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Minisymposium

M01. Hair Cells to Brain Cells — The Development and Evolution of the Primary Auditory System

Location: Virtual Only

Time: Monday, November 8, 2021, 10:45 AM - 11:45 AM

Description: Birds and mammals have auditory systems with similar functional architecture which must have arisen independently, since their last common ancestor did not exhibit a middle ear. The apparatus of the inner ear and the first-order hindbrain circuitry pose striking questions about evolution, development and functional convergence that have been illuminated by new genetic insights into the mechanisms of auditory circuit development and the analysis of the physiology of sound localisation.

Support: Royal Society Newton Fellowship (Wingate-Lipovsek)
NIDCD000436 (Carr)
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ISF 1515/16 (Sela-Donnenfeld)
BSF (US-IL) 2015087 (Wang, Sela-Donnenfeld)
DFG – EXC 2177/1 - 390895286 (Northwang). Chair

Abstract: Birds, reptiles and mammals have first order auditory systems with a similar functional architecture. One shared feature of their organisation is the ability to locate the origin of sounds in space by comparing signals arriving in either ear. However, because their last common ancestor did not exhibit a middle ear, the cells and neural circuits underlying how sound is transduced and processed must have evolved independently. Are the structures and circuits of sensory transduction and processing in the hindbrain homologous or analogous? If the former, then the evolution of the auditory processing would seem to be an inevitable consequence of conserved developmental constraints. If the latter, then the first order auditory system in birds and mammals would present a striking example of adaptive convergence. Thus, the apparatus of the inner ear and the first order hindbrain circuitry space therefore pose a striking question about development and evolution that has now been addressed through new genetic insights into the mechanisms of auditory circuit development and the analysis of the physiology of sound localisation. We will present findings from several model systems that exploited innovative strategies in developmental biology, genome editing, genetics, bioinformatics and electrophysiology. These will range from the molecular patterning of auditory hair cells and neurons, the developmental lineages of the first order neural circuit, the roles of transcription factors, microRNAs and RNA binding proteins, the guidance of axonal connections and evolutionary convergence at a functional level. Overall, the session will deliver an overview of one of the main questions in vertebrate auditory system evolution and cover recent advances that shine light on the development and function of this core model of sensory evolution.

M01.01. Chair

R. J. Wingate;

King's College London, London, UNITED KINGDOM.

Disclosures:

M01.02. Co Chair

D. Sela-Donenfeld;

The Hebrew University, Rehovot, ISRAEL.

Disclosures:

M01.03. Regulation of axonal growth in Atoh1⁺ hindbrain auditory neurons

D. Sela-Donenfeld;

Koret School of Veterinary Medicine, The Hebrew University, Rehovot, ISRAEL.

Disclosures: D. Sela-Donenfeld: None.

M01.04. Neurons and hair cells require Lmx1a/b for normal brainstem development

B. Fritsch;

Biology, University of Iowa, IOWA CITY, IA.

Disclosures: B. Fritsch: None.

M01.05. A microRNA guide to auditory development, dysfunction, and evolution

G. Northwang;

Department für Neurowissenschaften, Carl von Ossietzky University of Oldenburg, Oldenburg, GERMANY.

Disclosures:

M01.06. FMRP mechanism of auditory circuit development in birds and mammals

Y. Wang;

Department of Biomedical Sciences, Florida State University, Tallahassee, FL.

Disclosures: Y. Wang: None.

M01.07. Functional convergence of temporal processing

C. E. Carr;

University of Maryland, College Park, MD.

Disclosures: C.E. Carr: None.

M01.08. A comparative view on the development of hindbrain auditory and vestibular nuclei

R. J. Wingate;
King's Col London, London, UNITED KINGDOM.

Disclosures: R.J. Wingate: None.

Minisymposium

M02. Molecular Mechanisms of Cortical Development in Health and Disease

Location: Virtual Only

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

Description: Cortical development is a tightly regulated process that involves progenitor proliferation, neuronal differentiation and migration, programmed cell death, and circuit formation. Emerging studies provide evidence that coordinated molecular and cellular mechanisms govern cortical development and that perturbations of these processes lead to neurodevelopmental disorders. This **minisymposium** will highlight the latest findings of the rules and logic for cortical development from a diverse group of researchers.

Support: Academia Sinica (AS-CDA-107-L09) and Ministry of Science and Technology, Taiwan (108-2311-B-001-021) (Chou, SJ)
NIH R01 NS097525 and U01 MH106892 (Kwan, KY)
Institutional start-Up funds and R15 1R15CA235749-01 (Ge, XC)
NIH R01MH094589 and R01NS089777 (Chen, B)
National Science Council, Taiwan (NSC 100-2320-B-002-089-MY3) and College of Medicine, National Taiwan University (102C101-31 and 103C101-21) (Cheng HJ)
Severo Ochoa fellowship from the Spanish MINECO, BES-2015-071690,
Fellowship from MINECO, BES-2014-067594 (Nieto, M)
MINECO BFU2016-81887-REDT, MINECO PCIN-2015-176-C02-02/ERA-Net Neuron (Era-Net, MINECO), MCIU/AEI/FEDER, UE SAF2017-83117-R, Flag-Era JTC 2019. Transnational call. PCI2019-11872-2. (Nieto, M)

Abstract: The mammalian cerebral cortex is a remarkably complex organ responsible for the perception of sensory stimuli, the execution of motor actions, and the integration of all higher cortical functions involving learning, memory, cognition, and consciousness. It contains many neuronal cell types, and these neurons form connections within the cortex and with other regions of the central nervous system. Elucidating the mechanisms controlling cortical development is not only crucial to our understanding of brain function, but also important for the understanding of neuropsychiatric disorders. This timely mini-symposium will focus on early cortical development to discuss various regulatory mechanisms. The topics to be discussed include: (1) the significance of the expression of transcription factors in concentration gradients in patterning cortical regions (Chou, SJ); (2) The molecularly distinct roles of chromatin regulation in transcription and DNA damage repair during corticogenesis (Kwan, KY); (3) The exploration of the primary cilium as a signaling hub to integrate cohorts of signaling pathways to regulate brain

development (Ge, XC); (4) The signaling pathways that regulate the lineage progression of cortical radial glial cells (Chen, B); (5) The control of programmed cell death in postmitotic immature neurons during embryonic cortical development (Cheng, HJ); and (6) The development of transient callosal axons by most cortical neurons during their normal differentiation, and its implications for the concept of early neuronal fates, molecular and cellular plasticity, and alternative circuits associated to neurodevelopmental disorders (Nieto, M). These presentations will provide opportunities to stimulate interdisciplinary discussions about the control of early stages of cortical development. They will also shed new light on how neuropsychiatric disorders can be caused by abnormalities in early cortical development.

M02.01. Chair

S. Chou;

Academia Sinica, Taipei, TAIWAN.

Disclosures:

M02.02. Co Chair

X. Ge;

University of California, Merced, Merced, CA.

Disclosures:

M02.03. The role of *Blm*-s, a Bcl-2 family gene, in embryonic cortical development

H. Cheng;

UC Davis, Davis, CA.

Disclosures:

M02.04. Integration of cell signaling in the primary cilium of the developing cortex

X. Ge;

University of California, Merced, Merced, CA.

Disclosures:

M02.05. The determination of projection neuron identity in the developing cerebral cortex.

B. Chen;

University of California Santa Cruz, Santa Cruz, CA.

Disclosures:

M02.06. Maintenance of genome stability in cortical development

K. Y. Kwan;

Molecular and Behavioral Neuroscience Inst., University of Michigan, Ann Arbor, MI.

Disclosures:

M02.07. Early callosal fate of most cortical neurons

M. Nieto;

Centro Nacional de Biotecnología, CSIC, Madrid, SPAIN.

Disclosures: M. Nieto: None.

M02.08. Patterning of the cerebral cortex

S. Chou;

Academia Sinica, Taipei, TAIWAN.

Disclosures: S. Chou: None.

Minisymposium**M03. New Insights in Understanding Autism and Intellectual Disabilities — Focus on Mechanisms and Translational Approaches**

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Autism is frequently accompanied by intellectual disability (ID). Though different in etiology, they both overlap clinically and neuropathologically and are believed to share common neurobiological mechanisms, exemplified by shared mutations in genes for synaptic function, excitation-inhibition imbalance, and neuroinflammation. This minisymposium will present advances in the neurobiology of autism and ID and explore potential common therapeutic interventions.

Support: Simmons Foundation (SFARI 400101)
European Research Council (ERC, DISCONN Grant Agreement 802371))
Brain and Behavior Foundation (2017 NARSAD, Grant 25861
NIH (1R21MH116473-01A1)
Telethon Foundation (GGP19177)
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Wellcome Trust (UK)

Abstract: Autism and intellectual disability (ID) are the most common neurodevelopmental disorders in humans. Affecting 3-5% of the population, they present a major global health concern. Though different in etiology, they both overlap clinically and neuropathologically and are believed to share common neurobiological mechanisms, exemplified by shared mutations in genes for synaptic function, excitation-inhibition imbalance, and (neuro)inflammation. Despite the wealth of research, translation of discoveries into therapies for autism and ID remains a challenge. One of the major roadblocks is a lack of integration of knowledge arising from different disciplines. This minisymposium will summarize recent advances from a multi-

disciplinary perspective and explore how these may synergize into a more coherent picture of autism and ID, facilitating improved strategies for therapeutic target identification and drug development.

M03.01: Chair

M. M. Petrinovic;

King's College London, London, UNITED KINGDOM.

M03.02: Co Chair

D. Cash;

King's College London, London, UNITED KINGDOM.

M03.03: Neurobiology of autism spectrum disorder, learning through genetically engineered mouse and monkey models

Y. Zhou;

McGill University, Montreal, QC, CANADA.

Disclosures: Y. Zhou: None.

M03.04. Parsing autism heterogeneity with cross-species fMRI

A. Gozzi;

Functional Neuroimaging Laboratory, Istituto Italiano di Tecnologia, Rovereto, ITALY.

Disclosures: A. Gozzi: None.

M03.05. Translating genetics of neurodevelopmental disorders into disease mechanisms targeting postsynaptic pathways

F. Laumonnier;

Faculty of Medicine, INSERM and University of Tours, Tours, FRANCE.

Disclosures: F. Laumonnier: None.

M03.06. How do overlapping E/I, synaptic and neuroinflammatory mechanisms for autism and intellectual disabilities relate to clinical presentation?

A. Strydom;

King's College London, London, UNITED KINGDOM.

Disclosures:

M03.07. Genetics-first approaches into the neurobiology of neurodevelopmental disorders

S. De Rubeis;

Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York, NY.

Disclosures:

M03.08. Therapeutic potential of GABAA alpha5 subunit-containing receptors in neurodevelopmental disorders

M. Hernandez;

F. Hoffmann-La Roche Ltd, Basel, SWITZERLAND.

Disclosures: M. Hernandez: A. Employment/Salary (full or part-time);; Employee at F. Hoffmann-La Roche Ltd.

Minisymposium**M04. RASopathies — Pathophysiology and Therapeutic Directions**

Location: Virtual Only

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

Description: RASopathies are a family of genetic syndromes that frequently exhibit developmental delay, intellectual disability, epilepsy, and an increased risk of autism and ADHD. Many RASopathy mutations target regulators of Receptor Tyrosine Kinase-RAS-MAPK signaling. This minisymposium will focus on recent advances in our understanding of the impairments in human brain development and distinct cellular subtypes in model systems that contribute to neurological abnormalities in RASopathies.

Support: R00NS076661
R01NS097537
NRF-2016R1E1A1A01941939
NRF-2017M3C7A1026959
R01NS082283
2015-04-009A
W81-WH-10-10116

Abstract: The ubiquitous RAS/MAPK pathway plays a critical role in the development and function of neuronal and glial subtypes. The RASopathies are a family of genetic syndromes that exhibit mutations in regulators of Receptor Tyrosine Kinase-RAS-MAPK signaling. Neurofibromatosis type 1 and Noonan syndrome are common RASopathies that occur in roughly 1:2000 births. Developmental delay, intellectual disability, epilepsy, and an increased risk of autism, ADHD, and cancer are often observed in these syndromes. Here, we will present recent work focused on using diverse model systems and brain imaging to identify candidate neuropathological mechanisms that may contribute to these deficits. In addition to the RASopathies and specific forms of autism, abnormal RAS/MAPK activity has also been observed in epilepsy, TBI, and perinatal hypoxia. A better understanding of RASopathy effects on the developing nervous system will be relevant to multiple pathological conditions and inform the design of personalized therapeutic approaches for related abnormalities.

M04.01. Chair

J. M. Newbern;

School of Life Sciences, Arizona State University, Tempe, AZ.

Disclosures:

M04.02. Co Chair

J. M. Weimer;

Children's Health Research Center, Sanford Research, Sioux Falls, SD.

Disclosures:

M04.03. Cell type-specific RAS signaling activations in learning disabilities

Y. Lee;

Seoul National University College of Medicine, Seoul, KOREA, REPUBLIC OF.

Disclosures: Y. Lee: None.

M04.04. Bridging the gaps in our knowledge: brain development of children with noonan syndrome

T. Green;

Stanford University, Palo Alto, CA.

Disclosures: T. Green: None.

M04.05. "Differential functions of MEK-ERK1/2 signaling in cortical excitatory and inhibitory neuron development"

J. Newbern;

School of Life Sciences, Arizona State University, Tempe, AZ.

Disclosures:

M04.06. "Disruption of a core GABAergic inhibitory neuron gene, Lhx6, in a Nf1 RASopathy mouse model"

D. Vogt;

Department of Pediatrics and Human Development, Michigan State University, Grand Rapids, MI.

Disclosures: D. Vogt: None.

M04.07. "Using large animal models of Neurofibromatosis Type 1 to better understand the neurological etiology of RASopathies"

J. M. Weimer;

Children's Health Research Center, Sanford Research, Sioux Falls, SD.

Disclosures:

M04.08. “White matter decompaction correlates with systems-level deficits in NF1 RASopathy”

N. Ratner;

Dept Pediatrics, Div. Exp. Hematol., Cincinnati Children's Hosp, Cincinnati, OH.

Disclosures:

Minisymposium

M05. The Logic of Developing Neocortical Circuits in Health and Disease

Location: Virtual Only

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

Description: A vast number of diverse neurons builds an intricate and complex circuitry that account for the sensory and cognitive abilities of the neocortex. The principles and underlying mechanisms governing the assembly of neocortical circuits in early life remain elusive. This minisymposium will uncover recent evidence demonstrating the dynamic role of neuronal populations in the development of functional neocortical ensembles and highlight the cellular hubs of disease-related miswiring.

Support: Hanganu-Opatz is supported by ERC-2015-CoG 681577; German Research Foundation (Ha 4466/10-1, Ha4466/11-1, Ha4466/12-1, SPP 1665, SFB 936 B5) Cardin is supported by Simons Foundation SFARI Pilot Award; Simons Foundation SFARI Research Award; SMS Foundation Research Award; Ludwig Family Foundation Award; NIMH R01 MH113852; NIHCD U01 NS094358; NIMH R01 MH102365
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De Marco is supported by NIH (NIMH 1R01MH110553-01); Pediatric Epilepsies Award (Citizens United for Research in Epilepsy); Leon Levy Fellowship Award (Leon Levy Foundation); Frueauff Foundation Award

M05.01. Chair

I. L. Hanganu-Opatz;

University Medical Center Hamburg-Eppendorf, Hamburg, GERMANY.

Disclosures:

M05.02. Co Chair

S. Butt;

Dept. of Physiology, Anatomy & Genetics, University of Oxford, Oxford, UNITED KINGDOM.

Disclosures:

M05.03. Mechanisms generating cell-type diversity in developing cerebral cortex

S. Hippenmeyer;

Institute of Science and Technology Austria, Klosterneuburg, AUSTRIA.

Disclosures: S. Hippenmeyer: None.

M05.04. Network activity in the development of inhibitory circuit

N. De Marco Garcia;

Brain and Mind Research Institute, Weill Cornell Medical College, New York, NY.

Disclosures: N. De Marco Garcia: None.

M05.05. Developmental perturbation of PV interneurons disrupts state-dependent circuit dynamics

J. A. Cardin;

Department of Neurobiology, Yale University, New Haven, CT.

Disclosures: J.A. Cardin: None.

M05.06. Transient circuits of neocortex provide a scaffold for emergent perception in a modality-specific manner

S. J. Butt;

Dept. of Physiology, Anatomy & Genetics, University of Oxford, Oxford, UNITED KINGDOM.

Disclosures: S.J. Butt: None.

M05.07. Human iPSC-derived cortical organoids as a model for neocortical oscillatory circuit development

B. Voytek;

Cognitive Science, University of California, San Diego, La Jolla, CA.

Disclosures: B. Voytek: None.

M05.08. Activity-dependent maturation of prefrontal gamma oscillations sculpts cognitive performance

I. L. Hanganu-Opatz;

University Medical Center Hamburg-Eppendorf, Hamburg, GERMANY.

Disclosures: I.L. Hanganu-Opatz: None.

