid cognitive and behavioral abnormalities. Such animal models provide the opportunity to define the molecular and cellular mechanisms by which a brief episode of status epilepticus transforms a normal brain into an epileptic brain, insights essential for identifying novel molecular targets for preventive and/or disease modifying therapies. Recent discoveries have identified targets and therapies to prevent development of temporal lobe epilepsy and associated comorbidities in experimental animals. This presentation will review these recent discoveries and

focus in particular on the causal role of excessive neurotrophin signaling in development of temporal lobe epilepsy. A brief episode of status epilepticus induces striking and prolonged increases in expression of the neurotrophin, brain derived neurotrophic factor (BDNF). This is paralleled by striking increases in the activation of the BDNF receptor receptor tyrosine kinase, TrkB. Transient inhibition of TrkB signaling in adult mice, initiated following the completion of status epilepticus and continued for just two weeks, can prevent both epilepsy and comorbid anxiety-like disorders examined many weeks after cessation of TrkB inhibition. Insights into the cellular consequences of excessive activation of TrkB that contribute to development of temporal lobe epilepsy are beginning to emerge. Pharmacological approaches to TrkB signaling aimed at prevention of temporal lobe epilepsy will be discussed.

Disclosures: J.O. McNamara: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Cofounder and member of Board of Directors of NeurOp Inc.

Special Lecture

184. Nanoscopy with Focused Light: Principles and Applications

Location: Hall B1

Time: Sun, Oct. 18, 2015, 1:00 PM - 2:10 PM

Speakers: *S. W. HELL;
Max Planck Inst. for Biophysical Chem., Göttingen, Germany

Abstract: For many centuries the perfection of lenses was sufficient to steadily improve the resolution of light microscopes. This strategy ground to a halt once the diffraction barrier was discovered in 1873. From then onwards, it was accepted that the resolution of any light-focusing microscope is limited to about half the wavelength of light in use, meaning > 200 nanometers. This barrier was ill news for the life sciences, as light microscopy is the only viable method by which we can unobtrusively image the interior of living cells and tissues. The development of stimulated emission depletion (STED) microscopy at the turn of this century showed, however, that the diffraction barrier could be radically overcome and that it was possible to design lens-based “super-resolution” fluorescence microscopes, which could resolve features down to the nanometer scale. At its most fundamental level, this breakthrough is based on the fact that in modern super-resolution microscopes, tiny features in the sample are no longer discerned by the phenomenon of focusing alone. Moreover, the molecules in question — or specific molecular tags — can be distinguished by selectively transferring them between two different states (e.g., on/off) using a diffracted pattern of light. If two neighboring molecules are in different states at the same time, they can be easily separated. Therefore, in super-resolution microscopy,

fluorescent molecular tags fulfill a double role: Not only do they highlight the features of interest, but they also provide the (pair of) states required for separation. It is the symbiosis of tags and optical design that has made the microscopes of today "super". The advent of super-resolution fluorescence microscopy is highly relevant to the neurosciences. Many features and organelles in neurons are just below the diffraction limit in size, so are only now becoming visible in their functional context, typically the brain. This is true for the so-called dendritic spines. Being the neurons' receiving ends they play a fundamental role in neuronal communication, yet many details of their precise function and purpose is hitherto unknown. Recently, STED microscopy has been used to reveal the morphology and the tiny motion of these basic organelles in a living mouse brain. These experiments not only provided important insights about dendritic spines, but they also indicated that at some point we will be able to directly visualize how synapses work on the molecular level. Understanding the basic molecular function of these neuronal junctions is one of the keys to unlocking the mysteries of information storage and learning, and will eventually give us clues on neuronal diseases.

Disclosures: S.W. Hell: A. Employment/Salary (full or part-time):; Max Planck Institute. B. Contracted Research/Research Grant (principal investigator for a drug study, collaborator or consultant and pending and current grants). If you are a PI for a drug study, report that research relationship even if those funds come to an institution.; -. C. Other Research Support (receipt of drugs, supplies, equipment or other in-kind support); -. D. Fees for Non-CME Services Received Directly from Commercial Interest or their Agents (e.g., speakers' bureaus); -. E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); shares Abberior (Instruments) GmbH. F. Consulting Fees (e.g., advisory boards); Consultant to Abberior Instruments and Leica Microsystems.

