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## **Lecture**

### **L01. Special Lecture — Lineage-Dependent Assembly of the Neocortex**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 10:00 AM - 10:45 AM

**Description:** The ability of the neocortex to command higher-order brain functions depends on the intricate circuitry comprised of a vast number of diverse neurons. The principles and mechanisms that control the assembly and operation of neocortical circuits remain poorly understood. This lecture will focus on the search for the common commodities of neocortical circuits at both the structural and functional levels, with the premise that developmental origin and lineage relationships guide the formation of neuronal ensembles for functional circuit assembly.

Lineage-Dependent Assembly of the Neocortex

**Authors:** \*S.-H. SHI

Tsinghua Univ., Beijing, China

**Disclosures:** S. Shi: None.

## **Lecture**

### **L02. Special Lecture — The Assembly and Function of Hippocampal Circuits**

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 11:00 AM - 11:45 AM

**Description:** Our memories are stored in the hippocampus. This lecture will aim at describing how and when the circuits used to store memories emerge during development. It will show how studying early development, from embryonic neurogenesis to perinatal neuronal maturation, provides an interesting framework to gain understanding of mature hippocampal function at the circuit level.

L02. The Assembly and Function of Hippocampal Circuits

**Authors:** \*R. COSSART

INMED, INSERM U1249, Marseille, France

**Abstract:** Our memories are stored in the hippocampus. This lecture will aim at describing how and when the circuits used to store memories emerge during development. It will show how studying early development, from embryonic neurogenesis to perinatal neuronal maturation, provides an interesting framework to gain understanding of mature hippocampal function at the circuit level.

**Disclosures:**

## **Lecture**

### **L03. Albert and Ellen Grass Lecture — From the Neuroscience of Individual Variability to Climate Change**

**Location:** Virtual Only

**Time:** Tuesday, November 9, 2021, 2:30 PM - 3:15 PM

**Description:** Years of basic neuroscience research on the modulation of the small circuits found in the crustacean stomatogastric ganglion (STG) have led us to study the effects of temperature and other perturbations on the motor patterns produced by the STG. While the impetus for this work was the study of individual variability in the parameters determining intrinsic and synaptic conductances, we are confronting substantial fluctuations in the stability of the networks to extreme temperature; these may correlate with changes in ocean temperature. When studied under control conditions, these wild-caught animals appear to be unchanged, but it is only when challenged by extreme temperatures that we reveal the consequences of warming ocean temperatures.

From the Neuroscience of Individual Variability to Climate Change

**Authors: \*E. E. MARDER**

Volen Ctr. and Biol. Dept., Volen Ctr., Waltham, MA

**Disclosures: E.E. Marder:** None.

## **Lecture**

### **L04. Special Lecture — Proteins, Synapses, and Memory — Local Translation of mRNAs in Neurons**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 11:00 AM - 11:45 AM

**Description:** An individual neuron in the brain possesses approximately 10,000 synapses, many of which are hundreds of microns away from the cell body and can process independent streams of information. During synaptic transmission and plasticity, remodeling of the local proteome occurs via the regulated synthesis and degradation of proteins. This lecture will describe recent studies aimed at understanding the unique features of neuronal mRNAs and the interesting mechanisms neurons exploit to supply and regulate synaptic proteins.

Proteins, Synapses, and Memory — Local Translation of mRNAs in Neurons

**Authors: E. M. SCHUMAN**

Max Planck Inst. for Brain Res., Frankfurt, Germany

**Disclosures:**

## Lecture

### L05. CLINICAL NEUROSCIENCE LECTURE— Expanding Mechanisms and Therapeutic Targets for Neurodegenerative Disease

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 11:00 AM - 11:45 AM

**Description:** This lecture will discuss our progress in using genetic screens in simple model organisms to define mechanisms of human neurodegenerative diseases, such as amyotrophic lateral sclerosis (ALS). In addition to teaching us about the cellular and molecular defects underpinning neurodegenerative disease, these screens have revealed novel human disease genes and new therapeutic targets.