Minisymposium

M06. Glia — The Glue Holding Memories Together

Location: Virtual Only

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

Description: The contribution of astrocytes and oligodendrocytes to neuronal plasticity and behavior has expanded vastly in recent years. Specifically, research of the role of glia in memory acquisition and consolidation is flourishing thanks to the assimilation of new techniques into the field. To integrate knowledge from ongoing work, this minisymposium will bring together experts that study different angles of this topic: the effect of oligodendrocytes and astrocytes; recent and remote memory; and experimental and theoretical aspects.

Support: US NIH Director's Pioneer Award (DP1NS111132)
US National Institute for Neurological Disorders and Stroke ((R01NS092597)
Canadian Institutes of Health Research (CIHR) FDN143227 and PJT15616
Swiss National Science Foundation (SNSF) 31003A-173124/1
National Center of Competence in Research (NCCR) 51NF40-160620
Synopsis Foundation Grant 2018-PI01
RTI2018-094887-B-I00 and RYC-2016-20414.

Abstract: Experience sculpts brain structure and function. Glial cells - oligodendrocytes and astrocytes - modulate neural circuit establishment, refinement, temporal dynamics, plasticity and ultimately function. Astrocytes are central to synaptogenesis and synaptic function, actively participate in information processing through the release of their own signaling molecules (termed gliotransmitters) and promote circuit homeostasis through neurotransmitter uptake and K⁺ buffering. Oligodendroglial lineage cells form new myelin throughout life, including both intrinsic, activity-independent myelination and adaptive, activity-regulated myelination. Activity-dependent modulation of the myelinated infrastructure of the nervous system has emerged as a dimension of adaptive change during development and in adulthood. Myelination is a richly dynamic process, with oligodendrocyte precursor cell proliferation, oligodendrogenesis and myelin structural changes regulated by neuronal activity. Mechanisms mediating neuron-oligodendroglial interactions include activity-regulated neurotrophin signaling and neuron-oligodendroglial synaptic communication. Plasticity of myelin and consequent changes to conduction velocity and circuit dynamics may powerfully influence memory encoding and consolidation. Just as myelin plasticity contributes to cognitive function; disruption of the

mechanisms mediating adaptive myelination with loss of myelin plasticity underlies cognitive impairment following chemotherapy exposure.

M06.01. Chair

M. Monje;

Stanford University, Stanford, CA.

Disclosures:

M06.02. Astrocytes: the missing link in synaptic plasticity

M. Navarrete;

Instituto Cajal, CSIC, Madrid, SPAIN.

Disclosures: M. Navarrete: None.

M06.03. Myelin plasticity in health and disease

M. Monje;

Stanford University, Stanford, CA.

Disclosures:

M06.04. New insight into how astrocytes integrate hippocampal memory circuits

A. Volterra;

University of Lausanne, Lausanne, SWITZERLAND.

Disclosures: A. Volterra: None.

M06.05. Memory from the perspective of systems astroscience

E. Galea;

Universitat Autònoma de Barcelona, Barcelona, SPAIN.

Disclosures: E. Galea: None.

M06.06. Circuit-specific effects of astrocytes on plasticity and memory.

A. Kol;

Neurobiology, The Hebrew University, Jerusalem, ISRAEL.

Disclosures: A. Kol: None.

M06.07. Disruption of oligodendrogenesis impairs memory consolidation in adult mice

P. Steadman;

The Hospital for Sick Children, Toronto, ON, CANADA.

Disclosures: P. Steadman: None.

Minisymposium

M07. Molecular Mechanisms of Glial Engulfment

Location: Virtual Only

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

Description: Engulfment of neuronal cell bodies and fragments is critical for neural health and proper circuit connectivity. These tasks are carried out by distinct glial subtypes in the central and peripheral nervous systems, but the mechanisms that govern engulfment activity are still unclear. This minisymposium will focus on recent molecular and cellular discoveries for glial phagocytic function across species and offer comparative insights and discussion regarding the development and disease-relevant function of glia.

Support: Simons Foundation for Aging Research, American Federation for Aging Research
Pew Charitable Trusts, Burroughs Wellcome Fund
NIH
Grants in Aid from SigmaXi
University of Virginia Brain Institute
Canadian Institute for Health Research; Natural Sciences Engineering research of Canada; Department of Defense, USA; ALS Canada; Packard center for research on ALS

Abstract: Development and maintenance of neural circuits requires elimination of exuberant or damaged axons, dendrites, as well as synapses. Glial cells support this process by clearing these neuronal projections through phagocytic mechanisms. For example, glia engulf synaptic material to refine circuits during mammalian development and throughout life. Sensory glia-like cells, such as retinal pigment epithelium (RPE) in the retina, engulf specialized outer segments of sensory photoreceptor neurons. Aberrant engulfment by mammalian glia is implicated in neurological disease. Notably, glial clearance of degeneration process is also observed in *Drosophila* during metamorphosis and post-injury, suggesting broad conservation of this process. However, many mechanistic properties of glial engulfment remain poorly understood. This mini-symposium explores molecular and cell-biological insights across peripheral and central nervous systems, various species (vertebrates and invertebrates), and distinct developmental windows (developmental pruning versus post-development) with regard to glial clearance of neuronal material. We discuss how astrocytes and microglia clear neuronal fragments in the CNS, how RPE and sensory organ glia clear neuronal fragments throughout animal life, and how glial clearance is executed in the developing spinal cord and neuromuscular junction. Understanding molecular mechanisms across these diverse contexts will offer novel insight into conserved principles and functional nuances of evolutionarily conserved and disease-relevant glial engulfment events.

M07.01. Chair

A. Singhvi;

Basic Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA.

Disclosures:

M07.02. Co Chair

M. A. Logan;

Neurology/Jungers Center, Oregon Health and Science University, Portland, OR.

Disclosures: M.A. Logan: None.

M07.03. Glial contributions to motoneuron function and plasticity

R. Robitaille;

Univ of Montreal, Montreal, QC, CANADA.

Disclosures: R. Robitaille: None.

M07.04. Neuronal activity regulates synaptic phosphatidylserine exposure

N. Hewitt-Scott;

Children's Hospital, Harvard, Boston, MA.

Disclosures: N. Hewitt-Scott: None.

M07.05. Glial engulfment in *C. elegans*

S. A. Raiders;

Fred Hutchinson Cancer Research Center, Seattle, WA.

Disclosures: S.A. Raiders: None.

M07.06. Glial-mediated debris clearance during development and disease

S. Kucenas;

University of Virginia, Charlottesville, VA.

Disclosures: S. Kucenas: None.

M07.07. Müller glia: mechanism of clearance phagocytosis and role in retinal degeneration

S. Finnemann;

Department of Biological Sciences, Fordham University, New York City, NY.

Disclosures: S. Finnemann: None.

Minisymposium

M08. Spatio-Temporal Regulation of RNA in Neurons

Location: Virtual Only

Time: Thursday, November 11, 2021, 12:00 PM - 1:00 PM

Description: RNA is regulated from transcription to degradation to provide spatial and temporal control over gene expression. This is especially important for morphologically and functionally complex neurons. Accordingly, RNA dysregulation is a causal factor in several neurological diseases, from autism to dementia. This minisymposium will address the unconventional, activity-driven, and cell-specific roles for RNA that have expanded our understanding of RNA regulation in driving neuron function and behavior.

Support: R00MH109626

M08.01. Chair

S. Farris;

Fralin Biomedical Research Institute, Virginia Tech, Roanoke, VA.

Disclosures:

M08.02. Synapse to nucleus signaling via dendritic translation of a transcription factor

B. L. Bloodgood;

Division of Biological Science, Neurobiology, UC San Diego, La Jolla, CA.

Disclosures: B.L. Bloodgood: None.

M08.03. Dysregulation of UPF1 and nonsense-mediated decay in ALS/FTD

J. U. Guo;

Department of Neuroscience, Yale University School of Medicine, New Haven, CT.

Disclosures: J.U. Guo: None.

M08.04. Modulation of neuronal activity-induced transcription by recent past activity and putative function thereof

R. N. Saha;

University of California Merced, Merced, CA.

Disclosures:

M08.05. Temporal regulation of *Arc* transcription dynamics: immediate early and beyond

S. Das;

Albert Einstein College of Medicine, Bronx, NY.

Disclosures:

M08.06. Neural-specific coordination of alternative splicing and alternative polyadenylation

P. Miura;

Department of Biology, University of Nevada, Reno, Reno, NV.

Disclosures:

M08.07. RNA metabolism in developing neurons

A. Riccio;

Neuroscience, University of College London, London, UNITED KINGDOM.

Disclosures: A. Riccio: None.

Minisymposium

M09. The Rich Dynamism of GABAergic Cell Types — Specificity and Diversity of Inhibitory Plasticity in Microcircuit Function

Location: Virtual Only

Time: Tuesday, November 9, 2021, 12:00 PM - 1:00 PM

Description: A dynamic landscape of excitation and inhibition in the cortex sustains complex cognitive functions. Given the rich diversity of GABAergic circuits, it is unclear how inhibition changes in adaptive behavior. This minisymposium will highlight recent advances in identifying and manipulating specific GABA cell populations to understand how varied forms of GABA plasticity enable multiplexing of inhibition to match synaptic excitation and drive cortical plasticity in development and learning.

Support: NIDCD DC012557
Max Planck Society
European Research Council
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Human Frontier Science Program
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Wellcome Trust ref 101029. Chair

M09.01. Chair

S. C. Song;

New York University, New York, NY.

Disclosures:

M09.02. Co Chair

C. Q. Chiu;

Universidad de Valparaíso, Centro Interdisciplinario De Neurociencia De Valparaíso, Valparaíso, CHILE.

Disclosures:

M09.03. Learning-related plasticity in cortical layer 1

M. B. Pardi;

Max Plank Institute for Brain Research, Frankfurt, GERMANY.

Disclosures: M.B. Pardi: None.

M09.04. Input-specific inhibitory STDP of layer 2/3 auditory cortex

S. C. Song;

New York University, New York, NY.

Disclosures: S.C. Song: None.

M09.05. Parvalbumin neurons are sensitive to brief visual deprivation throughout life

S. J. Kuhlman;

Carnegie Mellon University, Pittsburgh, PA.

Disclosures:

M09.06. Top-down learning through inhibitory circuits

C. Clopath;

Imperial College London, London, UNITED KINGDOM.

Disclosures:

M09.07. Rules and mechanisms of plasticity at parvalbumin and somatostatin inhibitory synapses in the hippocampus

J. Mellor;

University of Bristol, Bristol, UNITED KINGDOM.

Disclosures: J. Mellor: None.

M09.08. Diverse mechanisms of GABAergic regulation of signaling in neuronal dendrites

C. Q. Chiu;

Centro Interdisciplinario De Neurociencia De Valparaíso, Universidad de Valparaíso, Valparaíso, CHILE.

Disclosures: C.Q. Chiu: None.

Minisymposium

M10. Axon Damage Signaling and Its Relevance to Disease

Location: Virtual Only

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

Description: This minisymposium will present current research on mechanisms of axonal damage responses in the contexts of neuronal injury, repair, and inflammation, and will probe the role of these pathways in neurodegenerative diseases, neuropathies and developmental plasticity. Presentations will focus on the current understanding of neuronal stress signaling based on studies that span multiple model organisms and combined *in vivo* and molecular work. Speakers will discuss shared molecular pathways that may exist between axonal damage signaling, developmental degeneration, and neurodegenerative diseases.

Abstract: Axons perform a fundamental role in neurotransmission and have been referred to as the neuron's Achilles' heel. Since they extend over long distances in the brain and body, they are vulnerable to physical damage, cytoskeletal defects, and impairments in axonal transport. Additionally, repair over long distances has great challenges in the adult nervous system. It is therefore logical that neurons are equipped with mechanisms for sensing and responding to axonal damage. What are these mechanisms and how do they work? Recent work has brought attention to a conserved axonal dual leucine zipper kinase DLK as a key regulator of both degenerative and regenerative responses to axonal damage and stress. Current studies of DLK's mechanism and function is one theme of this mini-symposium. We also probe the intersection of axonal damage signaling with mechanisms of neuronal development, neuroinflammation, and neurodegenerative disease.

M10.01. Chair

C. E. Le Pichon;

NICHHD, National Institute of Health, Bethesda, MD.

Disclosures:

M10.02. Co Chair

C. A. Collins;

Molecular Cellular and Developmental Biology, University of Michigan, Ann Arbor, MI.

Disclosures:

M10.03. Axon injury responses in neurodegenerative disease

C. E. Le Pichon;

NICHHD, National Institutes of Health, Bethesda, MD.

Disclosures:

M10.04. Shared mechanisms of axon regeneration and degeneration

A. B. Byrne;

Department of Neurobiology, UMass Medical School, Worcester, MA.

Disclosures: A.B. Byrne: None.

M10.05. Harnessing the axonal stress response for CNS axon regeneration and neuroprotection

T. A. Watkins;

Baylor College of Medicine, Houston, TX.

Disclosures: T.A. Watkins: None.

M10.06. Anterograde signaling in the control of axon degeneration

D. J. Simon;

Dept of Biochemistry, Weill Cornell Medical College, New York, NY.

Disclosures:

M10.07. Conserved mechanisms and new functions for axonal damage signaling

C. A. Collins;

Molecular Cellular and Developmental Biology, University of Michigan, Ann Arbor, MI.

Disclosures: C.A. Collins: None.

M10.08. Neuroimmune response to nerve injury

W. Zeng;

Institute for Immunology and School of Medicine, Tsinghua University, 北京, CHINA.

Disclosures: W. Zeng: None.

Minisymposium

M11. Mitochondrial Neurodegenerative Diseases — From Pathogenic Mechanisms to Therapeutic Opportunities

Location: Virtual Only

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

Description: Mitochondria are energy powerhouses and central regulators of cell fate, redox balance and ion homeostasis. They act as central hubs for pathways involved in neural development, synaptic activity and connectivity. Mitochondrial dysfunction underlies neurological diseases ranging from childhood inherited mitochondrial disorders to adult neurodegenerative disorders. This minisymposium will provide insight into the pathogenic mechanisms of mitochondria as a springboard for designing targeted therapeutic tools.

Support: Canada Research Chair Funds (internal) to Mireille Kachro
NIAMS R01 AR073217; NIH DP2 ES030449 to Prashant Mishra
Start-up Funds from Virginia Polytechnic Institute and State University to Alicia Pickrell

Glenn Foundation Postdoctoral Fellowship to Li Li; DoD (W81XWH-16-1-0282, X.W.); NIA (R21AG06131501, X.W.)
Astra Zeneca (NCR-19-14517) to Ajit Divakaruni
NIH DP5 OD026398 to Isha Jain

Abstract: This minisymposium on mitochondrial neurodegenerative diseases will highlight recent advances in this fast-growing field. A rising body of evidence has revealed mitochondrial dysfunction as a pivotal element for a myriad of diseases, including inherited neurodevelopmental disorders, stroke, autism, neurodegenerative diseases and brain aging. Mitochondria contribute to the pathophysiological states beyond dysfunctional ATP synthesis via dysregulation of cell fate, cellular homeostasis, mitophagy, cerebral nutrient preference, oxidative stress response and hypoxia. This minisymposium will emphasize six intertwined functional themes: 1) mitochondrial plasticity as a key regulator of neural cell fate; 2) phenotypic associations between accumulation of mutations in the mitochondrial genome and the adult stem cell population; 3) the functional link between mitochondrial quality control and cell homeostasis: implications for Parkinson's disease ; 4) Novel biomarkers and therapeutic targets to personalize patient's prognosis and to curtail Parkinson's disease and age-related diseases; 5) therapeutic potentials of switching cerebral nutrient preference to improve mitochondrial functions and quality control in the context of cognitive impairment and Alzheimer's disease; and 6) turning the oxygen rheostat: a novel therapeutic approach for rare inborn errors of energy metabolism and age-associated pathologies. This minisymposium will engage scientists and physicians to convert an ongoing challenge into an opportunity by understanding the pathogenic mechanisms to gain crucial insight into personalizing prognosis and therapeutic interventions.

M11.01. Chair

A. E. Chiaramello;

Dept Anat and Cell Biology, George Washington University Medical Center, Washington, DC.

Disclosures:

M11.02. Co Chair

J. Scafidi;

Dept of Neurology and Center for Neuroscience Research, Children's National Medical Center, Washington, DC.

Disclosures: J. Scafidi: None.

M11.03. Targeting substrate transport proteins to treat neurodegenerative diseases

A. Divakaruni;

Molecular and Medical Pharmacology, University of California, Los Angeles, Los Angeles, CA.

Disclosures:

M11.04. Mitochondrial homeostasis in parkinson's disease

L. Li;

Neurosurgery, Stanford University School of Medicine, Palo Alto, CA.

Disclosures: L. Li: None.

M11.05. Turning the oxygen dial as a therapy

I. Jain;

Physiology, Gladstone Institutes, San Francisco, CA.

Disclosures: I. Jain: None.

M11.06. Investigating consequences of mitochondrial dysfunction in stem cells

P. Mishra;

Pediatrics, Children's Medical Center Research Institute, University of Texas Southwestern Medical Center, Dallas, TX.

