Piezo1 and Piezo2 are mechanically activated channels including mechanosensation, sound, and shear stress (cardiovascular tone), etc. Pain (somatosensation), sound (hearing), which can be identified using functional genomics, remains elusive. The lecture focuses on the molecular mechanisms involved in sensing mechanical force and how these proteins mediate membrane fusion at the synapse.

**Theme A: Development**

Building a Synapse Through Nuclear Export of Large RNA Granules and Exosomes

Vivian Budnik, PhD
University of Massachusetts Medical School

Monday, Nov. 17, 8:30–9:40 a.m.

Studies in Drosophila are uncovering novel conserved mechanisms for synapse development and plasticity. These include signaling pathways from the membrane to the nucleus, promoting the nuclear assembly and export of ribonucleoprotein granules and their synaptic localization. In addition, pre- and postsynaptic compartments are shaped through transsynaptic transmission of exosomes carrying transmembrane proteins and RNA. This lecture shares lessons from the study of viruses and Wnt signaling that lead to these discoveries and highlights their importance in disease.

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

How Do You Feel? The Role of Mechanically Activated Ion Channels in Touch, Pain, Hearing, and Beyond

Ardem Patapoutian, PhD
The Scripps Research Institute, Howard Hughes Medical Institute

Tuesday, Nov. 18, 11:30 a.m.–12:40 p.m.

Mechanosensation is perhaps the last sensory modality not understood at the molecular level. Ion channels that sense mechanical force are postulated to play critical roles in sensing touch/pain (somatosensation), sound (hearing), shear stress (cardiovascular tone), etc. However, the identity of ion channels involved in mechanical force has remained elusive. This lecture focuses on the identification, using functional genomic approaches, and characterization of novel mechanically activated channels including Piezo1 and Piezo2.

Exocytosis of Synaptic Vesicles: A Molecular Perspective

Reinhard Jahn, PhD
Max Planck Institute for Biophysical Chemistry, Germany

Wednesday, Nov. 19, 8:30–9:40 a.m.

Neurotransmitter release from neurons is mediated by Ca\(^{2+}\)-dependent exocytosis of synaptic vesicles. The molecular machinery involves SNARE proteins that carry out membrane fusion together with other conserved proteins such as SM and CATCHR. Furthermore, specialized proteins such as synaptotagmins and complexins convey Ca\(^{2+}\) regulation. Jahn will discuss new insight on the mechanisms by which these proteins mediate membrane fusion at the synapse.

**Theme C: Disorders of the Nervous System**

The Glymphatic System and Its Possible Roles in CNS Diseases

Maiken Nedergaard, MD, DMSc
University of Rochester

Sunday, Nov. 16, 10–11:10 a.m.

Past work has focused on cellular recycling of proteins involved in neurodegeneration. This lecture expands the traditional framework to include a macroscopic clearance system — the glymphatic system — by which the brain exports waste products of neural metabolism. Glymphatic clearance is driven by convective CSF influx and is especially active during sleep. Macromolecules, such as amyloid beta, are literally swept out of CNS for ultimate degradation in the liver. As such, the glymphatic system represents a novel and unexplored target for treatment of neurological disease.

**Theme D: Sensory and Motor Systems**

The Brain Is Needed to Cure Spinal Cord Injury

Kim Quang Do, PhD
Center for Psychiatric Neuroscience, Lausanne University Hospital, Switzerland

Monday, Nov. 17, 10–11:10 a.m.

Recovery after neuronal damage is learned by the spared neural systems. Isa’s research team is studying the mechanism of recovery of hand dexterity after partial spinal cord injury using nonhuman primate models by combining multidisciplinary approaches, such as kinetic analysis, electrophysiology, brain imaging, neuroanatomy, and genetic
Manipulation with viral vectors. Isa will talk about the large-scale circuit reorganization that occurs through training and is critical for recovery, spanning over the spinal cord, motor cortices, and even the limbic structures.

**Learning and Relearning Movement CME**

**Amy J. Bastian, PhD**

Kennedy Krieger Institute, Johns Hopkins University School of Medicine

Tuesday, Nov. 18, 8:30–9:40 a.m.

Human motor learning depends on a suite of brain mechanisms that are driven by different signals and operate on timescales ranging from minutes to years. Understanding these processes requires identifying how new movement patterns are normally acquired, retained, and generalized, as well as the effects of distinct brain lesions. The lecture focuses on normal and abnormal motor learning and how we can use this information to improve rehabilitation for individuals with neurological damage.