L05. Expanding Mechanisms and Therapeutic Targets for Neurodegenerative Disease

**Authors: \*A. D. GITLER**

Genet., Stanford Univ., Stanford, CA

**Abstract:** My goal is to discover the cellular and molecular mechanisms by which protein aggregates contribute to neurodegeneration and to harness these mechanisms to devise novel therapeutic strategies. We use the baker's yeast, *Saccharomyces cerevisiae*, as a simple, yet powerful, model system to study the cell biology underpinning protein-misfolding diseases, which include Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (ALS). We are focusing on the ALS disease proteins TDP-43 and FUS/TLS, as well as the *C9orf72* gene. We have generated yeast models to define mechanisms by which mutations in these genes might cause ALS. Because these proteins aggregate and are toxic in yeast, we have used these yeast models to perform high-throughput genomewide modifier screens to discover suppressors and enhancers of toxicity. Launching from the studies in yeast, we have extended our findings into animal models and even recently into human patients. For example, we discovered mutations in one of the human homologs of a hit from our yeast TDP-43 modifier screen in ALS patients. Mutations in this gene are relatively common (~5% of cases) making it one of the most common genetic risk factors for ALS discovered to date. These screens are also providing new and completely unexpected potential drug targets, underscoring the power of such simple model systems to help reveal novel insight into human disease.

**Disclosures: A.D. Gitler:** E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Maze Therapeutics. F. Consulting Fees (e.g., advisory boards); Maze Therapeutics, Prevail Therapeutics, Bridgebio, Third Rock Ventures.

## Lecture

### L06. Presidential Special Lecture — Spinal Cord Gateways to Restore Neurological Functions

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 3:30 PM - 4:30 PM

**Description:** A spinal cord injury disrupts the communication between the brain and spinal cord, leading to unrecoverable deficits in motor and autonomic functions. This lecture will discuss the mechanisms that enable electrical spinal cord stimulation to target the circuits controlling these functions. This understanding translated into targeted neuromodulation therapies that restore walking after paralysis and precisely controlled hemodynamics in people suffering from orthostatic hypotension.

Spinal Cord Gateways to Restore Neurological Functions

**Authors: \*G. COURTINE**

Swiss Federal Inst. of Technol. Lausanne, Univ. Hosp. of Lausanne, Geneva, Switzerland

**Award Winner:** Donald B. Lindsley Prize in Behavioral Neuroscience

**Authors: \*S. VYAS**

Columbia Univ., New York, NY

**Award Winner:** Nemko Prize in Cellular or Molecular Neuroscience

**Authors: \*D. TINGLEY**

Neurosci., NYU Neurosci. Inst., New York, NY

**Award Winner.**

**Authors: \*N. LI**

Baylor Col. of Med., Houston, TX

**Award Winner:** Peter and Patricia Gruber International Research Award Co-Recipient #1

**Authors: \*M. OZEL**

New York Univ., New York, NY

**Award Winner:** Peter and Patricia Gruber International Research Award Co-Recipient #2

**Authors: \*A. OLIVA**

Columbia Univ., New York, NY

**Award Winner:** Jennifer N. Bourne Prize in Brain Ultrastructure Co-Recipient #1

**Authors: \*W.-C. LEE**

BCH / Harvard Med. Sch., Boston, MA

**Award Winner:** Jennifer N. Bourne Prize in Brain Ultrastructure Co-Recipient #2

**Authors: \*C. IMIG**

Mol. Neurobio., Dept. of Neuroscience, Univ. of Copenhagen, Copenhagen, Denmark

**Lecture**

**L07. Special Lecture — Itchy and Scratchy — Molecular and Cellular Mechanisms of Acute and Chronic Itch**

**Location:** Virtual Only

**Time:** Thursday, November 11, 2021, 1:30 PM - 2:15 PM

**Description:** Itch is defined as an unpleasant sensation that evokes a desire to scratch. In contrast to acute itch that is transient and protective, chronic itch is a persistent, debilitating condition for which there are few treatment options. This lecture will discuss studies aimed at understanding the neural mechanisms that mediate acute itch and chronic itch associated with skin, systemic and neurological disorders.

L07. Itchy and Scratchy — Molecular and Cellular Mechanisms of Acute and Chronic Itch

**Authors: \*D. M. BAUTISTA**

Mol. and Cell Biol., Univ. of California, Berkeley, Berkeley, CA

**Abstract:** Itch is defined as an unpleasant sensation that evokes a desire to scratch. In contrast to acute itch that is transient and protective, chronic itch is a persistent, debilitating condition for which there are few treatment options. This lecture will discuss studies aimed at understanding the neural mechanisms that mediate acute itch and chronic itch associated with skin, systemic and neurological disorders.