Disclosures: P. Mishra: None.

M11.07. Mitochondrial plasticity directs the fate of neural stem cells

M. Khacho;

Biochemistry, Microbiology and Immunology, University of Ottawa, Ottawa, ON, CANADA.

Disclosures: M. Khacho: None.

M11.08. Mitochondrial quality control dictates cell death and division

A. M. Pickrell;

Neuroscience, Virginia Tech, Blacksburg, VA.

Disclosures: A.M. Pickrell: None.

Minisymposium

M12. Pathogenic Mechanisms and Therapeutic Approaches for Frontotemporal Dementia

Location: Virtual Only

Time: Monday, November 8, 2021, 12:00 PM - 1:00 PM

Description: Autosomal dominant mutations in three genes — MAPT, GRN, and C9ORF72 — are responsible for the majority of cases of genetic frontotemporal degeneration (FTD), the most common form of dementia in people under the age of 60. This minisymposium will highlight exciting unpublished advances in our understanding of the pathogenesis of FTD caused by these mutations, novel models and methodologies that have facilitated these advances, and their implications for the development of targeted therapies.

M12.01. Chair

F. Gao;

368 Plantation Street, University of Massachusetts Medical School, Worcester, MA.

Abstract: x

Disclosures:

M12.02. Regulation of dipeptide repeat protein production in *C9ORF72* iPSC-derived neurons

S. Almeida;

University of Massachusetts Medical School, Worcester, MA.

Disclosures: S. Almeida: None.

M12.03. The neurotrophic effects of lysosomal progranulin

A. Arrant;

Neurology, The University of Alabama at Birmingham, Birmingham, AL.

Disclosures: A. Arrant: None.

M12.04. Chemical suppression of progranulin nonsense mutations as a novel therapeutic strategy in frontotemporal lobar degeneration

H. B. Nygaard;

University of British Columbia, Vancouver, BC, CANADA.

Disclosures: H.B. Nygaard: F. Consulting Fees (e.g., advisory boards); Biogen Advisory Board, Roche scientific advisor.

M12.05. Transposable element activation in Alzheimer's disease and related tauopathies

B. Frost;

University of Texas Health San Antonio, San Antonio, TX.

Disclosures: B. Frost: 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.; Transposon Therapeutics, Contracted Research, MD Anderson, Contracted Research. F. Consulting Fees (e.g., advisory boards); MD Anderson, Consulting Fees.

M12.06. Understanding repeat RNA metabolism in *C9ORF72*-FTD/ALS

S. Sun;

Johns Hopkins University, Baltimore, MD.

Disclosures:

M12.07. Patient-based structural biology of tauopathies

A. Fitzpatrick;
Columbia University, New York, NY.

Disclosures:

Minisymposium

M13. Shining Light on Neuronal Plasticity After Stroke

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Recent applications of optics in neuroscience provided tremendous opportunity for investigating and manipulating brain rewiring *in vivo* before and after stroke. Speakers will present optical methods for developing rehabilitative strategies and imaging the effects of these maneuvers at the cellular and systems level. This minisymposium will feature basic stroke research with translational potential and showcase recent advancements in optical tools for *in vivo* investigation of neuronal plasticity.

Support: Branco Weiss Fellowship
Wrangell Habilitation Scholarship
NIH - R01NS102870
NIH - K25NS083754
McDonnell Center for Systems Neuroscience
H2020 EXCELLENT SCIENCE - European Research Council (ERC) under grant agreement ID n. 692943 BrainBIT
European Union's Horizon 2020 Research and Innovation Programme under Grant Agreement No. 720270 (HBP SGA1), No. 785907 (HBP SGA2) and 654148 (Laserlab-Europe).

Abstract: Stroke is a significant cause of morbidity and mortality, and a major socioeconomic burden. Facilitating the repair and recovery processes is among the highest priorities in translational neuroscience with the ultimate goal of improving functional outcome. In the context of injury progression and mechanisms of repair, our understanding of changes in each cell type and specific interactions between cells, would improve with imaging of structural and functional changes of specific cell populations. Owing to recent advances in optical imaging and optogenetics, we are closer than ever to achieving the goal of understanding causal relationships between neuronal rewiring and recovery of impaired functions. This also opens up tremendous possibilities to investigate and manipulate brain rewiring *in vivo* before and after stroke. We are able to observe and modify neuronal activity in real-time- in the intact and stroke impaired brain in rodents. This symposium will discuss the application of state-of-the-art techniques using light in the field of basic stroke research: From combined approaches of optogenetics with fMRI, laser speckle imaging, 2-photon microscopy, and electrophysiology to the usage of robotics in rehabilitation. The methods discussed are useful to our understanding from stroke pathology to the behavioral outcome and may allow discovering new targets for therapeutic approaches.

Furthermore, the presented methods open up new possibilities to assess the efficiency of rehabilitative strategies by understanding their external influence for intrinsic repair mechanisms on a neurobiological basis. Closing the long-standing gap of translating findings in basic stroke models to clinical relevant strategies for stroke patients is our priority. Thus, this *bench to bedside* topic is of interest to a wide audience interested in the development and application of new optical technologies, advanced network analysis methods, mechanisms of brain plasticity and injury, and optimizing rehabilitative strategies. The symposium will have translational relevance. Results span from analyses of functional and structural network changes during stroke recovery to preclinical evaluation of rehabilitative approaches.

M13.01. Chair

A. Wahl;

University of Zurich, Zuerich, SWITZERLAND.

Disclosures:

M13.02. Co Chair

A. Allegra Mascaro;

European Laboratory for Non-Linear Spectroscopy, Sesto Fiorentino, ITALY.

Disclosures: A. Allegra Mascaro: None.

M13.03. Combining robotic therapy and neuromodulation after stroke: novel approaches in a mouse model

C. Spalletti;

IN-CNR, Pisa, ITALY.

Disclosures: C. Spalletti: None.

M13.04. The role of interhemispheric network activity on stroke recovery

A. Bauer;

Departments of Radiology and Biomedical Engineering, Washington University in St. Louis, Washington, MO.

Disclosures:

M13.05. Gamma range activation of inhibitory neurons in the acute phase after stroke attenuates vascular and behavioural dysfunction

M. Balbi;

The Queensland Brain Institute, The University of Queensland, Brisbane, AUSTRALIA.

Disclosures: M. Balbi: None.

M13.06. Closing the loop from neuronal rewiring to functional recovery after stroke

A. Wahl;

University of Zurich, Zuerich, SWITZERLAND.

Disclosures: A. Wahl: None.

M13.07. Unraveling rehabilitation-induced cortical plasticity via multi-scale imaging and manipulation of neuronal activity after stroke

A. Allegra Mascaro;

Neuroscience Institute, National Research Council, Pisa, ITALY.

Disclosures: A. Allegra Mascaro: None.

Minisymposium

M14. Applying Mind-Body Approaches to Treat Pain — From Animal Model to Clinical Settings

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Chronic pain is a common disorder. An increase in the misuse and complications of opioids has resulted in a serious substance abuse crisis, highlighting the need for non-opioid treatments of chronic pain. Mind-body approaches, including acupuncture, meditation, and Tai Chi, are widely-used nonpharmacologic self-regulation approaches for the management of pain. This symposium will focus on recent developments in the neurophysiological mechanisms and clinical implications of mind-body approaches.

M14.01. Chair

J. Kong;

Psychiatry, Massachusetts General Hospital, Charlestown, MA.

Disclosures:

M14.02. Co Chair

Y. Chae;

Kyung Hee University, Seoul, KOREA, REPUBLIC OF.

Disclosures:

M14.03. The role of orexin-endocannabinoid and opioid signaling in the analgesic effect produced by mind-body approaches

Y. Chen;

China Medical University, Taichung, TAIWAN.

Disclosures: Y. Chen: None.

M14.04. The puzzle of myofascial pain: a mind and body approach

H. M. Langevin;

NCCIH, Bethesda, MD.

Disclosures: H.M. Langevin: None.

Minisymposium

M14. Applying Mind-Body Approaches to Treat Pain — From Animal Model to Clinical Settings

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Chronic pain is a common disorder. An increase in the misuse and complications of opioids has resulted in a serious substance abuse crisis, highlighting the need for non-opioid treatments of chronic pain. Mind-body approaches, including acupuncture, meditation, and Tai Chi, are widely-used nonpharmacologic self-regulation approaches for the management of pain. This symposium will focus on recent developments in the neurophysiological mechanisms and clinical implications of mind-body approaches.

M14.05. The role of cognitive factors in mind-body approaches

Y. Chae;

Kyung Hee University, Seoul, KOREA, REPUBLIC OF.

Disclosures: Y. Chae: None.

M14.06. Mind-body approaches on chronic musculoskeletal pain: findings from clinical studies

C. Wang;

Tufts Medical Center, Boston, MA.

Disclosures:

M14.07. Predicting clinical response to acupuncture in chronic low back pain - development and external validation of a prediction model

J. Kong;

School of Medicine, Stanford University, Palo Alto, CA.

Disclosures: J. Kong: None.

M14.08. Mind-body approaches in pain management: mechanisms and clinical implications

J. Kong;

Psychiatry, Massachusetts General Hospital, Charlestown, MA.

Disclosures: J. Kong: None.

Minisymposium

M15. Building Vision — Lessons From Cell Type-Specific Subcortical Circuits for Sensory Processing and Behavior

Location: Virtual Only

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

Description: How are neurons wired, and what does their organization tell us about how the brain processes information from the outside world? The visual system is a leading model for elucidating mechanisms governing neural circuit formation and sensory information processing. This minisymposium will highlight exciting discoveries in the study of subcortical visual circuits at every processing level, from retina to thalamus and midbrain, with new insights into cell type-specific roles in visual behavior.

Support: NIH F99/K00 - F99NS113459 (Sabbagh)
NIGMS P20 - P20GM103650 (Hoy)

M15.01. Chair

U. Sabbagh;

Fralin Biomedical Research Institute, Virginia Tech, Roanoke, VA.

Disclosures:

M15.02. Routing and modulation of retinal input to circuits underlying aversive behavior

K. Reinhard;

NeuroElectronics Research Flanders, KU Leuven, Leuven, BELGIUM.

Disclosures: K. Reinhard: None.

M15.04. A cell type-specific laminated architecture of GABAergic neurons in the vLGN

U. Sabbagh;

McGovern Institute, MIT, Cambridge, MA.

Disclosures: U. Sabbagh: None.

M15.05. Functional organization and modulation in the visual thalamus

L. Liang;

Department of Neuroscience, Yale University, New Haven, CT.

Disclosures:

M15.06. Mapping subcellular microcircuits in the retina and dLGN with 3DEM

J. L. Morgan;

Ophthalmology and Visual Sciences, Washington University School of Medicine, St. Louis, MO.

Disclosures:

M15.07. Genetic dissection of retinal circuits

Y. Peng;

Department of Ophthalmology, Stein Eye Institute, University of California, Los Angeles, Los Angeles, CA.

Disclosures: Y. Peng: None.

Minisymposium

M16. Neuroimmunology of Chronic Pain — From Rodents to Humans

Location: Virtual Only

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

Description: The signaling partnership between immune cells, glia, and neurons is now recognized as indispensable for chronic pain. However, these multi-cellular interactions have not yet been exploited to effectively diagnose and treat chronic pain. Covering a range of model systems and pain disorders, this translational minisymposium will highlight recent advances in understanding fundamental signaling mechanisms, novel therapeutic targets, and technological developments to diagnose chronic pain.

Support: NIH RF1NS113840
Australian Research Council Future Fellowship (FT180100565)
Australian Research Council Centre of Excellence for Nanoscale BioPhotonics (CE140100003)
Rita Allen Foundation
NIH K22NS096030
NIH K08NS094547
NIH R01NS095937

Abstract: The signaling partnership between immune cells, glia, and neurons is now recognized as indispensable for chronic pain. The role of sex differences in the immune response to orthopedic injury and recovery. Using time-of-flight mass spectrometry (CyTOF), sex-related cell-type specific attributes were identified that may be key to the higher prevalence of chronic pain conditions after injury in females. Using a combination of behavioral and biochemical assays, coupled with genetic and pharmacologic manipulation, activation of Nrf2—a transcription factor and master regulator of the antioxidant response—will be shown to control the severity of neuropathic pain. These studies may validate Nrf2 as a new therapeutic target for neuropathic pain. The cell-specific roles for endocannabinoid signaling through CB₁ receptors will be delineated using new conditional knockout mouse models. Initial work highlights a cell

type-dependent dissociation between CB₁R analgesia and inflammation. This work begins to settle long-standing controversies in cannabinoid analgesia that will have an impact on the design of therapeutics targeting this system. It will demonstrate how pain processes can be decoupled from inflammation in rheumatoid arthritis. The presentation will highlight the role of autoantibodies to collagen type II signalling through neuronal Fcγ receptors to drive arthritis-induced pain. The use of spectral color features in peripheral blood to build predictive measures of pain state will be described across several clinical populations. A summary of these clinical explorations will be presented, along with the bridge to scale these minimally invasive, cost effective and “labless” diagnostics to human pain populations and livestock farming settings. The role of the 18kDa translocator protein (TSPO), which is dramatically upregulated in activated glial cells, will be discussed as a positron emission tomography (PET) imaging marker of neuroinflammation in pain and other human disorders. These studies provide a rationale for a more aggressive exploration of glial cells as therapeutic targets for human pain. Collectively, the presentations will provide nuanced insight into neuron-immune interactions, and offer new diagnostic and therapeutic targets for chronic pain.

M16.01. Chair

P. M. Grace;

Symptom Research, University of Texas MD Anderson Cancer Center, Houston, TX.

Disclosures:

M16.02. Co Chair

M. R. Hutchinson;

University of Adelaide, Adelaide, AUSTRALIA.

Disclosures:

M16.03. Whole system immune phenotyping of male and female mice reveals striking sex differences and similarities in the immune response to painful injury

V. L. Tawfik;

Department of Anesthesiology, Perioperative, Stanford University School of Medicine, Redwood City, CA.

Disclosures:

M16.04. Quantifying pain through light-based technologies – the journey to clinical translation

M. R. Hutchinson;

Univ Adelaide, Adelaide, AUSTRALIA.

Disclosures: M.R. Hutchinson: 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.; Novartis, Abbott, Regeneus, Alyra. E. Ownership Interest (stock, stock options, royalty, receipt of

intellectual property rights/patent holder, excluding diversified mutual funds); Bionomics, Alyra. F. Consulting Fees (e.g., advisory boards); APRA, Regeneus.

M16.05. The cell-specific role of cannabinoid receptor 1 (CB1R) in neurogenic inflammation and analgesia

M. D. Burton;

Behavioral and Brain Sciences, University of Texas At Dallas, Richardson, TX.

Disclosures: M.D. Burton: None.

M16.06. Alleviating neuropathic pain and neuroinflammatory mechanisms via activation of Nrf2.

P. M. Grace;

Symptom Research, University of Texas MD Anderson Cancer Center, Houston, TX.

Disclosures: P.M. Grace: 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.; Biogen Inc..

M16.07. Towards neuroinflammatory PET signatures of human pain disorders

M. L. Loggia;

Radiology, Massachusetts General Hospital / Harvard Medical School, Charlestown, MA.

Disclosures: M.L. Loggia: None.

M16.08. Unraveling roles of autoantibodies and neuronal Fcγ receptors in mechanisms contributing to joint pain in arthritis

C. I. Svensson;

Karolinska Institutet, Stockholm, SWEDEN.

Disclosures: C.I. Svensson: None.

Minisymposium

M17. New Insights Into the Neural Mechanisms of Sound Encoding

Location: Virtual Only

Time: Monday, November 8, 2021, 10:45 AM - 11:45 AM

Description: The encoding of sound makes enormous demands on the neural circuitry of the auditory system. This minisymposium will highlight recent research that has utilized a diversity of techniques — imaging, single cell RNA sequencing, and electrophysiology — to provide new insights into the mechanisms that contribute to the fast and reliable encoding of sound over a range of intensities. Importantly, these mechanisms also underlie susceptibility of the auditory system to insulting noise, ototoxic drugs and aging.

Abstract: The encoding of sound makes enormous demands on the neural circuitry of the auditory system. This mini-symposium highlights recent progress identifying the molecular, cellular, and circuit-level specializations that contribute to the fast and reliable encoding of sound along the length of the auditory pathway—from the sensorineural structures in the inner ear to descending projections from the brain. These findings illustrate that the auditory system is more dynamically regulated than previously appreciated and that the encoding of sound requires various intrinsic and also experience-dependent mechanisms. Importantly, hearing loss is the most common sensory impairment (affecting an estimated 460 million people). As this mini-symposium highlights, the specializations that enable proper sound encoding, in turn, contribute to the vulnerability of the auditory system to insulting noise, ototoxic drugs and age-related loss of function (including presbycusis, hyperacusis, and tinnitus). Thus, insights into these specializations will facilitate the development of mechanistic-based treatments for hearing loss. Finally, the auditory system is an excellent model system to examine neural circuits, from developmental formation and refinement, to synaptic signaling, to regulation by neuromodulatory circuits, and finally to susceptibility to neural degeneration. Therefore, this mini-symposium highlights the relevance to other sensory systems and disciplines of neuroscience.