Peter and Patricia Gruber Lecture

191. Nature and Nurture in Synapse Development, Maturation, and Disease

Location: Hall B1

Time: Sun, Oct. 18, 2015, 2:30 PM - 3:40 PM

Speakers: C. J. SHATZ;
Stanford Univ., Stanford, CA

Abstract: Neural activity is needed to fine tune brain circuits. MHC Class I molecules and the PirB receptor, thought to function only in immunity, act at neuronal synapses to regulate synapse pruning and plasticity. Changes in expression could contribute to autism and schizophrenia, and possibly to synapse loss in Alzheimer's disease

Disclosures: **C. J. Shatz: None.**

Peter and Patricia Gruber Lecture

191. Nature and Nurture in Synapse Development, Maturation, and Disease

Location: Hall B1

Time: Sun, Oct. 18, 2015, 2:30 PM - 3:40 PM

Speakers: M. E. GREENBERG:
Dept. of Neurobio., Harvard Med. Sch., Boston, MA

Abstract: This lecture will discuss how sensory experience controls gene expression to regulate critical steps in synapse and neural circuit development. It will also describe how mutations in components of the signaling networks that mediate sensory experience-dependent gene transcription can lead to neurological disorders such as Rett syndrome.

Disclosures: M.E. Greenberg: None.

Presidential Special Lecture

192. The Molecular Logic of Neural Circuits: Implications for Autism and Schizophrenia

Location: Hall B1

Time: Sun, Oct. 18, 2015, 5:15 PM - 6:25 PM

Support: HHMI

MH052804

MH092931

NS077906

MH086403

MH104172

SFARI 307762

Speakers: T. C. SÜDHOF;
Howard Hughes Med. Institute, Stanford Univ. Sch. of Med., Stanford, CA

Abstract: Neural circuits process information by transmitting and computing signals at synapses. Neural circuits thus critically depend on the number and location of synapses between the neurons of that circuit and on the properties of their synapses. We hypothesize that these

parameters of synapses are determined by interactions between pre- and postsynaptic cell-surface recognition molecules and/or signaling molecules, and we refer to the rules that these molecules impose on circuits as the molecular logic of neural circuits. Several cell-surface and signaling molecules that contribute to the molecular logic of neural circuits have been characterized, most prominently presynaptic neurexin cell-adhesion molecules and their various postsynaptic ligands. Although neuropsychiatric disorders such as autism and schizophrenia are poorly understood, recent progress in human genetics, revolutionized by advances in sequencing technologies, have identified mutations in a large number of genes that predispose to autism and schizophrenia. No common theme unites the affected genes, but several of these genes encode proteins that function at the synapse, including notably the neurexins. We thus further hypothesize that at least a subset of autism and schizophrenia syndromes are produced by specific impairments in the molecular logic of neural circuits, such that the input/output relationship in particular circuits is shifted, resulting in a skewed information processing capacity of the brain for a selected set of tasks. In support of this latter hypothesis, we observed that specific autism- and schizophrenia-associated gene mutations in neurexins and their ligands cause selective alterations in a subset of synapses and circuits, and that these alterations induce discrete, specific behavioral abnormalities. Although the analysis of the molecular logic of neural circuits and of the impaired circuit logic in neuropsychiatric disorders is only at the beginning, the conceptual framework that we sketch out in this project might allow a better understanding of how brain circuits process information and of how such information processing becomes altered in autism and schizophrenia.

Disclosures: **T.C. Südhof:** B. Contracted Research/Research Grant (principal investigator for a drug study, collaborator or consultant and pending and current grants). If you are a PI for a drug study, report that research relationship even if those funds come to an institution.; Roche. D. Fees for Non-CME Services Received Directly from Commercial Interest or their Agents (e.g., speakers' bureaus); Limes Schlosskliniken. E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); REATA Pharmaceuticals, Circuit Therapeutics, Elysium LLP, Buenobel. F. Consulting Fees (e.g., advisory boards); REATA Pharmaceuticals, Genentech, Elysium, Buenobel.

Special Lecture

269. Global Positioning System Mechanisms of Migrating Monarch Butterflies

Location: Hall B1

Time: Mon, Oct. 19, 2015, 8:30 AM - 9:40 AM

Support: NIH grant GM086794

AFOSR grant FA9550-10-1-0480

Speakers: *S. M. REPPERT;
Univ. of Massachusetts Med. Sch., Worcester, MA

Abstract: Studies of the eye-popping migration of the eastern North American monarch butterfly have revealed mechanisms behind its navigation using a time-compensated sun compass. Daylight cues, such as the sun itself and polarized light, are processed through both eyes and integrated through complex circuitry in the brain's central complex, the presumed site of the sun compass. Monarch circadian clocks have a distinct molecular mechanism, and those that reside in the antennae provide time compensation. Recent evidence shows that migrating monarchs can also use a light-dependent inclination magnetic compass for orientation in the absence of directional daylight cues. The orientation mechanisms at the disposal of migrant monarchs, along with the availability of powerful nuclease-mediated genetic approaches for studying and manipulating these mechanisms, make migratory monarchs an exemplary model system. Taken together, the migratory monarch rivals even the most complex of vertebrate systems, for studying orientation, long-distance migration, and environmental spatial awareness in animals.