**Disclosures: D.M. Bautista:** None.

**Lecture**

**L08. Special Lecture — Themes and Variations: Circuit Mechanisms of Behavioral Evolution**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 10:00 AM - 10:45 AM

**Description:** Animals display enormous diversity in their behaviors, yet little is known about how nervous systems evolve to produce such variation. Recent advances have ushered in a new era in comparative neurobiology, making it possible to directly compare neural circuits in closely related species and glean insight into the origins and underpinnings of behavioral diversity. This lecture will describe how applying such a comparative approach to the study of courtship behaviors in different *Drosophila* species has revealed the types of circuit changes that can fuel behavioral evolution.

Themes and Variations: Circuit Mechanisms of Behavioral Evolution

**Authors: \*V. RUTA**

The Rockefeller Univ., New York, NY

**Disclosures: V. Ruta:** None.

**Lecture**

### **L09. Special Lecture — Learning From Prediction Error**

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 10:00 AM - 10:45 AM

**Description:** Can artificial intelligence be a useful guide for how the brain learns? This lecture will examine the cerebellum, a major learning site in which neurons group into populations that learn from prediction errors. Unlike an artificial network, neuronal populations respond only to a specific region of the error space. Thus, the fundamental unit of learning is not a single neuron, but a population of neurons that share a single teacher. The result is a memory that exhibits remarkable features: multiple timescales and resistance to erasure.

**Authors: \*R. SHADMEHR**

Dept. of Biomed. Engin., Johns Hopkins Univ., Baltimore, MD

**Abstract:** Artificial intelligence provides algorithms for learning, but can the algorithms be a guide for how the brain learns? In the cerebellum, neurons group into populations that learn from prediction errors. However, unlike an artificial network, each neuron responds only to a specific region of the error space. Furthermore, the neurons that learn from the same error signal appear to group together into a single population. Thus, unlike an artificial neural network, in the cerebellum the fundamental unit of learning is not a single neuron, but a population of neurons that share a single teacher. This anatomical design of the cerebellum produces a learning system that exhibits remarkable features of memory: multiple timescales, resistance to erasure, and spontaneous recovery.

**Disclosures:**

**Lecture**

### **L10. Presidential Special Lecture — From Development to Repair — How Growth Factors and Stem Cells Build the Brain**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 8:30 AM - 9:30 AM

**Description:** This lecture will focus on how neural stem cells build the mammalian brain and generate the right numbers and types of neurons and glial cells, and how this can go awry in

response to genetic or environmental perturbations associated with neurodevelopmental disorders. This lecture will also describe how knowledge gained from these basic studies can lead to brain repair strategies based on harnessing endogenous neural precursor cells in the human brain.

From Development to Repair — How Growth Factors and Stem Cells Build the Brain

**Authors: \*F. D. MILLER**

Univ. of British Columbia, Vancouver, BC, Canada

**Disclosures:**

**Award Winner:** Award for Education in Neuroscience, Co-Recipient #1

**Authors: \*G. E. HUE**

Neuroscience& Behavioral Biol., Emory Univ., Atlanta, GA

**Award Winner:** Award for Education in Neuroscience, Co-Recipient #2

**Authors: \*A. KHAN**

The Univ. of Texas at El Paso, El Paso, TX

**Award Winner:** Science Educator Award

**Authors: \*S. L. MACKNIK**

SUNY Downstate Hlth. Sci. Univ., Brooklyn, NY

**Award Winner:** Science Educator Award

**Authors: \*D. L. YOURICK**

Headquarters, Walter Reed Army Inst. of Res., Silver Spring, MD

**Award Winner:** Net Generation (Pre/Post Doctorate)

**Authors: \*S. A. NETTLES**

Neurosci., Washington Univ. In St. Louis, Saint Louis, MO

**Award Winner:** Next Generation (Junior Faculty)

**Authors: \*H. MARUSAK**

Wayne State Univ., Detroit, MI

**Lecture**

**L11. Presidential Special Lecture — Integration of Mind and Metabolism**

**Location:** Virtual Only

**Time:** Thursday, November 11, 2021, 3:30 PM - 4:30 PM

**Description:** Current and traditional models of ingestive behavior implicate the consciously perceived hedonic qualities of food as driving overeating, whereas subliminal signals arising from the gut serve to curb our uncontrolled desire for calories. This lecture will present recent evidence suggesting that the inverse is true. More specifically, evidence from human and animal models will be presented demonstrating that it is the subliminal metabolic signals that ascend to the brain to regulate not only food reinforcement, but also perception and higher order cognitive functions.