M17.01. Chair

S. Pyott;

Hanzeplein 1, University Medical Center Groningen, Groningen, NETHERLANDS.

Disclosures:

M17.02. Co Chair

T. Moser;

Institute for Auditory Neuroscience, University Medical Center Goettingen, Goettingen, GERMANY.

Disclosures:

M17.03. Using zebrafish to light up how hair cell sensory systems function

K. Kindt;

Section on Sensory Cell Development and Function, NIDCD/NIH, Bethesda, MD.

Disclosures: K. Kindt: None.

M17.04. The origins and impact of spiral ganglion neuron diversity for responding to sound

L. V. Goodrich;

Harvard Medical School, Boston, MA.

Disclosures:

M17.05. Sound encoding in the auditory nerve

J. Bourien;

University of Montpellier, Montpellier, FRANCE.

Disclosures:

M17.06. Molecular determinants of auditory neuron excitability

S. Pyott;

Hanzeplein 1, University Medical Center Groningen, Groningen, NETHERLANDS.

Disclosures: S. Pyott: None.

M17.07. Mechanisms of sound intensity encoding: what heterogeneous afferent synapses and neurons are good for

T. Moser;

Institute for Auditory Neuroscience, University Medical Center Goettingen, Goettingen, GERMANY.

Disclosures:

M17.08. Oscillatory networks of auditory attention extend to the cochlea

C. Dragicevic;

Departamento de Neurociencias, University of Chile, Santiago, CHILE.

Disclosures: C. Dragicevic: None.

Minisymposium

M18. Decoding Speech From Neural Signals

Location: Virtual Only

Time: Tuesday, November 9, 2021, 12:00 PM - 1:00 PM

Description: This minisymposium will present recent advances in decoding speech from direct neural recordings using electrocorticography, stereoelectroencephalography, and Utah arrays. These talks will cover progress toward synthesizing speech with brain-computer interfaces, as well as remaining challenges for restoring speech to patients. Speakers will also delve into the neuroscience of speech production and perception, with special emphasis on how improved biological understanding can inform translational efforts.

Support: Hamilton: The Texas Speech-Language-Hearing Foundation
Herff: Dutch Research Council (NWO)
Berezutskaya: European Research Council (Advanced iConnect Project Grant ADV 320708) and the Netherlands Organisation for Scientific Research (Language in Interaction Project Gravitation Grant 024.001.006)

Abstract: Speaking is a fast and intuitive means of communication, and its loss is devastating to patients. Recent years have seen rapid progress in developing brain-computer interfaces (BCIs) aimed at restoring lost speech by decoding the neural correlates of speaking. There have been compelling proof-of-concept demonstrations, but further progress towards clinically viable speech BCIs requires a thorough understanding of speech-related brain processes, how to decode these signals, and how to do so effectively in people no longer able to speak. This minisymposium brings together six groups to provide a snapshot of the current state-of-the-art in speech decoding using non-invasive methods and electrocorticography, sEEG, and Utah arrays. Two related challenges in this endeavor is that it is difficult to obtain direct human brain recordings, and that animal models of speech are limited. One talk will highlight recent progress in establishing non-human primates as a model in which speech perception signals can be obtained using implanted electrodes that are similar to those being used in pilot human clinical trials. The other talks will present human data obtained from a range of different clinical activities. These presentations will showcase the utility of bringing a range of different recording methods to bear on this pressing medical challenge. Speech-related neural activity spans many brain areas involved in both speech perception and production. Key open questions for developing speech BCIs is what area(s) to record from, what representation(s) to decode (e.g., auditory or articulatory), and of what form the system's output should be (e.g., continuous audio, phonemes, discrete words). The talks will present results of decoding both speech perception and production, across many brain areas, and with different output types. Furthermore, the input signals for speech decoding are highly complex in both time and space (input channels). These talks will present the results of applying a variety of modern machine learning methods to extract meaning from these rich signals.

It is our hope that the interactions of this minisymposium's speakers and audience will foster an exciting exchange of ideas that accelerates our common effort to help patients who are unable to speak.

M18.01. Chair

S. D. Stavisky;

Neurosurgery, Stanford University, Stanford, CA.

Disclosures:

M18.02. Co Chair

C. Herff;

Neurosurgery, Maastricht University, Maastricht, NETHERLANDS.

Disclosures:

M18.03. Towards reconstructing intelligible speech from the human auditory cortex

N. Mesgarani;

Columbia University, New York, NY.

Disclosures:

M18.04. Optimizing decoding and evaluation strategies for speech reconstruction from ECoG

J. Berezutskaya;

Brain, Cognition, and Behavior, Donders Institute, Utrecht, NETHERLANDS.

Disclosures: J. Berezutskaya: None.

M18.05. Advances in speech neuroprosthetic technology

D. A. Moses;

Neurosurgery, UCSF, San Francisco, CA.

Disclosures: D.A. Moses: None.

M18.06. Invasive and noninvasive approaches to decoding naturalistic speech from the brain

L. S. Hamilton;

Neurology/Communication Sciences and Disorders, University of Texas At Austin, Austin, TX.

Disclosures:

M18.07. Decoding speech from spike-based neural population recordings in the NHP auditory cortex

J. Lee;

Brown University, providence, RI.

Disclosures: J. Lee: None.

M18.08. Decoding speech from stereotactic EEG

C. Herff;

Neurosurgery, Maastricht University, Maastricht, NETHERLANDS.

Disclosures:

Minisymposium

M19. Rodents and Monkeys — The Two Dominant Mammalian Models and Their Respective Contributions to Neuroscience

Location: Virtual Only

Time: Monday, November 8, 2021, 12:00 PM - 1:00 PM

Description: Rodents and monkeys are the two most common mammalian models to study the nervous system. The rodent model offers a suite of powerful genetic and molecular tools that enable a detailed dissection of the role of neuronal circuits in behavior, while the primate model offers a complex behavioral repertoire and a nervous system that is more similar to that of

humans. This minisymposium will emphasize the respective contributions of the two models across multiple lines of brain research.

Abstract: Rodents and monkeys are the two most common mammalian models to study the nervous system. The rodent model offers a suite of powerful genetic and molecular tools that enable a detailed dissection of the role of neuronal circuits in behavior. The primate model offers a complex behavioral repertoire and a nervous system that is more similar to that of humans. In this minisymposium, we will discuss the respective contributions of the two models across three lines of inquiry in systems neuroscience - social cognition, vision, somatosensation - and one in clinical neuroscience. Each of the primary talks involves pairing a primate and a rodent researcher together to highlight the advantages and disadvantages of each model in these topics in order to emphasize the complementary nature of these model systems. Several recent legislative actions in the US Congress have sought to significantly constrain or eliminate biomedical research in nonhuman primates. These efforts highlight the lack of awareness in both the scientific and lay audience regarding the pivotal role of nonhuman primate models, particularly in neuroscience. By highlighting the complementary roles of rodent and primate models neuroscientific research, as well as the areas of research that can only be studied in primates, we aim to counter these legislative actions by re-educating the scientific community about these critical issues and highlighting opportunities for intersections between these foundational animal models to further our understanding of neural systems.

M19.01. Chair

C. T. Miller;

Cortical Systems and Behavior Laboratory, University of California, San Diego, La Jolla, CA.

Disclosures:

M19.02. Co Chair

M. A. Basso;

Psychiatry and Biobehavioral Sciences and Neurobiology, University of California, Los Angeles, Los Angeles, CA.

Disclosures:

M19.03. Challenges facing primate neuroscience research in the next decade and the importance of complementary model systems.

M. A. Basso;

Psychiatry and Biobehavioral Sciences and Neurobiology, UCLA, Los Angeles, CA.

Disclosures:

M19.04. Social genes, brains and behavior in rodents and primates: implications for understanding neuropsychiatric disorders

G. Feng;

Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA.

Disclosures: G. Feng: 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.; Deerfield Management.

M19.05. Vibrissae and fingertips: what can rodents and primates teach us about touch?

D. H. O'Connor;

Department of Neuroscience, The Johns Hopkins University School of Medicine, Baltimore, MD.

Disclosures:

M19.06. Studying vision in natural contexts across rodents and primates.

C. M. Niell;

Institute of Neuroscience, University of Oregon, Eugene, OR.

Disclosures: C.M. Niell: None.

M19.07. Thalamic functions in distributed cognitive control: comparative perspectives

S. Kastner;

Princeton Neuroscience Institute, Princeton University, Princeton, NJ.

Disclosures: S. Kastner: None.

M19.08. A comparison of rodents and primates as animal models of parkinson's disease (PD)

M. E. Emborg;

Wisconsin National Primate Research Center, UW Madison, Madison, WI.

Disclosures: M.E. Emborg: None.

Minisymposium

M20. The Neural Encoding and Representation of Time for Sensorimotor Control and Learning

Location: Virtual Only

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

Description: The ability to produce and learn actions in the real world with precise timing is critical for survival in any mobile organism. Yet, a study on mechanisms involved in timed motor behavior and its relation to time perception is relatively scarce. This symposium will present new evidence from behavioral, computational, and neural studies in humans and non-

human primates, proposing the pivotal link between sensorimotor control and temporal processing as well as new theoretical frameworks on timing.

Support: CONACYT A1-S-8430
NSF BCS 1460633
NSF DGE 1633722.
NIH-R01-HD087089
NSF-M3X-1825942
NSF-CRCNS-1723998
NWO 016.Vidi.185.137 .

Abstract: This mini-symposium is motivated to provide converging behavioral, computational, and neural evidence to support the link between the mechanisms involved in sensorimotor control and those in time and rhythm perception. While multidisciplinary approaches are taken across individual presentations, each presentation is also intertwined, which enables us to capitalize on new breakthroughs in emerging areas. Specifically, our presentations will be divided mainly into three parts. In the first part, we will discuss human behavioral mechanisms: how the practice of human complex movements without explicit periodicity, such as throwing enhances the accuracy of motor timing (Sternad), which also, in turn, can be generalized to the improvement of time perception in isolation later (Song). In the second part, Merchant and Balasubramaniam will reveal neural data from motor regions of non-human primates using single-unit recording (Merchant) and humans using the continuous theta-burst transcranial magnetic stimulation (cTBS) (Balasubramaniam) which enables such a close interplay between sensorimotor control and time perception reported in the first part possible. In the last part, Haegens and Jazayeri will challenge the current prevailing theoretical frameworks. Using the method of psychophysics, MEG and ECoG recordings, Haegens will revisit the neural entrainment hypothesis by demonstrating that periodic stimulus streams rather than frequency-specific neural entrainment of ongoing oscillations lead to a general phase reset. Jazayeri will present a dynamic systems perspective as a potential unified framework for understanding the computational principles of temporal flexibility in a range of sensorimotor behaviors sequence generation based on non-human primate studies. Taken together, this session as a whole will provide a new opportunity to extend our understanding of our joint efforts to understand ecologically valid situations. It will also blur the conventional distinctions and emphasizing the interactions between motor, perceptual, and cognitive processes for sensorimotor behaviors in complex environments.

M20.01. Chair

J. Song;
Cognitive, Linguistic, Brown University, Providence, RI.

Disclosures:

M20.02. The role of the motor system in the processing of rhythm and time

R. Balasubramaniam;

Cognitive, University of California, Merced, Merced, CA.

Disclosures: R. Balasubramaniam: None.

M20.03. Tricks to achieve timing or throwing accuracy in sensorimotor control

D. Sternad;

Departments of Biology, Electrical, Northeastern University, Boston, MA.

Disclosures: D. Sternad: None.

M20.04. Brain dynamics in the primate motor system during rhythmic timing

H. Merchant;

Inst. de Neurobiologia UNAM, Queretaro, MEXICO.

Disclosures: H. Merchant: None.

M20.05. Entrainment revisited

S. Haegens;

Dept. of Psychiatry, Columbia University, New York, NY.

Disclosures:

M20.06. Improved motor timing enhances time perception

J. Song;

Cognitive, Linguistic, Brown University, Providence, RI.

Disclosures: J. Song: None.

M20.07. Timing in the brain through the lens of dynamical systems

M. Jazayeri;

Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA.

Disclosures: M. Jazayeri: None.

Minisymposium

M21. Endogenous Opioid Signaling in the Nucleus Accumbens — New Methods of Analysis, Novel Functions, and Emerging Therapeutic Opportunities

Location: Virtual Only

Time: Monday, November 8, 2021, 12:00 PM - 1:00 PM

Description: This minisymposium will highlight recent developments in understanding endogenous opioid signaling, with a focus on the nucleus accumbens — a brain region highly

enriched in opioid peptides and receptors. Speakers will describe newly developed tools that are facilitating discovery of novel functions for endogenous opioid signaling. Progress in this field is leading toward new therapeutic opportunities for engaging endogenous opioid signaling while avoiding risk for abuse or addiction.

Support: NIH R01 DA048946
NIH R21 DA050120

Abstract: The opioid epidemic plaguing the United States has brought great attention to exogenous opioid effects on the brain. However, it simultaneously highlights our limited understanding of endogenous opioid signaling, including the identity of opioid peptides and localization of opioid receptors responsible for cellular and behavioral effects. Emerging technologies are now leading to breakthroughs in the detection of endogenous opioid peptides and their functional effects on opioid receptors in the nucleus accumbens - a key brain region for motivated behavior that is also implicated in the pathophysiology of many brain disorders. Speakers in this session will describe cutting-edge methods for measuring release of opioid peptides and other neurotransmitters in freely moving rodents, as well as genetically encoded fluorescent sensors for detecting opioid receptor activation. Next, speakers will discuss interactions between dopamine receptor activation and enkephalin synthesis that regulate cocaine reward, and a novel opioid-sensitive projection from the dorsal raphe to the nucleus accumbens that regulates motivation. Finally, speakers will describe the extracellular enzymes that degrade opioid peptides and terminate endogenous opioid signaling, and discuss the possibility that these peptidase enzymes represent therapeutic targets for regulating mood. Manipulation of endogenous opioid signals that are naturally present in the brain may provide an alternative therapeutic strategy to engage opioid signaling pathways for clinical benefit, while minimizing iatrogenic effects like dependence, abuse, and addiction.

M21.01. Chair

P. E. Rothwell;

Neuroscience, University of Minnesota, Minneapolis, MN.

Disclosures:

M21.02. Co Chair

L. Dobbs;

Waggoner Center for Alcohol and Addiction Research, The University of Texas at Austin, Austin, TX.

Disclosures:

M21.03. Dopamine D2 receptors bidirectionally regulate striatal enkephalin expression to affect cocaine reward

L. Dobbs;

Waggoner Center for Alcohol and Addiction Research, University of Texas at Austin, Austin, TX.

Disclosures: L. Dobbs: None.

M21.04. Dynorphin/kappa-opioid receptor regulation of motivational and emotional circuitry

H. Tejada;

National Institutes of Health, Bethesda, MD.

Disclosures:

M21.05. Biased signaling modifies the rewarding properties of mu-opioid receptors

D. Castro;

Department of Anesthesiology, University of Washington, Seattle, WA.

Disclosures: D. Castro: None.

M21.06. Rapid and sensitive detection of opioid peptides

R. Al-Hasani;

Pharmaceutical and Administrative Sciences, St. Louis College of Pharmacy, St. Louis, MO.

Disclosures:

M21.07. Angiotensin converting enzyme governs endogenous opioid signaling in nucleus accumbens circuits

P. E. Rothwell;

Neuroscience, University of Minnesota, Minneapolis, MN.

Disclosures: P.E. Rothwell: None.

M21.08. Drug dependence dials down the effects of endogenous opioids

E. Bagley;

The University of Sydney, Sydney, AUSTRALIA.

Disclosures: E. Bagley: None.

Minisymposium

M22. From Circuits to Chromatin — Epigenetic (Dys)regulation in Cognitive Health and Disease

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Neural circuit activity cooperates with transcriptional mechanisms within each cell to guide and adapt behavior. Fundamental to this interaction are chromatin-based processes that mediate activity-dependent gene expression and neural plasticity so as to encode new information. This minisymposium will explore the epigenetic controls that operate in distinct neural subtypes to shape behavior and provide translational insights about circuit-chromatin dysregulation in neuropsychiatric disorders.

Abstract: Neural circuit activity cooperates with transcriptional mechanisms within each cell to guide and adapt behavior. Fundamental to this interaction are chromatin-based processes that mediate activity-dependent gene expression and neural plasticity so as to encode new information. This minisymposium will explore the epigenetic controls that operate in distinct neural subtypes to shape behavior and provide translational insights about circuit-chromatin dysregulation in neuropsychiatric disorders.

M22.01. Chair

P. Mews;

Neuroscience, Icahn School of Medicine at Mount Sinai, New York, NY.

Disclosures:

M22.02. Histone variant macroH2A1 is a positive regulator of memory

I. Zovkic;

Department of Psychology, University of Toronto Mississauga, Mississauga, ON, CANADA.