Disclosures: S. M. Reppert: None.

Special Lecture

276. Development and Reprogramming of Neuronal Diversity in the Central Nervous System

Location: Hall B1

Time: Mon, Oct. 19, 2015, 11:30 AM - 12:40 PM

Support: NIH grant NS062849

NIH grant NS073124

NIH grant NS078164

NYSCF grant- Robertson Investigator Award

Speakers: *P. ARLOTTA;
Stem Cell and Regenerative Biol., Harvard Univ., Cambridge, MA

Abstract: The mammalian central nervous system (CNS) contains an unparalleled diversity of neuronal subtypes, which are largely generated during embryonic development and maintained unchanged in the adult. This lecture will cover progress made in understanding the regulatory, molecular logic that shapes neuronal diversity in the embryo, consider its importance for CNS

assembly and function, and discuss recent evidence for the unexpected capacity of central neurons to post-mitotically "reprogram" their class-specific features.

Disclosures: P. Arlotta: None.

David Kopf Lecture on Neuroethics

277. Giving Voice to Consciousness: Neuroethics, Human Rights, and the Indispensability of Neuroscience

Location: Hall B1

Time: Mon, Oct. 19, 2015, 10:00 AM - 11:10 AM

Support: Support contributed by: David Kopf Instruments

Health Policy Investigator Award Robert Wood Johnson Fdn

Buster Foundation

Jerold B. Katz Foundation

Charles A. Dana Foundation

James S. McDonnell Foundation

Weill Cornell CTSC NIH UL1-RR024966

Soros Project on Death in America Faculty Scholar Award

Speakers: *J. J. FINS;
Div. Med. Ethics, Weill Med. College, Cornell Univ., New York, NY

Abstract: The ability of neuroprosthetics to restore functional communication in patients with disorders of consciousness has the potential to reintegrate patients into the nexus of family and community. As a worthy scientific pursuit, Fins will argue that this effort is a moral imperative which links respect for persons with the reemergence of voice out of covert consciousness. As such, it is a human rights issue for a population too long marginalized. For rights to come to mind, patients will need greater access to medical care and research and the skilled engagement of the neuroscience community.

Disclosures: J. J. Fins: None.

Albert and Ellen Grass Lecture

366. Receptors, Neurons, and Circuits: The Biology of Mammalian Taste

Location: Hall B1

Time: Mon, Oct. 19, 2015, 3:15 PM - 4:25 PM

Support: Support contributed by The Grass Foundation

NIH R01 DA035025-03

NIH R01 NS076774-04

Speakers: C. ZUKER;
Howard Hughes Med. Institute, Columbia Univ., New York, NY

Abstract: The taste system is one of our fundamental senses, responsible for detecting and responding to sweet, bitter, umami, salty, and sour stimuli. Scientists study the logic of taste coding as a platform to understand how the brain creates an internal representation of the outside world and transforms sensory signals at the periphery into percepts, actions, and behaviors.

Disclosures: C. Zuker: Other; Scientific Co-founder of Senomyx and Elcelyx Therapeutics.

Presidential Special Lecture

367. Immune Mechanisms of Synapse Loss in Health and Disease

Location: Hall B1

Time: Mon, Oct. 19, 2015, 5:15 PM - 6:25 PM

Support: NIH RO1NS071008

NIH R01NS084298

CHDI Foundation

Coins for Alzheimer's Research Trust

The Broad Institute, Stanley Center

John Merck Scholars Program

Simons Foundation

Speakers: B. A. STEVENS;
Neurol., Boston Children's Hospital, Harvard Med. Sch., Boston, MA

Abstract: How synapses are eliminated in the developing and diseased brain remains a mystery. During development, synaptic pruning is required for precise wiring and emerging evidence implicates immune-related molecules and immune cells called microglia. This talk will review research on how these pathways regulate the formation, refinement, and elimination of specific axons and synapses during development. The discoveries suggest ways of protecting synapses in neurodegenerative and psychiatric disorders involving synapse loss.