#### L11. Integration of Mind and Metabolism

**Authors: \*D. M. SMALL**

Yale Univ., New Haven, CT

**Disclosures:**

**Award Winner:** Bernice Grafstein Award for Outstanding Accomplishments in Mentoring Recipient

**Authors: \*J. BECKER**

Univ. of Michigan, Ann Arbor, MI

**Award Winner:** Mika Salpeter Lifetime Achievement Recipient

**Authors: \*C. DULAC**

Harvard Univ., Cambridge, MA

**Award Winner:** Janett Rosenberg Trubatch Career Development Award Recipient #1

**Authors: \*S. A. LIDDELOW**

NYU Grossman Sch. of Med., New York, NY

**Award Winner:** Janett Rosenberg Trubatch Career Development Award Recipient #2

**Authors: \*L. A. O'CONNELL**

Stanford Univ., Stanford, CA

**Award Winner:** Patricia Goldman-Rakic Hall of Honor

**Authors: \*L. UNGERLEIDER**

Natl. Inst. of Mental Hlth., Bethesda, MD

#### Lecture

#### L12. Special Lecture — Stress Contagion, Shadows, and Synaptic Imprinting

**Location:** Virtual Only

**Time:** Thursday, November 11, 2021, 1:30 PM - 2:15 PM

**Description:** The ability of organisms to respond, cope, and adapt to stress is critical for survival. This lecture will focus on the canonical controllers of the endocrine response to stress, the hypothalamic corticotropin-releasing hormone (CRH) neurons. It will show how these cells are critical for gating innate defensive behaviors and transmitting stress to others. It will also provide insights into the mechanisms that allow for the opening of distinct temporal plasticity windows at synapses between CRH neurons after stress.

#### L12. Stress Contagion, Shadows, and Synaptic Imprinting

**Authors: \*J. S. BAINS**

Hotchkiss Brain Inst., Univ. of Calgary, Calgary, AB, Canada

**Abstract:** The ability organisms to respond, cope and adapt to stress is critical for survival. This lecture will focus on the canonical controllers of the endocrine response to stress, the hypothalamic CRH neurons. It will show these cells are critical for gating innate defensive behaviors and transmitting stress to others. It will also provide insights into the mechanisms that allow for the opening of distinct temporal plasticity windows at synapses onto CRH neurons after stress.

**Disclosures:**

#### Lecture

#### L13. Special Lecture — The Mysteries of Sleep — Toward the Molecular Substrate for "Sleepiness"

**Location:** Virtual Only

**Time:** Tuesday, November 9, 2021, 2:30 PM - 3:15 PM

**Description:** Sleep is a ubiquitous behavior in animal species with well-developed nervous systems. Despite the recent advances in understanding the executive neurocircuitry and neurochemistry for sleep/wake switching, the fundamental mechanism for homeostatic regulation of sleep, as well as the neural substrate for sleepiness (sleep need), remains mysterious. This lecture will discuss our exploratory approaches, including forward genetics in randomly mutagenized mice, for cracking open this uncharted black box.

The Mysteries of Sleep — Toward the Molecular Substrate for "Sleepiness"

**Authors: \*M. YANAGISAWA**

Univ. of Tsukuba, Tsukuba, Japan

**Abstract:** Although sleep is a ubiquitous behavior in animal species with well-developed central nervous systems, many aspects in the neurobiology of sleep remain mysterious. Our discovery of orexin, a hypothalamic neuropeptide involved in the maintenance of wakefulness, has triggered an intensive research examining the exact role of the orexinergic and other neural pathways in the regulation of sleep/wakefulness. The orexin receptor antagonist suvorexant, which