Disclosures: I. Zovkic: None.

M22.03. Epigenetic regulation of activity-dependent gene expression.

T. Abel;

Department of Molecular Physiology and Biophysics, University of Iowa, Iowa City, IA.

Disclosures: T. Abel: None.

M22.04. Long noncoding RNA-mediated epigenetic regulation of gene expression: enigmatic players in learning and memory

T. W. Bredy;

Queensland Brain Institute, The University of Queensland, St Lucia, AUSTRALIA.

Disclosures: T.W. Bredy: None.

M22.05. Epigenetic priming underpins persistent changes in activity-induced transcription linked to drug relapse.

P. Mews;

Neuroscience, Icahn School of Medicine at Mount Sinai, New York, NY.

Disclosures: P. Mews: None.

M22.06. Divergent circuit specific adaptations in the striatal epigenome control the transition to addiction in males and females.

E. S. Calipari;

Pharmacology, Vanderbilt University, Nashville, TN.

Disclosures: E.S. Calipari: None.

M22.07. A dopamine-induced gene expression signature regulates neuronal function and cocaine response.

J. J. Day;

Neurobiology, University of Alabama At Birmingham, Birmingham, AL.

Disclosures: J.J. Day: None.

Minisymposium

M23. Mechanisms of Stress in the Brain and Beyond

Location: Virtual Only

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

Description: The brain is the central organ in adapting to social and physical stress via multiple interacting mediators, from neurogenesis to metabolic factors to epigenetic mechanisms. Stress causes an imbalance in brain networks subserving cognition, reward-seeking behavior, and mood. This minisymposium will discuss recent basic and translational findings on neurobiological mechanisms of neuroplasticity relating to stress as well as avenues for novel treatments to promote resilience to stress-related diseases, such as depression.

Support: Hope for Depression Research Foundation

Abstract: This mini-symposium highlights the discovery of steroid hormone receptors in the brain (McEwen BS et al, Nature 1968) and focuses on recent advances in neurobiological mechanisms of the effects of stress on brain circuits subserving cognition, reward-seeking behavior and mood. The mini-symposium also recognizes the critical relationship between the brain and the rest of the body in determining individual stress responsiveness. Dr. Nasca will introduce the session and discuss the effects of stress on mitochondrial health, and how therapeutic interventions acting on mitochondrial metabolism can modulate hippocampal glutamatergic function and behavior. Dr. Turner will present recent findings on the role of the connective tissue growth factor (CTGF) in vulnerability and resilience to stress, and she will discuss how CTGF may represent a therapeutic target in major depressive disorders. Dr. Hultman will present new data on brain networks involving activity patterns originating in the prefrontal cortex and ventral striatum and converging in ventral hippocampus to mediate responsiveness to stress. The next speaker Dr. Anacker will show calcium imaging data on the

role of activity of the ventral dentate gyrus (vDG) neurons in determining vulnerability to stress, and how targeting neurogenesis confers resilience to chronic stress by inhibiting the activity of vDG mature granule cells. Dr. Issler will present new data on the role of long non-coding RNAs (lncRNAs) in depression, the complex region- and sex-specific patterns of lncRNAs regulation, and will discuss the causal role of specific lncRNAs in the responsiveness to social defeat stress. This talk will be followed by Dr. Parekh presentation on findings of prefrontal cortical function in stress models and reward-seeking behavior. Dr. Silveira will present data on the relationship between stress, metabolism and neurodevelopment through gene networks, including hippocampal insulin-receptor related gene pathways. Together, these presentations will support the notion that multiple interacting mediators, from neurogenesis to metabolic factors to epigenetic mechanisms, brain networks and mitochondrial functioning modulate the responsiveness to stress, and can serve as targets for new therapeutic avenues. This mini-symposium brings together a group of early-career scientists from laboratories supported by the Hope for Depression Research Foundation (HDRF) Task force, a network which Dr. Bruce McEwen initiated together with other major neuroscientists with the goal to better understand the neurobiology of depression in order to develop novel more effective therapeutic strategies.

M23.01. Chair

C. Nasca;

Neuroendocrinology, The Rockefeller University, New York, NY.

Disclosures:

M23.02. Co Chair

C. A. Turner;

Molec, University of Michigan, Ann Arbor, MI.

Disclosures:

M23.03. Stress, genetics and mood: impact of covid-19 on a college freshman sample

C. A. Turner;

Molec, University of Michigan, Ann Arbor, MI.

Disclosures: C.A. Turner: None.

M23.04. Non-coding RNAs in stress susceptibility and resilience

O. Issler;

Mount Sinai School of Medicine, New York, NY.

Disclosures: O. Issler: None.

M23.05. Brain networks underlying stress induced behavioral dysfunction

R. Hultman;

Dept. of Molecular Physiology and Biophysics, Iowa Neuroscience Institute, Iowa City, IA.

Disclosures:

M23.06. Integrating stress, metabolism and neurodevelopment through gene networks

P. Pelufo Silveira;

McGill University, Montreal, QC, CANADA.

Disclosures: P. Pelufo Silveira: None.

M23.07. Stress regulation of hippocampal neurogenesis and dentate gyrus function

C. Anacker;

Columbia University, New York, NY.

Disclosures:

M23.08. Molecular endophenotypes of mood and cognitive disorders: from computational approaches to new targets for personalized treatment models

B. Bigio;

Neuroendocrinology, The Rockefeller University, New York, NY.

Disclosures: B. Bigio: None.

Minisymposium

M24. Neural Mechanisms of Rodent Vocal Communication

Location: Virtual Only

Time: Tuesday, November 9, 2021, 12:00 PM - 1:00 PM

Description: Vocal communication is a complex social behavior that requires the coordination of multiple brain regions. Despite decades of work on the neural basis of bird song, parallel research on mammals has lagged far behind. This minisymposium will present current perspectives on the neural mechanisms underlying mammalian vocal production and perception. It will highlight research from a variety of rodent species and span multiple levels of analysis, from genes to circuits.

Support: German Research Foundation (grants WO 1732/1-1, WO 1732/4-1, WO 1732/4-2)
National Institutes of Health (grants MH 115267, NS 107616, HD 088411, DC 12557, DC 013826, MH 117778)
Simons Foundation Society of Fellows
National Science Foundation GRFP
Leon Levy Foundation

Pew Scholars and Fellows Program
McKnight Foundation

Abstract: Using vocalizations to compete and cooperate is common across the animal kingdom. Vocalizations involve the coordination of several neural systems, and yet, a detailed understanding of these mechanisms has lagged behind parallel work in avian models. The last decade saw a surge in ethological, genetic, and neuroscience tools to explore naturalistic behaviors in rodents. This minisymposium brings together six speakers to share their perspectives on the biological substrates underlying production and perception of rodent vocalizations. The speakers will share current research on traditional and non-model rodent species with experimental approaches ranging across levels of analysis, from genes, to synapses and circuits. M Wöhr will talk about how the production and responses to discrete 22-kHz and 50-kHz rat vocalizations are modulated by brain regions like amygdala and nucleus accumbens, dopamine and serotonin systems, and the cross-disorder risk gene CACNA1C. Ö Sungur will discuss how genetic mouse models for Autism Spectrum Disorders (e.g., Shank1) differ from wildtype mice in their vocal development and vocal production across social contexts. A Barker will present work on how naked mole rats learn dialects that encode social information about individual and colony identity and adapt these dialects to different social contexts. Switching to motor production, K Tschida will tell us about specific forebrain synaptic pathways that elicit or suppress vocal production in lab mice. Continuing the theme of motor control, A Banerjee will discuss the role of motor cortex in controlling various aspects of vocalizations using neotropical Alston's singing mice as a novel model system. Finally, M Gustison will talk about how vocal behavior contributes to pair-bond formation in prairie voles and how variation in vocal behavior relates to brain-wide patterns of neural activation. Collectively, the goal of these talks is to provide new insights into the evolution of mammalian neural substrates that govern vocal communication. These insights will also have translational value for understanding the etiology of social communication and related neuropsychiatric disorders.

M24.01. Chair

M. L. Gustison;

University of Texas at Austin, Austin, TX.

Disclosures:

M24.02. Co Chair

A. Banerjee;

Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

Disclosures:

M24.03. Cortical mechanisms of vocal interaction in singing mice

A. Banerjee;

Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

Disclosures:

M24.04. Brain-wide networks underlying vocal behavior during prairie vole pair-bonding

M. L. Gustison;

Department of Integrative Biology, University of Texas at Austin, Austin, TX.

Disclosures:

M24.05. Ultrasonic vocalizations in genetic mouse models for autism spectrum disorder

Ö. Sungur;

Behavioral Neuroscience, Philipps University Marburg, Marburg, GERMANY.

Disclosures: Ö. Sungur: None.

M24.06. Neural circuits for the context-dependent control of vocal communication

K. A. Tschida;

Department of Psychology, Cornell University, Ithaca, NY.

Disclosures: K.A. Tschida: None.

M24.07. Ultrasonic communication in rats: brain and behavior

M. Wöhr;

Department of Biology, KU Leuven, Leuven, BELGIUM.

Disclosures: M. Wöhr: None.

M24.08. Cultural transmission of vocal dialect in the naked mole-rat

A. Barker;

Max Planck Institute for Brain Research, Frankfurt, GERMANY.

Disclosures:

Minisymposium

M25. Alternative Conceptions of Habits and Their Underlying Neural Circuits

Location: Virtual Only

Time: Thursday, November 11, 2021, 12:00 PM - 1:00 PM

Description: The study of habitual behavior and its underlying circuits is impeded by its operationalization as an absence of goal-directed behavior. This minisymposium will focus on new approaches to characterize habit and its neural mechanisms. This transdisciplinary topic will catalyze insightful discussions about the concept of habit that will be addressed under multiple

complementary perspectives, from computational and animal modeling to translation in human research on decision making and psychopathologies.

Support: R01DA035943 / R01AA026306
R01AA026077 / F31AA027439
GRANT ISS-1822571 from NSF
Grant 61454 from the John Templeton Foundation
R01DA030672 / R01MH080318 / R01MH112688
UNIVERSITY OF MINNESOTA J.B. JOHNSTON LAND GRANT CHAIR IN
THE NEUROSCIENCES
UNIVERSITY OF MINNESOTA ACADEMIC INVESTMENTS IN
RESEARCH PROGRAM

Abstract: The study of habitual behavior and the underlying circuits is impeded by its operationalization as an absence of goal-directed behavior. The goal of this symposium is to gather researchers interested in the topic of habitual learning and its underlying neural circuits but questioning the standard methods typically used to conceptualize and assess habits. In this symposium, we will see that alcohol-induced habits can be associated with enhanced contingency control and stronger use of recent action history in a self-paced lever pressing task in mice. Corticostriatal circuits underlying these processes will be presented. In the second presentation, new evidence will be shown describing how the phasic DLS activity at behavior onset regulates how vigorous and habitual the full behavior is. The next presentation will show that presentation of salient reward-predictive cues in a discrete-trial lever pressing task in rats promotes rapid behavioral chunking (and ultimately habit), encoded by a specific activity pattern in the dorsal striatum. Another presentation will aim at examining the development of the transition from deliberative and goal-directed strategies to procedural and habitual strategies and the corresponding representational changes in the dorsal striatum of rats as they form habitual action chains in navigation tasks. Current paradigms do not allow determining whether the imbalance between goal-directed and habitual control found in several psychopathologies results from abnormal habit formation or reduced goal-directed control. A novel outcome revaluation paradigm in humans will be presented to address this issue by providing direct evidence for inflexibility as a consequence of habit learning. Finally, the last speaker will open novel perspectives on habit by presenting a new neurocomputational model of habitual decision-making. The transdisciplinary approach of this symposium will allow the field moving forward by investigating habit under multiple angles and by showing that habit is not necessarily a negative result, that is, an absence of goal-directed control, but is in fact characterized by specific features that can be investigated.

M25.01. Chair

Y. Vandaele;

Department of Psychological and Brain Sciences, Johns Hopkins University, Baltimore, MD.

Disclosures:

M25.02. Habits in humans: in search of a better experimental measure

S. De Wit;

Department of Clinical Psychology, University of Amsterdam, Amsterdam, NETHERLANDS.

Disclosures:

M25.03. Procedural learning - action chains as habitual behavior

A. D. Redish;

University of Minnesota, Minneapolis, MN.

Disclosures:

M25.04. A new computational account of habits, biases, and the cost of overriding them

N. Daw;

Princeton University, Princeton, NJ.

Disclosures:

M25.05. Enhanced contingency control in actions insensitive to devaluation

C. Gremel;

University of California San Diego, San Diego, CA.

Disclosures:

M25.06. Roles for the dorsolateral striatum in invigorating old and new habits

K. S. Smith;

Psychological and Brain Sciences, Dartmouth College, Hanover, NH.

Disclosures: K.S. Smith: None.

M25.07. Neural correlates of habits and behavioral chunking in the dorsal striatum

Y. Vandaele;

Department of Psychological and Brain Sciences, Lausanne University Hospital - CHUV, Prilly, SWITZERLAND.

Disclosures: Y. Vandaele: None.

Minisymposium

M26. Early Life Experience and Developmental Plasticity of Neuromodulatory Circuits

Location: Virtual Only

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

Description: Early life is a critical time period where experience interacts with development to shape the neuronal circuitry of emotion, reward, and affect. This minisymposium highlights

emerging work from rodent model and human studies suggesting that early life experiences such as adversity, enrichment, and exposure to drugs of abuse alter the developmental trajectory of monoaminergic circuits, with significant consequences for the physiology and behavior of the adult animal.

Support: Brain and Behavior Research Foundation NARSAD Young Investigator Award
Jacobs Foundation Early Career Research Fellowship
NIH DP2 MH122401
NIH R00 MH109569
NIH R01-MH117791
NIH R00MH115096
Brain and Behavior Research Foundation NARSAD Young Investigator Award

M26.01. Chair

A. M. Polter;
Department of Pharmacology and Physiology, George Washington University, Washington, DC.

Disclosures:

M26.02. Co Chair

C. J. Peña;
Princeton Neuroscience Institute, Princeton University, Princeton, NJ.

Disclosures:

M26.03. Effects of early life experience on dopamine circuit structure and function

T. N. Lerner;
Physiology, Northwestern, Chicago, IL.

Disclosures: T.N. Lerner: None.

M26.04. Early life stress and the maturation of synaptic inputs to the dorsal raphe

A. M. Polter;
Department of Pharmacology and Physiology, George Washington University, Washington, DC.

Disclosures:

M26.05. Early childhood stress is associated with blunted development of ventral tegmental area functional connectivity

A. Mackey;
University of Pennsylvania, Philadelphia, PA.

Disclosures: A. Mackey: None.

M26.06. Impact of early life stress on ventral tegmental area chromatin state and response to future stress

C. J. Pena;

Princeton Neuroscience Institute, Princeton University, Princeton, NJ.

Disclosures: C.J. Pena: None.

M26.08. The sensitivity to the developmental effects of drugs of abuse in adolescence is sexually dimorphic

C. Flores;

Dept of Psych, McGill University, Montreal, QC, CANADA.

Disclosures: C. Flores: None.

Minisymposium

M27. Hallucinogens in Mental Health — Preclinical and Clinical Studies on LSD, Psilocybin, MDMA, and Ketamine

Location: Virtual Only

Time: Monday, November 8, 2021, 12:00 PM - 1:00 PM

Description: A revamped interest for the study of hallucinogens has recently emerged, especially for their potential application in psychiatric disorders. Combining behavioral, neurophysiological, optogenetic, biomolecular and neuroimaging approaches in both humans and rodents, this minisymposium will give an update on the therapeutic findings in the field of hallucinogenic drugs in mental diseases. Differences between psychedelic (i.e., LSD) versus non-psychedelic (i.e., ketamine) hallucinogens will be also highlighted.

M27.01. Chair

G. Gobbi;

McGill University, Montreal, QC, CANADA.

Disclosures:

M27.02. Co Chair

J. Mitchell;

University of California, San Francisco, San Francisco, CA.

Disclosures:

M27.03. Therapeutic efficacy of MDMA-assisted psychotherapy for PTSD and co-morbid conditions

J. Mitchell;

UCSF: Department of Neurology, San Francisco, CA.

Disclosures:

M27.04. Clinical data that reshapes our understanding of ketamine's antidepressant mechanism

B. Heifets;

Stanford University, Stanford, CA.

Disclosures:

M27.05. Lysergic acid diethylamide (LSD) promotes sociability through a dorsal raphe-cortical circuit involving mTOR complex 1 in excitatory neurons

D. De Gregorio;

Università Vita-Salute San Raffaele, Milano, ITALY.

Disclosures: D. De Gregorio: 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.; Dr De Gregorio is consultant at Diamond Therapeutics Inc., Toronto (ON0, Canada).

M27.06. Eukaryotic initiation factor 4E-binding proteins mediate the antidepressant response to ketamine in mice

A. Aguilar-Valles;

Carleton University, Ottawa, ON, CANADA.

Disclosures: A. Aguilar-Valles: None.