Disclosures: B.A. Stevens: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Annexon. F. Consulting Fees (e.g., advisory boards); Annexon.

Special Lecture

452. Strange Synapses and Circuits of the Basal Ganglia

Location: Hall B1

Time: Tue, Oct. 20, 2015, 8:30 AM - 9:40 AM

Support: MH100568

NS046579

HHMI

Speakers: B. SABATINI;
Neurobio., Harvard Med. Sch., Boston, MA

Abstract: The basal ganglia are a phylogenetically old and evolutionarily conserved set of nuclei crucial for goal-oriented motor action. Nevertheless, many aspects of their circuitry, function, and regulation remain mysterious. Sabatini will present recent work from his group revealing complex and unexpected interactions between nuclei of the basal ganglia. These include the surprisingly widespread use of multiple fast acting neurotransmitters by neuromodulatory systems. The results will be discussed in terms of action initiation and reinforcement.

Disclosures: B. Sabatini: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); cofounder of Optogenix LLC.

Special Lecture

459. Uncertainty, Choice, and Dopamine

Location: Hall B1

Time: Tue, Oct. 20, 2015, 10:00 AM - 11:10 AM

Support: Canadian Institutes of Health Research MOP-133579

Speakers: *S. B. FLORESCO;
Univ. of British Columbia, Vancouver, BC, Canada

Abstract: We routinely evaluate choices where decisions and actions may or may not yield different types of rewards. These situations trigger competitive decision biases that reflect interplay between different prefrontal cortical, amygdalar, striatal, and habenular nodes within dopaminergic circuitry. This lecture will discuss some of the interactions between these circuits that shape decision biases and underlie conflicting urges when evaluating options that vary in terms of potential risks and rewards.

Disclosures: S.B. Floresco: B. Contracted Research/Research Grant (principal investigator for a drug study, collaborator or consultant and pending and current grants). If you are a PI for a drug study, report that research relationship even if those funds come to an institution.; Contract and consulting work for Pfizer Inc.. Other; Pfizer.

Special Lecture

460. Cortical Control of Arm Movements: A Dynamical Systems Perspective

Location: Hall B1

Time: Tue, Oct. 20, 2015, 11:30 AM - 12:40 PM

Support: NIH NINDS EUREKA Grant 1-R01-NS066311

NIH NINDS Grant Supplement to 1-R01-NS066311

NIH Director's Pioneer Award 1-DP1-OD006409

DARPA DSO/BTO Grant N66001-10-C-2010

NIH NINDS T-R01NS076460

Simons Foundation

Burroughs Wellcome Foundation

Speakers: *K. V. SHENOY;
Electric Engineer & Neurosci, Stanford Univ., Stanford, CA

Abstract: Our ability to move is central to everyday life. Investigating the neural control of movement in general, and the cortical control of volitional arm movements in particular, has been a major research focus in recent decades. Studies have involved primarily either attempts to account for single-neuron responses in terms of tuning for movement parameters or attempts to decode movement parameters from populations of tuned neurons. Even though this focus on encoding and decoding has led to many seminal advances, it has not produced an agreed-upon conceptual framework. Interest in understanding the underlying neural dynamics has recently increased, leading to questions such as how does the current population response determine the future population response, and to what purpose? We review how a dynamical systems perspective may help us understand why neural activity evolves the way it does, how neural activity relates to movement parameters, and how a unified conceptual framework may result.

Disclosures: K. V. Shenoy: None.

Special Lecture

545. Inhibition and Excitation in the Cerebellar Nuclei

Location: Hall B1

Time: Tue, Oct. 20, 2015, 1:00 PM - 2:10 PM

Speakers: *I. M. RAMAN;
Neurobio., Northwestern Univ., Evanston, IL

Abstract: Neurons in the cerebellar nuclei integrate high-frequency inhibition from convergent Purkinje cells with excitation from diverse mossy fibers to generate cerebellar outputs that lead to regulation of precise motor behaviors. This lecture will include a discussion of the synaptic and cellular specializations of Purkinje neurons, mossy fibers, and neurons of the cerebellar nuclei that contribute to information coding by the cerebellum in mice.

Disclosures: I. M. Raman: None.

Special Presentation

553. Title TBD

Location: Hall B1

Time: Tue, Oct. 20, 2015, 4:00 PM - 5:00 PM

Speakers: F. COLLINS;
NIH, Washington, DC

Abstract: TBD

Disclosures: F. Collins: None.