specifically block the endogenous waking system, has been approved as a new drug to treat insomnia. Also, since the sleep disorder narcolepsy-cataplexy is caused by orexin deficiency, orexin receptor agonists are expected to provide mechanistic therapy for narcolepsy; they will likely be also useful for treating excessive sleepiness due to other etiologies. Despite the fact that the executive neurocircuitry and neurochemistry for sleep/wake switching has been increasingly revealed in recent years, the mechanism for homeostatic regulation of sleep, as well as the neural substrate for "sleepiness" (sleep need), remains unknown. To crack open this black box, we have initiated a large-scale forward genetic screen of sleep/wake phenotype in mice based on true somnographic (EEG/EMG) measurements. We have so far screened >8,000 heterozygous ENU-mutagenized founders and established a number of pedigrees exhibiting heritable and specific sleep/wake abnormalities. By combining linkage analysis and the next-generation whole exome sequencing, we have molecularly identified and verified the causal mutation in several of these pedigrees. Biochemical and neurophysiological analyses of these mutations are underway. Since these dominant mutations cause strong phenotypic traits, we expect that the mutated genes will provide new insights into the elusive pathway regulating sleep/wakefulness. Indeed, through a systematic cross-comparison of the Sleepy mutants and sleep-deprived mice, we have recently found that the cumulative phosphorylation state of a specific set of mostly synaptic proteins may be the molecular substrate of sleep need.

**Disclosures:**

**Lecture**

**L15. Special Lecture — Brain Mechanisms Underlying the Vulnerability for Compulsive Drug Use**

**Location:** Virtual Only

**Time:** Thursday, November 11, 2021, 2:30 PM - 3:15 PM

**Description:** Substance use disorder (SUD) is characterized by heavy drug use despite harmful consequences. A key feature is that only a fraction of those exposed to addictive substances develops SUD. The individual variability seen in patients is also replicated in rodents. Our research investigates the factors and synaptic mechanisms that contribute to SUD vulnerability. We found that unbalance in the activation of dopamine D1 and D2 receptors in basal ganglia is a mechanism that contributes to vulnerability by promoting anatomical and functional alterations that enhance drug reward and reinforcement.

L15. Brain Mechanisms Underlying the Vulnerability for Compulsive Drug Use

**Authors:** \*V. A. ALVAREZ

Lab. of Neurobio. of Compulsive Behaviors, Natl. Inst. on Alcohol Abuse and Alcoholism, Bethesda, MD

**Disclosures:** V.A. Alvarez: None.

**Lecture**

## **L16. Special Lecture — Dentate Gyrus Circuits for Encoding, Retrieval, and Discrimination of Episodic Memories**

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 10:00 AM - 10:45 AM

**Description:** The dentate gyrus (DG) plays a key role in hippocampal memory formation. Recent studies recorded and manipulated the activity of different DG neuron types during memory tasks and provided exciting new insights into the mechanisms of DG computational processes, particularly those underlying the encoding, retrieval, and discrimination of similar memories. In this lecture, DG-dependent mnemonic functions will be brought to light. The mechanistic links between cellular properties and the computations performed by the DG will also be discussed.

L16. Dentate Gyrus Circuits for Encoding, Retrieval, and Discrimination of Episodic Memories

**Authors: \*M. BARTOS**

Univ. of Freiburg, Freiburg, Germany

**Abstract:** The dentate gyrus (DG) is the input stage of the hippocampal trisynaptic loop. It transforms entorhinal inputs into a sparse output pattern that is relayed to CA3. Lesion studies have implicated the DG in a variety of cognitive functions required to form memories. Intriguingly however, not all hippocampus dependent mnemonic functions are impaired by DG lesions. This indicates that it performs specific computations that are required only during certain operating modes of the hippocampus. There is a wealth of information about the network architecture and characteristics of the various DG neuron types and an extensive body of theoretical works has proposed ways in which these architectural elements may underlie DG's function in cognitive operations. However, until recently little data were available on the activity of identified neuron types in the DG of behaving animals. Recent studies have recorded and manipulated the activity of specific DG neuron classes during various memory tasks and their results signify major revisions of current models of DGs' computational function. Here, several DG-dependent behaviors will be brought in light of recent findings and attempt to explore possible mechanistic links between cellular as well as network activities and the computations carried out by the DG neuronal network.

**Disclosures: M. Bartos:** None.

### **Lecture**

## **L17. Special Lecture — Neuronal Mechanisms of Value-Based Decision-Making — A Brain-Machine Interface Approach**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 11:00 AM - 11:45 AM

**Description:** Decision-making is an unobservable cognitive process, making it challenging to investigate the underlying neuronal mechanisms. This lecture will discuss how techniques borrowed from the brain-machine interface field, such as decoding population activity and closed-loop control, can be used to understand how cognitive processes such as decision-making are implemented at the neuronal level. This approach could also lead to the development of novel devices for the treatment of neuropsychiatric disorders that involve impaired decision-making.