M27.07. Pharmacological neuroimaging studies with psychedelics in humans

K. H. Preller;

University of Zurich, Zurich, SWITZERLAND.

Disclosures: K.H. Preller: A. Employment/Salary (full or part-time);; F.Hoffmann-La Roche.

M27.08. Persistent antidepressant-like effects of psychedelics using innate and stress-related rat models

M. Hibicke;

LSU Health Sciences Center, New Orleans, LA.

Disclosures: M. Hibicke: None.

Minisymposium

M28. Limbic Neuropeptidergic Modulators of Emotion and Their Therapeutic Potential for Anxiety and Post-Traumatic Stress Disorder

Location: Virtual Only

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

Description: This minisymposium will present translational research focusing on recent advances in the role of limbic neuropeptides in the regulation of emotion — particularly fear and anxiety — in animal models and human studies. Discussion will focus on the latest research on peptidergic neuromodulators including oxytocin; angiotensin II; vasopressin; and neurokinin B/tachykinin 2 and their unique potential for pharmacotherapy of anxiety disorders and post-traumatic stress disorder.

Abstract: Post-traumatic stress disorder (PTSD) is characterized by inability to properly discriminate between threat and safety and to extinguish learned fear. First-line pharmacotherapies for psychiatric disorders have limited therapeutic efficacy in PTSD. In this mini-symposium we will present recent advances in our understanding of the role of limbic neuropeptides in the regulation of defensive behaviors in both pre-clinical animal models and clinical human studies. We will demonstrate the role of Neurokinin B/Tac2-expressing cells in the bed nucleus of the stria terminalis (BNST) in trauma-induced aggressive behavior. We will also show that Neurokinin 3 receptor (Nk3R) neuropeptide antagonist osanetant enhances memory consolidation in female mice whereas it impairs memory consolidation in males. These results are dependent on the modulation of sex hormones. Clinical studies showed promising effects of angiotensin II receptors blockade for reducing PTSD symptom severity. We will present pre-clinical studies regarding angiotensin receptors in the central amygdala and their role in fear extinction, specifically showing that inhibition of angiotensin receptor type 1 (AT₁Rs) facilitates fear extinction. Neuropeptide oxytocin has demonstrated a unique anxiolytic potential, but its role in the modulation of fear memory appears more complex and brain-site specific. We will show that oxytocin facilitates fear discrimination to predictable vs. unpredictable threats in rat fear-potentiated startle by inhibiting output neurons of the BNST. Using human imaging studies, we will also focus on identifying neural targets of intranasal oxytocin treatment and efficacious treatment strategies, including definition of the best dosage needed for target engagement in male and female volunteers. Finally, we will present data from human subjects showing that highly selective vasopressin 1A receptor antagonist SRX246 reduces anxiety but not fear-potentiated startle in male and female participants. This mini-symposium will address sex-specific effects of limbic neuropeptides in both rodents and humans and the importance of sex-specific pharmacotherapies for PTSD.

M28.01. Chair

J. Dabrowska;

Rosalind Franklin University of Medicine and Science, North Chicago, IL.

Disclosures:

M28.02. Co Chair

P. J. Marvar;

George Washington University, Washington, DC.

Disclosures:

M28.03. Stress-enhanced fear learning, violence, and the neuropeptide Tac2

M. Zelikowsky;

Department of Biology, University of Utah, Salt Lake City, UT.

Disclosures: M. Zelikowsky: None.

M28.04. Sex-opposite effects of the neurokinin 3 receptor neuropeptide in memory consolidation

R. Andero Gali;

Institute of Neurosciences, UAB, Barcelona, SPAIN.

Disclosures: R. Andero Gali: None.

M28.05. Oxytocin facilitates fear memory to predictable threats by inhibiting output neurons of the bed nucleus of the stria terminalis (BNST).

J. A. Dabrowska;

Chicago Medical School RFUMS, North Chicago, IL.

Disclosures: J.A. Dabrowska: None.

M28.06. Clinical translation of oxytocin neuroscience to the treatment of anxiety and posttraumatic stress disorders

R. Hurlemann;

Psychiatry, University of Oldenburg Medical Campus, Bad Zwischenahn, GERMANY.

Disclosures: R. Hurlemann: F. Consulting Fees (e.g., advisory boards); Rovi, Lundbeck/Otsuka, Atheneum Consultations.

M28.07. The role of Angiotensin II in Fear Memory and therapeutic opportunities for PTSD

P. J. Marvar;

The George Washington University, Washington DC, DC.

Disclosures: P.J. Marvar: None.

Minisymposium

M29. Neurobiology of Threat-Induced Behaviors

Location: Virtual Only

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

Description: Different innate and learned threats induce distinct defensive behaviors. However, this diversity of threats and behaviors is rarely explored. This minisymposium will discuss experiments using numerous threats in rodents, including aggressive conspecifics, predators, thermal threats and carbon dioxide. Speakers will discuss circuits that control defensive aggression, escape jumps, escape runs, freezing, avoidance and aversion. They will also consider how sex differences and developmental processes affect these phenomena.

Abstract: Post-traumatic stress disorder (PTSD) is characterized by inability to properly discriminate between threat and safety and to extinguish learned fear. First-line pharmacotherapies for psychiatric disorders have limited therapeutic efficacy in PTSD. In this mini-symposium we will present recent advances in our understanding of the role of limbic neuropeptides in the regulation of defensive behaviors in both pre-clinical animal models and clinical human studies. We will demonstrate the role of Neurokinin B/Tac2-expressing cells in the bed nucleus of the stria terminalis (BNST) in trauma-induced aggressive behavior. We will also show that Neurokinin 3 receptor (Nk3R) neuropeptide antagonist osanetant enhances memory consolidation in female mice whereas it impairs memory consolidation in males. These results are dependent on the modulation of sex hormones. Clinical studies showed promising effects of angiotensin II receptors blockade for reducing PTSD symptom severity. We will present pre-clinical studies regarding angiotensin receptors in the central amygdala and their role in fear extinction, specifically showing that inhibition of angiotensin receptor type 1 (AT₁Rs) facilitates fear extinction. Neuropeptide oxytocin has demonstrated a unique anxiolytic potential, but its role in the modulation of fear memory appears more complex and brain-site specific. We will show that oxytocin facilitates fear discrimination to predictable vs. unpredictable threats in rat fear-potentiated startle by inhibiting output neurons of the BNST. Using human imaging studies, we will also focus on identifying neural targets of intranasal oxytocin treatment and efficacious treatment strategies, including definition of the best dosage needed for target engagement in male and female volunteers. Finally, we will present data from human subjects showing that highly selective vasopressin 1A receptor antagonist SRX246 reduces anxiety but not fear-potentiated startle in male and female participants. This mini-symposium will address sex-specific effects of limbic neuropeptides in both rodents and humans and the importance of sex-specific pharmacotherapies for PTSD.

M29.01. Chair

A. Adhikari;

Psychology, University of California, Los Angeles, Los Angeles, CA.

Disclosures:

M29.02. Peptidergic pathways that transmit multimodal threat signals to the amygdala

Abstract: Thought-provoking talks by experts in the field will expose the audience to the study of threat-induced defensive behaviors using state-of-the-art methods in systems neuroscience, illustrating a wide range of threats and defensive behaviors.

S. Han;

PBL-H, Salk Institute For Biological Studies, La Jolla, CA.

Disclosures: S. Han: None.

M29.03. Hierarchical representations of aggression in a social behavior network

A. Falkner;

Princeton University, Princeton, NJ.

Disclosures:

M29.04. Role of insula to amygdala projection neurons in anxiety- and valence-related behaviors

A. Beyeler;

INSERM 1215, Bordeaux Cedex, FRANCE.

Disclosures:

M29.05. Prefrontal circuits underlying the emergence of persistent fearful memories

L. Denardo;

10920 WILSHIRE BOULEVARD, 5TH FLOOR, University of California, LOS ANGELES,, CA.

Disclosures:

M29.06. Hypothalamic control of escape from geometrically complex environments

A. Adhikari;

Psychology, UCLA, Los Angeles, CA.

Disclosures: A. Adhikari: None.

M29.07. Paraventricular nucleus CRH neurons encode stress controllability and regulate defensive behavior selection

N. Daviu;

Hotchkiss Brain Institute, University of Calgary, Calgary, AB, CANADA.

Disclosures:

Minisymposium

M30. Neurophysiology of Social Behaviors — From Information Gathering to Interaction

Location: Virtual Only

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

Description: This minisymposium will feature some of the latest findings on how neurons in frontal-subcortical circuits perform specialized functions for social behaviors across rodents and primates. The talks will examine different aspects of social behavior, including approach and defeat, social reward learning, decision simulation, social gaze interaction, and group behavior. They will also highlight new developments in innovative behavioral paradigms and neural technologies to address the unique complexity of social neuroscience.

Abstract: This session will feature latest findings on how neurons in frontal-subcortical circuits perform specialized functions for social behaviors across rodents and primates. The talks examine different aspects of social behavior, including approach and defeat, social reward learning, decision simulation, social gaze interaction, and group behavior. They also highlight new developments in innovative behavioral paradigms and neural technologies to address the unique complexity of social neuroscience. The neurobiology of social behaviors is a rapidly developing field. This symposium showcases some of the most exciting new data in the field across both primates and rodents. Together, the talks develop an emerging theme of how neurons in prefrontal cortex and connected subcortical structures fulfill specialized but complementary functions in social behaviors. The symposium also highlights recent advances in modeling naturalistic social behaviors with well-controlled neurophysiological paradigms.

M30.01. Chair

F. Grabenhorst;

Physiology, Development and Neuroscience, University of Cambridge, Cambridge, UNITED KINGDOM.

Disclosures:

M30.02. Co Chair

R. Baez-Mendoza;

Harvard Medical School, Boston, MA.

Disclosures:

M30.03. Neurobiology of sexually dimorphic social behavior

T. Kimchi;

Department of Neurobiology, Weizmann Institute of Science, Rehovot, ISRAEL.

Disclosures: T. Kimchi: None.

M30.04. Understanding the role of the medial prefrontal cortex in social reward-related behaviors.

M. Murugan;

Emory University, Atlanta, GA.

Disclosures: M. Murugan: None.

M30.05. Widespread social gaze signals in the primate prefrontal cortex during live social gaze interaction

S. W. Chang;

Yale University, New Haven, CT.

Disclosures: S.W. Chang: None.

M30.06. Social reward learning: basic mechanisms and therapeutic opportunities

G. Dölen;

Johns Hopkins University, Baltimore, MD.

Disclosures:

M30.07. Functions of primate amygdala neurons in decision-making and social decision simulation

F. Grabenhorst;

University of Cambridge, Cambridge, UNITED KINGDOM.

Disclosures: F. Grabenhorst: None.

M30.08. Neuronal representations of social identity and agency in groups of three interacting macaques

R. Baez-Mendoza;

Harvard Medical School, Boston, MA.

Disclosures: R. Baez-Mendoza: None.

Minisymposium

M31. Manifold Learning and Investigation of Neuronal Population Dynamics

Location: Virtual Only

Time: Monday, November 8, 2021, 10:45 AM - 11:45 AM

Description: Recent technological advances enable simultaneous recordings from large neuronal populations, opening up new possibilities for data-driven approaches to studying the neural code. Methods for dimensionality estimation and reduction help to identify neuronal dynamics that reside on lower-dimensional manifolds, and highlight key features of the computational process executed by the circuit. This minisymposium will discuss the prospects of such novel data analysis techniques to transform neuroscience research.

Abstract: Measuring neuronal tuning curves has been instrumental for many discoveries in neuroscience but requires a priori assumptions regarding the identity of the encoded variables.

We applied unsupervised learning to large-scale neuronal recordings in behaving mice from circuits involved in spatial cognition and uncovered a highly-organized internal structure of ensemble activity patterns. This emergent structure allowed defining for each neuron an ‘internal tuning-curve’ that characterizes its activity relative to the network activity, rather than relative to any predefined external variable, revealing place-tuning and head-direction tuning without relying on measurements of place or head-direction. Similar investigation in prefrontal cortex revealed schematic representations of distances and actions, and exposed a previously unknown variable, the ‘trajectory-phase’. The internal structure was conserved across mice, allowing using one animal’s data to decode another animal’s behavior. Thus, the internal structure of neuronal activity itself enables reconstructing internal representations and discovering new behavioral variables hidden within a neural code.

M31.01. Chair

A. Rubin;

Weizmann Institute of Science, Rehovot, ISRAEL.

Disclosures:

M31.02. Highly nonlinear manifolds emerge from simple neural population models

R. Chaudhuri;

University of California, Davis, Davis, CA.

Disclosures: R. Chaudhuri: None.

M31.03. Speaker

S. A. Solla;

Northwestern University, Chicago, IL.

Disclosures:

M31.04. The internal structure of neuronal codes for space

A. Rubin;

Weizmann Institute of Science, Rehovot, ISRAEL.

Disclosures: A. Rubin: None.

M31.05. Hierarchical network dynamics and behavior

Y. Bitterman;

Friedrich Miescher Institute For Biomedical Research, Basel, SWITZERLAND.

Disclosures:

M31.06. New methods for identifying latent manifold structure from neural data

A. Wu;

Center for Theoretical Neuroscience, Columbia University, new york, NY.

Disclosures: A. Wu: None.

M31.07. Grid cell modules are stable tori - a study of neural data with persistent cohomology

B. A. Dunn;

Mathematical Sciences, NTNU, Trondheim, NORWAY.

Disclosures: B.A. Dunn: None.

Minisymposium

M32. Posterior Cingulate Cortex — Progress in Anatomy and Physiology

Location: Virtual Only

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

Description: This minisymposium will highlight recent progress in understanding the organization and function of the posterior cingulate cortex (PCC). Speakers will discuss anatomical, electrophysiological and neuroimaging studies of the PCC in humans, non-human primates and rodents. Despite a growing interest in PCC function from multiple fields such as episodic memory, spatial navigation and cognitive control, a consensus is severely lacking. Speakers will address this issue with a comparative focus.

Support: NIH R01MH118257; NARSAD.
NIH R01DA038615.
Wellcome Trust.
NIH R00MH103479; R01MH116914.
Sloan Research Fellowship; Google Faculty Research Award.

Abstract: Despite convergent evidence highlighting the posterior cingulate cortex (PCC) as an anatomically and physiologically unique brain structure, it has generally been neglected by cognitive and systems neuroscience. This is evidenced by PCC being implicated in a host of cognitive operations, such as emotion, episodic memory, spatial navigation and cognitive control, with little consensus as to its core functional role. Furthermore, extant literature is highly variable in anatomically identifying the PCC and distinguishing it from related structures such as the retrosplenial cortex and precuneus. A clear understanding of the homology of PCC and its neighboring regions across species is consequently lacking. In short, the basic functional neuroanatomy of the PCC has yet to be established. To make progress on this front, improvements must be made in establishing a clear nomenclature of the PCC and surrounding anatomy, with a concern for comparative study. In turn, functional studies must seek to understand the potentially multiple functions of the PCC and their anatomical arrangement, rather than trying to isolate a singular function for this expansive region. In this minisymposium, speakers will review recent efforts towards these goals, highlighting progress in understanding

the anatomical and physiological properties of the PCC. Importantly, speakers will present data from human, non-human primate and rodent experiments with consideration towards comparative homologies (anatomical & functional). Specifically, speakers will address: i) multimodal, cross-species efforts to characterize the connectivity of PCC subregions; ii) electrophysiological studies of non-human primate PCC in economic choice and its interactions with orbitofrontal cortex; iii) comparative electrophysiology of spatial coding in non-human primate and rodent PCC regions; iv) systematic investigations of the sulcal patterning within PCC, which includes the observation of a previously unidentified sulcus displaying microanatomical, macroanatomical, and functional features that may serve as an important landmark within human PCC; v) invasive electrophysiological studies of human PCC, including single unit recordings, which reveal an executive and mnemonic functional role; vi) functional MRI studies of human recollection, using naturalistic movies and speech, which examine representations in PCC that are reactivated between encoding and retrieval, as well as aligned across individuals. Together, these presentations will provide an important update on structure-function relationships within PCC, making it of interest to a broad range of neuroscientists.

M32.01. Chair

B. L. Foster;

Department of Neurosurgery, Baylor College of Medicine, Houston, TX.

Disclosures:

M32.02. Brain activity patterns shared across people during memory for continuous natural events

J. Chen;

Psychological and Brain Sciences, Johns Hopkins University, Baltimore, MD.

Disclosures: J. Chen: None.

M32.03. Cognitive electrophysiology of the human PCC

B. L. Foster;

1 Baylor Plaza, BCM250, University of Pennsylvania, Philadelphia, PA.

Disclosures: B.L. Foster: None.

M32.05. Role of PCC in economic choice and prospection

B. Y. Hayden;

University of Minnesota Twin Cities, Minneapolis, MN.

Disclosures: B.Y. Hayden: None.

M32.06. Comparative connectivity of the PCC: Anatomy meets neuroimaging

S. R. Heilbronner;

Neuroscience, University of Minnesota, Minneapolis, MN.