Presidential Special Lecture

554. Grid Cells and Cortical Maps for Space

Location: Hall B1

Time: Tue, Oct. 20, 2015, 5:15 PM - 6:25 PM

Support: ERC

Research Council of Norway

Kavli Foundation

Speakers: *M.-B. MOSER;
Kavli Inst. for Systems Neurosci. and Ctr. for Neural Computation, Norwegian Univ. of Sci. and Technol., Trondheim, Norway

Abstract: The medial entorhinal cortex (MEC) is part of the brain's circuit for dynamic representation of self-location. The metric of this representation is provided by grid cells - cells with spatial firing fields that tile environments in a periodic hexagonal pattern. This lecture will discuss the morphological identity of cells that express this pattern, how they are organized, how they interact with the environment, and how grid cells and place cells contribute to a wider circuit for goal-directed navigation.

Disclosures: M.-B. Moser: None.

Special Lecture

639. The Genetic Logic of Synapse Formation and Axon Regeneration

Location: Hall B1

Time: Wed, Oct. 21, 2015, 8:30 AM - 9:40 AM

Support: NIH Grant NINDS 035546

NIH Grant NINDS 057317

Howard Hughes Medical Institute

Speakers: *Y. JIN;
Div. Biolog Sci, Neurobio. Section, Howard Hughes Med. Institute, Univ. of California-San Diego, LA JOLLA, CA

Abstract: Genetic dissection in *C. elegans* has long been a powerful approach to discover the function of genes and to elucidate the molecular and cellular network underlying how synapses form and function. Recent technological innovation using laser surgery of single axons and *in vivo* imaging has also made *C. elegans* a new model for axon regeneration. Importantly, genes regulating synaptogenesis and axon regeneration are highly conserved in function across animal phyla. This lecture will focus on the key findings and discuss implications to human health.

Disclosures: Y. Jin: None.

Special Lecture

645. Striatal Synaptic Dysfunction in Parkinson's and Huntington's Diseases

Location: Hall B1

Time: Wed, Oct. 21, 2015, 10:00 AM - 11:10 AM

Support: NIH NS34696

The JPB Foundation

CHDI Foundation

Speakers: *D. SURMEIER;
Prof. Dept. Physiology/ NUIN, Northwestern Univ. Med. Sch., Chicago, IL

Abstract: Traditional models of basal ganglia disorders are grounded in the assumption that network dysfunction is driven by alterations in intrinsic excitability of striatal neurons. Recent work has challenged this assumption, showing that mouse models of Parkinson’s disease have profound cell-specific alterations in striatal synaptic strength and connectivity. Cell-specific synaptic dysfunction also is being found in mouse models of Huntington’s disease. This talk will summarize this work and link it to the motor symptoms of these two diseases.

Disclosures: D. Surmeier: None.

Special Lecture

646. A Causal Analysis of the Attentional Network

Location: Hall B1

Time: Wed, Oct. 21, 2015, 11:30 AM - 12:40 PM

Support: NIH Grant EY017921

NIH Grant EY017292

NSF Grant CCF 1317348

NSF Grant CCF-1231216

McGovern Institute

Poitras Center

Speakers: *R. DESIMONE;
MIT, McGovern Inst. Brain Res. at MIT, CAMBRIDGE, MA

Abstract: The most behaviorally-relevant stimuli in scenes are selected for processing and control over behavior (“attention”). The effects of selection are widespread, making it difficult to distinguish cause from effect in the attentional network. However, the flow of control can be inferred through the analysis of timing and the use of “causal” methods such as pharmacological inactivation and optogenetics to establish the impact of one circuit on another. This lecture will explore the emerging new insights into the biological mechanism of attention.

Disclosures: R. Desimone: None.

Special Lecture

737. Neurocircuitry Controlling Feeding and Drinking Behaviors in Mice

Location: Hall B1

Time: Wed, Oct. 21, 2015, 1:00 PM - 2:10 PM

Support: NIH Grant R01 - DA24908

HHMI

Speakers: *R. PALMITER;
Univ. of Washington, Seattle, WA

Abstract: The development of genetic, viral, and optical technologies has revolutionized approaches for dissecting neuronal circuits that control basic behaviors and physiological process, including ingestion. Selective activation of specific neurons stimulates robust feeding or drinking, while activation of other neurons inhibits feeding or drinking. Deciphering the neuronal circuits engaged by these manipulations and the molecular phenotype of neurons involved is an ongoing endeavor.

Disclosures: R. Palmiter: None.