Neuronal Mechanisms of Value-Based Decision-Making — A Brain-Machine Interface Approach

**Authors: \*J. D. WALLIS**

Univ. of California, Berkeley, Berkeley, CA

**Disclosures:**

**Lecture**

**L18. Special Lecture — Watching the Brain in Action — Creating Tools for Functional Analysis of Neural Circuitry**

**Location:** Virtual Only

**Time:** Tuesday, November 9, 2021, 1:30 PM - 2:15 PM

**Description:** This lecture will focus on recent developments of genetically encoded indicators for neurotransmitters with an in-depth look at the design and screening of these sensors. Novel applications enabled by these optical probes will be discussed, as well as practical concerns in behaving animals. In combination with calcium/voltage imaging and optogenetics, the fast-growing sensor toolbox is well-poised to permit direct functional analysis of how chemical input signaling mediates the plasticity and function of brain circuits.

Watching the Brain in Action — Creating Tools for Functional Analysis of Neural Circuitry

**Authors: \*L. TIAN**

Biochem. and Mol. Med., Univ. of California, Davis, Davis, CA

**Disclosures:**

**Lecture**

**L19. David Kopf Neuroethics Lecture — The Law and Neuroprosthetics**

**Location:** Virtual Only

**Time:** Tuesday, November 9, 2021, 1:30 PM - 2:15 PM

**Description:** Neuroprostheses replace or enhance motor, sensory and mental functions through interactions with the nervous system, raising novel ethical and legal issues. The use of adaptive

systems and machine learning expand the range of possible applications while clouding questions of control, ownership and responsibility. This lecture will consider issues related to ownership; design defects; freedom to or from use; responsibility; privacy; and the use of neural data derived from neuroprostheses.

The Law and Neuroprosthetics

**Authors: J. A. CHANDLER**

Univ. of Ottawa, Ottawa, ON, Canada

**Disclosures:**

**Lecture**

**L20. Dialogues Between Neuroscience and Society — Life of a Neuron**

**Location:** Virtual Only

**Time:** Tuesday, November 9, 2021, 3:30 PM - 4:30 PM

**Description:** The SfN-ARTECHOUSE collaboration Life of a Neuron exhibit is an innovative approach to exploring the great mystery that is the human brain. As the co-founder of ARTECHOUSE, Sandro's experience with innovative, technology-driven storytelling acts as a bridge between art and science. This exhibit aims to immerse the audiences into the limitless possibilities of human creativity and imagination.

**Award Winner:** Peter Seeburg Integrative Neuroscience Prize

**Authors: \*P. JONAS**

Inst. of Sci. and Technol. (IST) Austria, Klosterneuburg, Austria

**Award Winner:** Julius Axelrod Prize

**Authors: \*D. GINTY**

Harvard Med. Sch., MA

**Award Winner:** Jacob P. Waletzky Award

**Authors: \*M. LOBO**

Anat. and Neurobio., Univ. of Maryland Baltimore, Baltimore, MD

**Award Winner:** Swartz Prize for Theoretical and Computational Neuroscience

**Authors: \*N. BRUNEL**

Duke Univ., Durham, NC

**Award Winner:** Ralph W. Gerard Prize in Neuroscience Co-Recipient #1

**Authors: \*J. H. KAAS**

Psychology, Vanderbilt Univ., Nashville, TN

**Award Winner:** Ralph W. Gerard Prize in Neuroscience Co-Recipient #2

**Authors: \*R. DESIMONE**

McGovern Institute, MIT, Cambridge, MA

**Title:** Speaker

**Authors: R. Kim**

Artechouse, Washington, DC.

L20.01. Life of a Neuron

**Authors: \*S. KERESLIDZE**

ARTECHOUSE, Washington, DC

L20.02. Speaker

**Title:** Life of a Neuron

**Authors: J. Morrison**

UC Davis, Davis, CA.

**Lecture**

**L21. History of Neuroscience — A New Department, a New Transmitter Compound, a War and a Movement — The 1960s to the Present of a Neurobiology Career**

**Location:** Virtual Only

**Time:** Thursday, November 11, 2021, 2:30 PM - 3:15 PM

**Description:** No field called neuroscience existed in 1960. Only two substances, acetylcholine and norepinephrine, were accepted as neurotransmitters, and only overseas — in the U.S., prominent investigators believed that communication between nerve cells and targets was entirely electrical. Two international conferences concluded that GABA was not a transmitter compound, while a devastating war and a civil rights movement were disrupting American society. This lecture will describe the studies that completed the evidence required to demonstrate that GABA was a transmitter compound and discuss our responsibility as scientists in great societal events that divide the nation.