Disclosures: S.R. Heilbronner: None.

M32.07. A new tripartite landmark in human posterior cingulate cortex

K. S. Weiner;

Psychology, University of California, Berkeley, Berkeley, CA.

Disclosures: K.S. Weiner: None.

Minisymposium

M33. Systems Neuroscience of Natural Behaviors in Rodents

Location: Virtual Only

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

Description: Animals have evolved in complex environments, producing a wide range of behaviors including navigation, foraging, prey capture, and social interactions. This **minisymposium** will highlight how recent advances in neural recordings, machine learning, and theoretical frameworks provide insight into the mechanisms of naturalistic behaviors. Speakers will demonstrate how ethology, ecology, and systems neuroscience can benefit from each other and how viewing individual behaviors as part of a whole increases understanding.

Support: Helen Hay Whitney Foundation; Hanna H Gray Fellowship
Howard Hughes Medical Institute
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NIH R01 EY023337 and 2T32HD007348-26
Humboldt-Universität zu Berlin
BCCN Berlin
DFG (BR 3479/11-1)

Abstract: The power of systems neuroscience techniques previously necessitated highly trained, high trial-count behaviors that repeat on the order of seconds. Yet animals evolved in complex environments, producing a wide range of behaviors over timescales ranging from milliseconds to days. Thanks to advances over the last few years in machine learning, miniaturization, and computation, it is now possible to study complex behaviors in more natural conditions while recording movement, performing temporally-specific perturbations, and recording from large numbers of neurons. The speakers in our minisymposium are at the forefront of using these techniques applied to newly available problems in rodents.

Bob Datta will highlight how, inspired by ethology, his group is developing and using Motion Sequencing to perform automatic analysis of spontaneous behaviors breaking up complex actions into “syllables” and using the “grammar” of behavior to understand the neural mechanisms that translate sensory inputs into behavioral outputs.

Jakob Voigts will show how spatial modalities such as vision, self-motion, knowledge of the environment, and position are related to each other in single neurons and at the population level in freely behaving mice.

Ahmed El Hady will present a theoretical framework that can be used to design experiments aimed at study the mechanistic underpinnings of patch foraging decisions.

Angie Michael will demonstrate how using naturalistic prey capture assays provide unique insights into visual processing like sampling and gaze stabilization.

Gowan Tervo will discuss his work studying the audience effect in rodents: how seeing others influences performance.

Ann Clemens uses intra- and juxta-cellular recordings during kinship behaviors to unveil the exquisite cellular organization of social sensory processing.

The chair, Emily Dennis, will frame the work by discussing the evolutionary and ecological contexts of these behaviors that lack traditional trial-based structures and highlight the ways each of our speakers leverage the power of systems level tools and analyses.

Together, we will demonstrate how ethology, ecology, and systems neuroscience can benefit from each other, and that viewing individual behaviors as part of a whole increases understanding.

M33.01. Chair

E. J. Dennis;

Neuroscience, Princeton University, West Windsor, NJ.

Disclosures:

M33.02. Dopamine specifies the statistics of spontaneous behavior

J. E. Markowitz;

Department of Neurobiology, Harvard Medical School, Boston, MA.

Disclosures: J.E. Markowitz: None.

M33.03. Mechanistic theory of patch foraging

A. El Hady;

Princeton Neuroscience Institute, Princeton, NJ.

Disclosures:

M33.04. Neural mechanisms for reasoning about and navigating large environments

J. Voigts;

Brain and Cog. Sci., MIT, Cambridge, MA.

Disclosures: J. Voigts: F. Consulting Fees (e.g., advisory boards); Co-founder & board member of Open Ephys, I do not receive any salary or fees for this role.

M33.05. Neural mechanisms of kinship behavior

A. Clemens;

University of Edinburgh, Edinburgh, UNITED KINGDOM.

Disclosures: A. Clemens: None.

M33.06. The audience effect: neural circuits of social facilitation in rodents

D. G. Tervo;

Howard Hughes Medical Institute Janelia Farm Research Campus, Ashburn, VA.

Disclosures:

M33.07. An open-source, high-throughput, semi-automated monitoring system for maternal behavior in mice

L. Pinerros Schuster;

New York University, New York City, NY.

Disclosures: L. Pinerros Schuster: None.

Minisymposium

M34. The Ontogeny of Hippocampus-Dependent Memories

Location: Virtual Only

Time: Thursday, November 11, 2021, 12:00 PM - 1:00 PM

Description: It has long been debated whether infants can make the types of memories that in adults are processed by the hippocampus-dependent memory system, and whether the hippocampus is involved in memory processes at this age. Recent evidence has revealed that the infant hippocampus can produce long-lasting memories, but that its ability to support memory processes matures with age. This minisymposium will present recent work to unravel the ontogeny and biological correlates of hippocampus-dependent memory development.

Support: R01NS104917 grant from the National Institute of Neurological Disorders and Stroke (NINDS) of NIH
R01MH121372 grant from the National Institute of Mental Health (NIMH) of NIH
MH106809
MH091844
MH065635
NIH F31HD090872
ERC-2019-STG Project 850769

M34.01. Chair

F. Donato;

University of Basel, Basel, SWITZERLAND.

Disclosures:

M34.02. Molecular mechanisms of long-term memory formation in the developing rodent hippocampus

C. M. Alberini;

Center for Neural Science, New York University, New York, NY.

Disclosures: C.M. Alberini: None.

M34.03. Co Chair

C. M. Alberini;

Center for Neural Science, New York University, New York, NY.

Disclosures:

M34.04. Early life events dictate hippocampal structure and function

A. Dranovsky;

Psychiatry, Columbia University, New York, NY.

Disclosures: A. Dranovsky: None.

M34.05. Children's memory for specific events: before or after semantic generalizations

N. S. Newcombe;

Temple University, Philadelphia, PA.

Disclosures:

M34.06. Developmental emergence of network dynamics supporting the cognitive map

F. Donato;

University of Basel, Basel, SWITZERLAND.

Disclosures: F. Donato: None.

M34.07. Ontogeny of preconfigured and replay sequences in the hippocampus

G. Dragoi;

Psychiatry, Yale University, New Haven, CT.

Disclosures: G. Dragoi: None.

M34.08. Spatial memory in human infancy: eye tracking evidence from contextual cueing

D. Amso;

Brown University, Providence, RI.

Disclosures: D. Amso: None.

Minisymposium

M35. Advancing Gene Therapy for Central Nervous System (CNS) Diseases

Location: Virtual Only

Time: Thursday, November 11, 2021, 12:00 PM - 1:00 PM

Description: Adeno-Associated Viral (AAV) vectors or genetically modified hematopoietic stem cells deliver gene products into cells and organisms and have broad implications for novel therapeutics. With technologies that can cross the blood-brain barrier or safely deliver therapeutics directly to the CNS, treating neurological diseases is now becoming a reality. This minisymposium will provide a broad perspective on gene therapy, including discussions of technological advancements to treat CNS diseases.

M35.01. Chair

D. Sliter;

Research and Early Development, REGENXBIO Inc., Rockville, MD.

Disclosures:

M35.02. Co Chair

J. Smith;

Regenxbio, Rockville, MD.

Disclosures:

M35.03. Preclinical gene therapy for neurologic aspects of TSC1 and TSC2

X. O. Breakefield;

Molecular Neurogenetics Unit, Harvard University / Massachusetts Gen Hospital, Charlestown, MA.

Disclosures: X.O. Breakefield: None.

M35.05. AAV gene therapy approaches for the CNS

N. Cartier;

INSERM, Paris, FRANCE.

Disclosures:

M35.06. Chloride homeostasis as gene therapy target to rescues cognitive deficits in a mouse model of down syndrome

A. Contestabile;

Fondazione Istituto Italiano di Tecnologia, Genova, ITALY.

Disclosures: **A. Contestabile:** E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Co-inventor on granted patent US 9,822,368; EP 3083959; JP 6490077. Co-inventor on the patent application WO 2018/189225 and IT 10201900000.

M35.07. Targeting neurodegenerative lysosomal storage diseases with gene therapy

M. Sena-Esteves;

University of Massachusetts Medical School, Worcester, MA.

Disclosures:

Minisymposium

M36. Emerging Applications and Methodologies for Neural Interrogation

Location: Virtual Only

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

Description: The future of neural interfaces will leverage multi-modal approaches based on emergent optical, biological, and physical principles for neural interrogation. These methodologies for multi-scale recording and mediation of neural activity will enable new applications for investigating neural function and developing therapies. Focusing on emergent methods for neural interrogation, this minisymposium will present innovative scientific and therapeutic applications of novel approaches to interface with the brain.

Abstract: Neuropsychiatric disorders, such as anxiety and depression, are network-wide disorders that affect multiple brain areas in frontolimbic and frontostriatal circuitries. Electrical stimulation has been hypothesized as a potential treatment for these conditions, but the optimal neural target and paradigm remain elusive. We posit that it's critical to take a systems-level approach to interrogate neural activity in order to develop an understanding of neural dysfunction throughout relevant neural circuitry and intervention strategies. In these work, we record neural activity in multiple brain areas in a nonhuman primate (NHP) animal model and deliver electrical stimulation in prefrontal cortex. In order to demonstrate a functional link between mood state and neural state, we used a pharmacological approach to create a proxy for an acute anxiety animal model in the NHP model. Two NHP subjects were used in this study and experimental sessions were divided into three blocks. During the first block, baseline physiology and behavior was established. At the beginning of the second block, beta-carbolines, a type of benzodiazepine inverse agonist, were administered to induce an acute anxiety-like state. Finally, in the third block a treatment was administered to the subjects in the form of either closed-loop stimulation or midazolam, a known anxiolytic drug in the class of benzodiazepines. Using biomarkers of mood state changes discovered during this behavior, we designed closed-loop microstimulation protocols to mediate these changes and restore the subjects to a baseline, non-anxious state. We applied unilateral microstimulation to prefrontal cortical sites to modulate the response evoked from the beta-carbolines administered in the second block. We found that

microstimulation was able to restore neural features across the neural circuitry to baseline levels and generate a profound anxiolytic physiological response.

M36.01. Chair

S. R. Santacruz;

Biomedical Engineering, University of Texas at Austin, Austin, TX.

Disclosures:

M36.02. Co Chair

L. Luan;

Electrical and Computer Engineering; Bioengineering, Rice University, Houston, TX.

Disclosures:

M36.03. Multimodal mapping of neural activity and cerebral blood flow reveals long-lasting neurovascular dissociations after small-scale strokes

L. Luan;

Electrical and Computer Engineering; Bioengineering, Rice University, Houston, TX.

Disclosures: L. Luan: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); L.L. holds equity ownership in Neuralthread Inc., an entity that is licensing ultraflexible neural electrode technology described herein.

M36.04. Multi-scale neural circuit responses to closed-loop microstimulation in acute anxiety monkey model

S. R. Santacruz;

Biomedical Engineering, University of Texas At Austin, Austin, TX.

Disclosures:

M36.05. Restoration of voluntary motor control of arm movements with electrical stimulation of the primary sensory afferents in monkeys

M. Capogrosso;

Neurological Surgery, University of Pittsburgh, Pittsburgh, PA.

Disclosures:

M36.06. Decoding the reorganization of cortical representation of tactile shape across learning with volumetric calcium imaging in mice

S. A. Hires;

Biological Sciences, University of Southern California, Los Angeles, CA.

Disclosures:

M36.07. Next generation tracking microscopy for whole brain imaging of internal states

J. Y. Li;

Max Planck Institute for Biological Cybernetics, Tuebingen, GERMANY.

Disclosures:

M36.08. An intravenous light source for minimally invasive optogenetics

G. Hong;

Materials Science and Engineering, Stanford University, Stanford, CA.

Disclosures: G. Hong: None.

Minisymposium**M37. Emerging Computational Research for Neurological and Psychiatric Disorders**

Location: Virtual Only

Time: Tuesday, November 9, 2021, 12:00 PM - 1:00 PM

Description: This minisymposium will discuss the breadth of computational resources and methodologies used to understand the complex heterogeneity of multiple neurological and psychiatric disorders. Speakers will discuss the availability of big data sets for computational research across these disorders as well as the current state of machine learning and statistical methods used for hypothesis generation. They will also identify novel computational models aiding in clinical decision support, improving diagnosis and treatment planning.

Abstract: Data-intensive research is a hot topic revolutionizing how science is done, and it has been considered the ‘fourth paradigm’ of research. With the wealth of available datasets across multiple disorders impacting neurological and psychological health, and the emergence of innovative computational modeling approaches, we are entering an exciting era for understanding how to more effectively diagnose and treat a broad range of brain-based disorders. This mini symposium will highlight several use cases of novel modeling approaches applied to neurological and psychiatric disorders applied to multiple fields of research where multi-modal health data are used. Our presentations will provide a data-intensive framework for leveraging these tools to enhance how to use biomedical data to improve human health. We will provide practical examples of how computational modeling can aid clinical decision support for physicians and clinical researchers who may be inundated with multi-modal data. By demonstrating how various computational tools can help identify novel hypotheses about disease presentation, progression and treatment trajectories, we will provide clinicians and researchers with a platform for making data-driven decisions in healthcare settings, clinical trial design, and treatment planning. With the raised concerns on the lack of reproducibility in research, new approaches and research models such as data-intensive research can tackle part of the problem. Beyond the already exposed reasons, this mini symposium can serve as an amplifier of these new

forms of thinking for all the neuroscience community, but especially to young researchers, and provide an educational forum that hopefully will contribute to improved research.

Abstract: Data-intensive research is a hot topic revolutionizing how science is done, and it has been considered the ‘fourth paradigm’ of research. With the wealth of available datasets across multiple disorders impacting neurological and psychological health, and the emergence of innovative computational modeling approaches, we are entering an exciting era for understanding how to more effectively diagnose and treat a broad range of brain-based disorders. This mini symposium will highlight several use cases of novel modeling approaches applied to neurological and psychiatric disorders applied to multiple fields of research where multi-modal health data are used. Our presentations will provide a data-intensive framework for leveraging these tools to enhance how to use biomedical data to improve human health. We will provide practical examples of how computational modeling can aid clinical decision support for physicians and clinical researchers who may be inundated with multi-modal data. By demonstrating how various computational tools can help identify novel hypotheses about disease presentation, progression and treatment trajectories, we will provide clinicians and researchers with a platform for making data-driven decisions in healthcare settings, clinical trial design, and treatment planning. With the raised concerns on the lack of reproducibility in research, new approaches and research models such as data-intensive research can tackle part of the problem. Beyond the already exposed reasons, this mini symposium can serve as an amplifier of these new forms of thinking for all the neuroscience community, but especially to young researchers, and provide an educational forum that hopefully will contribute to improved research.

M37.01. Chair

J. L. Nielson;

Psychiatry, University of Minnesota, Minneapolis, MN.

Disclosures:

M37.02. Co Chair

J. Huie;

Brain and Spinal Injury Center, University of California, San Francisco, San Francisco, CA.

Disclosures:

M37.03. Methods for bridging the gap between causal questions and modern neuroscience data

E. Kummerfeld;

University of Minnesota, Minneapolis, MN.

Disclosures:

M37.04. Overview of machine learning methods with applications in neuroscience research

S. Ma;

University of Minnesota, Minneapolis, MN.

Disclosures: S. Ma: None.

M37.05. Machine learning approaches identify independence of substance use trajectories from neuropsychiatric symptoms across multiple diagnostic categories.

J. L. Nielson;

Psychiatry, University of Minnesota, Minneapolis, MN.

Disclosures: J.L. Nielson: A. Employment/Salary (full or part-time); University of Minnesota. 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.; R01 MH116156.

M37.06. Machine intelligence reveals precise blood pressure associated with recovery in acute spinal cord injury

A. Torres-Espín;

University of California San Francisco, San Francisco, CA.

Disclosures:

M37.07. Multidimensional approaches for biomarker discovery in neurotrauma

J. Huie;

Brain and Spinal Injury Center, UCSF, San Francisco, CA.

Disclosures:

M37.08. Machine intelligence for translational neuroscience

A. R. Ferguson;

Department of Neurological Surgery, Brain and Spinal Injury Center (BASIC), UCSF, San Francisco, CA.

Disclosures:

Minisymposium

M38. Highlights From the Era of Open Source Online Tools — From Genes to Neurons, Circuits, Behaviors, and Whole Brain Data

Location: Virtual Only

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

Description: Open source science is a rapidly growing movement to address the reproducibility crisis and spearhead efficient data sharing. The movement's momentum has resulted in the recent emergence of many online data sharing repositories. These tools are changing the landscape and culture of neuroscience by maximizing data visibility and impact. In this minisymposium,

speakers will highlight different online resources that span across multiple neuroscience sub-disciplines and discuss how these tools can propel research forward.