**The Sensory Neurons of Touch CME**

**David D. Ginty, PhD**

Harvard Medical School, Howard Hughes Medical Institute

Wednesday, Nov. 19, 11:30 a.m.–12:40 p.m.

The somatosensory system endows us with enormous capacity for object recognition, texture discrimination, sensory-motor feedback, and social exchange. Innocuous touch of the skin is detected by physiologically distinct low-threshold mechanosensory neurons (LTMRs). Ginty’s research team has amassed a genetic toolbox that enables interrogation of the physiology, morphology, and function of LTMR subtypes and their synaptic target neurons in the spinal cord. Ginty will discuss morphological and physiological features of LTMRs and the organizational logic of LTMR projections and circuits in the central nervous system.

**Theme E: Integrative Systems: Neuroendocrinology, Neuroimmunology, and Homeostatic Challenge**

**What Drives Sleep-Wake Cycles: Identification of Molecules and Circuits in Drosophila CME**

**Amita Sehgal, PhD**

Perelman School of Medicine at the University of Pennsylvania, Howard Hughes Medical Institute

Sunday, Nov. 16, 8:30–9:40 a.m.

This lecture focuses on the cellular and molecular mechanisms that regulate sleep. The 24-hour rhythm of sleep is driven by a circadian clock, while the need to sleep comes from a homeostatic system, which ensures adequate sleep levels. The lecture shows how the use of *Drosophila* has led to the identification of mechanisms that generate a circadian clock and to some of the downstream circuitry required for circadian timing of behavior. It also highlights recent developments in identifying molecular components and cellular circuits that underlie homeostatic regulation.

**Surprising Origins of Sex Differences in the Brain CME**

**Margaret M. McCarthy, PhD**

University of Maryland School of Medicine

Sunday, Nov. 16, 1–2:10 p.m.

Brain sex differences are established early by genes, hormones, environment, and experience. Animal models reveal multiple endpoints modified by steroid hormones in a region-specific manner and that these changes underlie sex differences in adult behavior. This talk reviews the cellular and molecular mechanisms mediating masculinization involving inflammatory molecules, immune signaling, endocannabinoids, and epigenetic changes. Illuminating the biological origins of brain and behavior sex differences is essential for enhancing health and preventing disease.

**Theme F: Cognition and Behavior**

**Generating and Shaping Novel Action Repertoires CME**

**Rui M. Costa, DVM, PhD**

Champalimaud Foundation, Portugal

Tuesday, Nov. 18, 1–2:10 p.m.

Many actions are learned anew throughout life, likely through a process of trial and selection. Researchers investigated how novel self-paced actions are generated and how actions that lead to particular outcomes are then selected. Research found that dopamine is critical for the initiation of novel actions and that plasticity in cortico-basal ganglia circuits is essential for action selection. With iteration, actions become organized in modules, and neural substrates of chunking emerge in these circuits.

**Affective Neurosciences of Reward: Limbic Modules for Liking and Wanting CME**

**Kent C. Berridge, PhD**

University of Michigan, Ann Arbor

Wednesday, Nov. 19, 1–2:10 p.m.

Reward involves several different psychological components. “Wanting” a reward is generated by robust mesolimbic circuitry, whereas “liking” the same reward is generated by hedonic-hotspot circuitry that is neuroanatomically and neurochemically more restricted. This wanting-liking difference has implications for addiction disorders. Yet surprisingly, forms of positive wanting and negative fear share some of the same brain mechanisms. New insight on the generation of these intense “liking,” “wanting,” and other emotion states are emerging in affective neuroscience.

**Theme G: Novel Methods and Technology Development**

**Nanoscopy With Focused Light: Principles and Applications CME**

**Stefan W. Hell, PhD**

Max Planck Institute for Biophysical Chemistry, Germany

Saturday, Nov. 15, 2–3:10 p.m.

For most of the 20th century, scientists believed that lens-based light microscopy could not discern details finer than half the wavelength of light (>200 nm). In the 1990s, this barrier was overcome when it was discovered that fluorescent features could not be resolved virtually down to molecular dimensions. This lecture discusses the simple, yet powerful, physical principles that allowed researchers to overcome the diffraction limit, with special emphasis on STED and RESOLFT microscopy. The lecturer will exemplify the relevance of these nanoscopy techniques to neuroscience.