L21. A New Department, a New Transmitter Compound, a War and a Movement — The 1960s to the Present of a Neurobiology Career

**Authors: \*E. A. KRAVITZ**

Dept Neurobio, Harvard Med. Sch., Boston, MA

**Disclosures:**

**Lecture**

**L22. Presidential Special Panel — COVID and the Brain**

**Location:** Virtual Only

**Time:** Monday, November 8, 2021, 8:30 AM - 9:30 AM

**Speakers: \*I. J. KORALNIK**

Feinberg Sch. of Med., Northwestern Univ., Chicago, IL

**Speakers: \*S. SPUDICH**

Dept. of Neurol., Yale Univ. Sch. of Med., New Haven, CT

**Abstract:** Though originally considered primarily a respiratory pathogen, many people infected with SARS-CoV-2 suffer from nervous system complications during acute infection. Moreover, neurologic and psychiatric symptoms may persist months after initial infection in COVID-19 “long haulers”. This session will summarize the state of knowledge of the mechanisms of nervous system involvement in acute COVID-19, as well as the epidemiology and nascent understanding of neurologic post-acute sequelae of SARS-CoV-2 infection (Neuro-PASC), affecting millions of people in the U.S.

**Disclosures:**

**Lecture**

**L23. PETER AND PATRICIA GRUBER LECTURE: Neurogenetics: From Perception to Cognition - Christine Petit, Christopher A. Walsh**

**Location:** Virtual Only

**Time:** Wednesday, November 10, 2021, 12:00 PM - 1:30 PM

**Description:** *From the Genetics of Hearing Impairment to the Nascent Field of Auditory Therapy*

Christine Petit, MD, PhD

Hearing Institute, Institut Pasteur, Inserm, Paris, France

By laying the groundwork for deciphering the cellular and molecular mechanisms of hearing, the identification of the genes responsible for deafness has conferred a molecular dimension on this sensory system, particularly for the cochlea. Proof-of-concept has now been obtained for the treatment of several forms of deafness in mouse models, paving the way for clinical trials of cochlear gene therapy. The results obtained and the pros and cons of each of the strategies used will be discussed, together with the challenges that lie ahead at both the peripheral and central levels.

*Supported by FPA IDA05/Fondation pour l’Audition*

### *Genetic Architecture of the Human Cerebral Cortex*

Christopher A. Walsh, MD, PhD

Harvard Medical School, Boston Children's Hospital, and the Howard Hughes Medical Institute

Technical advances in gene mapping and sequencing allow study of the human cerebral cortex in ways inconceivable a few years ago. Humans show mutations in essentially every gene that constructs our brain, enlightening developmental mechanisms and how evolution played on our genome to sculpt the unique human brain. High throughput sequencing shows that every neuron in our brain has a unique genome, with somatic mutations indelibly marking its developmental origin and even its life history.

L23.01. From the Genetics of Hearing Impairment to the Nascent Field of Auditory Therapy

**Authors: \*C. PETIT**

Professor at Inst. Pasteur, Founding Director of the Hearing Institute, Professor Emeritus at Col. de France, Paris 15, France

**Abstract:** By laying the groundwork for deciphering the cellular and molecular mechanisms of hearing, the identification of the genes responsible for deafness has conferred a molecular dimension on this sensory system, particularly for the cochlea. Proof-of-concept has now been obtained for the treatment of several forms of deafness in mouse models, paving the way for clinical trials of cochlear gene therapy. The results obtained and the pros and cons of each of the strategies used will be discussed, together with the challenges that lie ahead at both the peripheral and central levels. *Supported by FPAIDA05/Fondation pour l'Audition*

**Disclosures:**

L23.02. Genetic Architecture of the Human Cerebral Cortex

**Authors: \*C. A. WALSH**

Harvard Med. School, Boston Children's Hospital, and the Howard Hughes Med. Inst., Boston, MA

**Abstract:** Technical advances in gene mapping and sequencing allow study of the human cerebral cortex in ways inconceivable a few years ago. Humans show mutations in essentially every gene that constructs our brain, enlightening developmental mechanisms and how evolution played on our genome to sculpt the unique human brain. High throughput sequencing shows that every neuron in our brain has a unique genome, with somatic mutations indelibly marking its developmental origin and even its life history.

**Disclosures:**