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U01MH114829
R01NS86082
R01MH111918
T32MH015174
U19MH114830
R01AG047589

Abstract: High digital connectivity and low scientific reproducibility has led to a new era in neuroscience. Open data sharing is a widespread movement reaching virtually all areas of neuroscience. This symposium highlights six free resources, spanning from genes to neurons, circuits, behaviors and whole brain data. GeneNetwork.org hosts genome and phenome quantitative data related to neurobiology and behavior with matched genetics and expression data for mouse, rat, and human. An overview will be provided for how to use GeneNetwork.org to probe causal mechanisms that link *Genes X & Y* with *Phenotypes A & B*, and vice versa. NeuroMorpho.Org provides unhindered access to >130k digital tracings of neurons and glia from a wide diversity of animal species, anatomical regions, and experimental methods, contributed by hundreds of laboratories worldwide. Users will be introduced to secondary analyses of this data, which to-date have resulted in nearly 2,000 peer-reviewed publications. Bridging single neuron structure to the mesoscale connectome, the Allen Mouse Brain Connectivity Atlas project has generated a comprehensive and quantitative description of brain-wide wiring patterns at the level of cell classes. The atlas currently consists of ~3,000 viral-mediated anterograde tracer experiments, including ~2,300 performed in Cre driver lines. All data is integrated into the Allen Mouse Brain Common Coordinate Framework and openly accessible through brain-map.org. Moving from structural to functional connectivity, MouseCircuits.org offers an interactive platform for consolidated circuit data generated by the research community using pathway-specific chemogenetic and/or optogenetic manipulations. Similarly, MouseBytes.ca is a web-based repository that enables researchers to store, share, visualize, and analyze cognitive data from automated tests based on touchscreen technology. WholeBrainSoftware.org enables atlas registration and visualization of anatomical data from single-molecule to mesoscale level across a diverse set of modalities. Together, these tools maximize scientific output, accelerate progress and enhance reproducibility.

Learning objectives: 1. Understand the driving forces leading to open source science and data sharing through the lens of the reproducibility crisis; 2. Take advantage of six contemporary free online tools spanning data from genes, to neurons, circuits, behavior and whole brain mapping; 3. Be inspired to promote open science and spearhead additional resources to accelerate collaborative discoveries

M38.01. Chair

D. Dumitriu;
Columbia University, New York, NY.

Disclosures:

M38.02. Mousecircuits.org: an online repository to guide the rodent circuit dissection era

K. R. Anderson;

Columbia University, New York, NY.

Disclosures: K.R. Anderson: None.

M38.03. From single molecule to mesoscale structures: using WebGL and WebAssembly for handling next-generation of neuroanatomical data

D. Fürth;

Cold Spring Harbor Laboratories, Cold Spring Harbor, NY.

Disclosures:

M38.04. Scaling up the morphological characterization of neuron types

G. A. Ascoli;

George Mason University, Fairfax, VA.

Disclosures: G.A. Ascoli: None.

M38.05. Speaker

L. Ng;

Allen Institute for Brain Science, Alexandria, VA.

Disclosures:

M38.06. Mousebytes: an open-access and web-based repository for cognitive data integration and sharing

S. Memar;

Department of BrainsCAN, The University of Western Ontario, London, ON, CANADA.

Disclosures: S. Memar: None.

M38.07. GeneNetwork.org: genetic analysis for all neuroscientists

D. G. Ashbrook;

Department of Genetics, Genomics and Informatics, The University of Tennessee Health Science Center, Memphis, TN.

Disclosures: D.G. Ashbrook: None.

Minisymposium

M39. Patch-seq — Everything You Always Wanted to Know, But Were Afraid to Ask

Location: Virtual Only

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

Description: Single cell transcriptomic approaches are revolutionising neuroscience and are most powerful when multiplexed with complementary techniques. By combining electrophysiology with mRNA sequencing at an individual neuron level, technically-demanding Patch-seq experiments are providing unparalleled insight into the links between gene expression and neuronal function. This minisymposium will showcase recent Patch-seq-enabled discoveries and share practical expertise that makes these experiments successful.

Abstract: Single-cell transcriptomic approaches are revolutionising neuroscience, and are most powerful when multiplexed with complementary techniques. By combining electrophysiology with mRNA sequencing at the individual-neuron level, technically-demanding “Patch-seq” experiments are starting to provide unparalleled insight into the links between gene expression and neuronal function. Here, six speakers at the coalface of Patch-seq technology will showcase their recent discoveries whilst sharing the practical expertise that makes their experiments so successful. Though single-cell transcriptomic studies are becoming widespread, techniques such as Patch-seq that combine multiple levels of investigation in individual neurons are still in their infancy. The unique practical and theoretical demands of this approach have just started to be surmounted, with only a few laboratories world-wide making a success of Patch-seq experiments. The time is now absolutely right to bring this novel expertise to a wider audience, to share insight that will drive this fledgling field forwards. Our session has three specific learning objectives. First, attendees will gain an appreciation of the power of combining multiple levels of analysis, including transcriptomics, at the single-neuron level. Second, we will share insider tips and tricks, both practical and analytical, for obtaining high-quality Patch-seq data. Lastly, we will discuss state-of-the-art knowledge from recent, unpublished work uncovering the relationships between gene expression and neuronal function

M39.01. Chair

S. Tripathy;

Psychiatry, University of Toronto, Toronto, ON, CANADA.

Disclosures:

M39.02. Co Chair

M. Lipovsek;

King's College London, London, UNITED KINGDOM.

Disclosures:

M39.03. Mapping patch-seq cells to reference datasets

D. Kobak;

Institute for Ophthalmic Research, Tübingen University, Tübingen, GERMANY.

Disclosures:

M39.04. Scaling up patch-seq: keys to maximizing triple modality data

K. Hadley;

Allen Institute for Brain Science, Seattle, WA.

Disclosures: K. Hadley: None.

M39.05. Assessing transcriptome quality in patch-seq datasets

S. Tripathy;

Psychiatry, University of Toronto, Toronto, ON, CANADA.

Disclosures: S. Tripathy: None.

M39.06. Cell type composition and circuit organization of clonally related excitatory neurons in the mouse neocortex

C. R. Cadwell;

Neuroscience, UCSF, San Francisco, CA.

Disclosures: C.R. Cadwell: None.

M39.07. Patch-seq of dopaminergic neurons: linking functional and transcriptional plasticity

M. Lipovsek;

Centre for Developmental Neurobiology, Kings College London, London, UNITED KINGDOM.

Disclosures: M. Lipovsek: None.

M39.08. Patch-seq of human neurons derived from induced pluripotent stem cells.

C. Bardy;

South Australian Health and Medical Research Institute, Flinders University, Adelaide, AUSTRALIA.

Disclosures: C. Bardy: None.

Minisymposium

M40. Spatial Transcriptomics Methods for Cell Type Identification in the Central Nervous System (CNS) — Methods and Analysis

Location: Virtual Only

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

Description: A comprehensive census of transcriptomic cell types present in the central nervous system is only possible by preserving tissue architecture and assaying these cells *in situ*. Spatial transcriptomics methods make this possible by reporting on the number and location of cell types in intact tissue. This minisymposium will serve as a forum for discussing data generation, processing, and analysis methods for these emerging techniques and provide a platform for sharing results and exploring the potential of these methods.

M40.01. Chair

J. L. Close;

Human Cell Types, Allen Institute for Brain Science, Seattle, WA.

Disclosures:

M40.02. Co Chair

B. Long;

Imaging, Allen Institute for Brain Science, Seattle, WA.

Disclosures:

M40.04. Segmentation-free inference of cell types from *in situ* transcriptomics data

N. Ishaque;

Berlin Institute for Health at Charité, Berlin, GERMANY.

Disclosures: N. Ishaque: None.

M40.05. Cell type characterization with multiplexed FISH in developing human brain

L. E. Borm;

615 Westlake Ave. N, Karolinska Institute, Stockholm, SWEDEN.

Disclosures: L.E. Borm: None.

M40.06. Merfish-based characterization of mouse primary motor cortex

M. Zhang;

12 Oxford Street Zhuang group, 034 Naito Building, Harvard University, Cambridge, MA.

Disclosures: M. Zhang: None.

M40.07. In situ sequencing for identification of cell types in mouse hippocampus

X. Qian;

Cartana, Stockholm, SWEDEN.

Disclosures:

Symposium

S01. The Neuronal Cytoskeleton — Nexus for Signaling, Development, and Plasticity

Location: Virtual Only

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

Description: This symposium will showcase the cellular infrastructure that supports neuronal development, stability and plasticity. The neuronal cytoskeleton is exquisitely regulated to balance long-term structural maintenance with the ability to make and break connections and repair damage. Speakers will address key questions about cytoskeletal regulation in development, signaling, disease and injury using multiple experimental approaches in vertebrate and invertebrate neurons.

Support: MMR: National Institutes of Health, USA, R01GM085115
TMG: National Institutes of Health, USA, R01NS099405
AK: National Institutes of Health, USA, R01NS105640
AK: National Institutes of Health, USA, R01MH116939
AK: Falk Trust, Amgen Foundation and Simons Foundation, USA
Broad Institute, USA, 6001032-5500001216
AK: Howard Hughes Medical Institute, USA, GR101933

Abstract: The neuronal cytoskeleton forms the basis of neuronal structure and transport, and is foundational to long-term neuronal function. Symposium speakers will highlight current research on the functions of the cytoskeleton in neuronal development, including axon pathfinding and establishment of polarity. The role of the cytoskeleton in critical functions in mature neurons, including signaling and injury responses will also be included. Vertebrate and invertebrate model systems will be introduced, and in vitro and in vivo approaches will be included to give a comprehensive overview of how our understanding of neuronal cell biology is being advanced.

S01.01. Chair

M. Rolls;
Pennsylvania State University, University Park, PA.

Disclosures:

S01.02. Co Chair

A. J. Koleske;
Yale University, New Haven, CT.

Disclosures:

S01.03. Controlling cytoskeleton remodeling during neuronal development

C. Hoogenraad;
Genentech, Inc., South San Francisco, CA.

Disclosures: C. Hoogenraad: None.

S01.04. Abnormal axon guidance of human tuberous sclerosis neurons is due to mTOR-independent defects in regulation of the cytoskeleton.

T. M. Gomez;

Dept of Neuroscience, University of Wisconsin Madison School of Medicine and Public Health, Madison, WI.

Disclosures: T.M. Gomez: None.

S01.05. Regulation of microtubule growth is essential for successful responses to axon and dendrite injury.

M. Rolls;

Penn State, University Park, PA.

Disclosures: M. Rolls: None.

S01.06. Disruption of cytoskeletal control mechanisms in neurodevelopmental disorders

A. Koleske;

Molecular Biophysics and Biochemistry, Yale University, New Haven, CT.

Disclosures: A. Koleske: F. Consulting Fees (e.g., advisory boards); Scipher Medicine.

Symposium

S02. Beyond the Textbook — Novel Perspectives in Axonal Function and Plasticity

Location: Virtual Only

Time: Monday, November 8, 2021, 10:45 AM - 11:45 AM

Description: When SfN formed 50 years ago, it was already known that axons propagate information in the form of action potentials across the nervous system. Although axonal impairment has long been known to be devastating for brain function in disease, until recently it was thought that axons showed minimal plasticity in healthy brains. This symposium will highlight recent advances in mechanisms controlling axonal function, how these mechanisms are plastic, and how axonal plasticity impacts brain function.

Support: Operating Grant (AJW, 130570), and Project Grant (AJW 153150), Canadian Institutes of Health Sciences
Discovery Grant (AJW, 05118), National Science and Engineering Research Council of Canada
Vici grant (MK, 865.17.003), Netherlands Organization for Scientific Research (NWO)
FUNCOPLAN (MG, 725729), H2020 ERC Consolidator Grant
Sir Henry Wellcome Post-doctoral Fellowship (EG, 103044), Wellcome Trust

S02.01. Chair

A. J. Watt;

Biology, McGill University, Montreal, QC, CANADA.

Disclosures:

S02.02. Co Chair

M. Engelhardt;

Institut of Anatomy and Cell Biology, Medical Faculty, Johannes Kepler University, Linz, AUSTRIA.

Disclosures: M. Engelhardt: None.

S02.03. Cell type-specific plasticity of the axon initial segment in vivo

M. S. Grubb;

King's College London, London, UNITED KINGDOM.

Disclosures: M.S. Grubb: None.

S02.04. Purkinje cell axonal swellings enhance action potential fidelity and cerebellar function

A. J. Watt;

Biology, McGill University, Montreal, QC, CANADA.

Disclosures:

S02.05. A role for the axon initial segment in homeostatic scaling of neuronal excitability

M. Engelhardt;

Institut of Anatomy and Cell Biology, Medical Faculty, Johannes Kepler University, Linz, AUSTRIA.

Disclosures: M. Engelhardt: None.

S02.06. Compartmentalized recurrent action potential initiation in human layer 5 pyramidal neurons

M. Kole;

Biophysics of Complex Cellular Systems, Netherlands Institute for Neuroscience, Amsterdam, NETHERLANDS.

Disclosures: M. Kole: None.

Symposium

S03. Liquid-Liquid Phase Separation in Physiology and Pathophysiology of Nervous System

Location: Virtual Only

Time: Monday, November 8, 2021, 9:30 AM - 10:30 AM

Description: Molecules within cells are segregated into functional domains to form various organelles. While some of these organelles are delimited by lipid membranes demarcating their constituents, others lack any membrane. Recently, liquid-liquid phase separation (LLPS) has revolutionized our view of how segregation of macromolecules can form membrane-less organelles. This symposium will present data showing what role LLPS plays and how it can change views of the physiology and pathophysiology of the nervous system.

Abstract: Molecules within cells are segregated into functional domains to form various organelles. While some are delimited by lipid membranes demarcating their constituents, others lack any membrane. Recently, liquid-liquid phase separation (LLPS) revolutionized our view of how segregation of macromolecules can form membrane-less organelles. While the concept of LLPS has been well-studied in the area of soft matter physics, its significance has only recently been recognized in the field of biology. LLPS in biological context occurs typically between macromolecules such as protein or RNA that have multivalent interactions and/or disordered regions. Interestingly these features are present in multiple molecules that exert key physiological functions within neurons. Not just within physiological context, the accumulation of abnormal proteins is also implicated in the pathogenesis of neurodegenerative diseases such as Alzheimer's disease, amyotrophic lateral sclerosis, and Parkinson's disease. Here as well, LLPS is most likely involved in the accumulation of these pathogenic proteins and can be a therapeutic target of drugs, as well. In this symposium, we will present our data showing what role LLPS plays and how it can change our view of physiology and pathophysiology of the nervous system. Zhang will talk about phase separation in synapse formation and function. Hayashi will talk how CaMKII-mediated phase-in-phase separation can subsynaptically segregate glutamate receptors. Ford will talk about the role of phase separation in tau physiology and pathology in consideration of non-stress granule organelles. Finally, McGurk will talk about phase separation of TDP-43 in motor neuron disease and dementia.

S03.01. Chair

Y. Hayashi;

Department of Pharmacology, Kyoto University Graduate School of Medicine, Kyoto-Shi, JAPAN.

Disclosures:

S03.02. Co Chair

M. Zhang;

Hong Kong University of Science and Technology, Kowloon, HONG KONG.

Disclosures:

S03.03. Physiological and pathological phase separation of Cytoplasmic polyadenylation element binding protein 3 in neurons

L. Fioriti;

Istituto di Ricerche Farmacologiche Mario Negri, MILANO, ITALY.

Disclosures: L. Fioriti: None.

S03.04. The role of phase separation in tau physiology and pathology: a consideration of non-stress granule organelles

L. Ford;

University of California, Santa Barbara, Santa Barbara, CA.

Disclosures: L. Ford: None.

S03.05. Phase separation in synapse formation and function

M. Zhang;

Hong Kong University of Science and Technology, Hong Kong, HONG KONG.

Disclosures:

S03.06. Subsynaptic segregation of glutamate receptors by CaMKII-mediated phase-in-phase separation

Y. Hayashi;

Department of Pharmacology, Kyoto University Graduate School of Medicine, Kyoto-Shi, JAPAN.

Disclosures: Y. Hayashi: None.

Symposium

S04. New Insights Into the Neurobiology of Repeat Expansion Diseases

Location: Virtual Only

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

Description: Over 40 neurological diseases are caused by repeat expansions, including Huntington's disease, motor neuron disease, frontotemporal dementia and ataxia. Advances in genetics have outpaced our understanding of the underlying neurobiology, leading to a current lack of therapies for these fatal diseases. This symposium will review new insights into how expanded repeats perturb multi-level function of the nervous system and discuss how emerging knowledge is driving creative and novel treatment strategies.

Support: R01NS038712-17 (Paulson)

R01NS096785 (Paulson)
U01NS106670 (Paulson)
R37NS027699 (Zoghbi)
R37NS040389 (Ranum)
R01NS098819 (Ranum)
R01NS085654 (Shakkottai)

Abstract: Over 40 neurological diseases are caused by repeat expansions including Huntingtondisease, amyotrophic lateral sclerosis (ALS), frontotemporal dementia (FTD) and many forms of ataxia. Advances in the genetics of repeatexpansion disease have outpaced our understanding of the underlyingneurobiology, leading to the current lack of therapies for these fatal diseases. Leadingexperts in the field will review new insights into how expanded repeats perturbmulti-level function of the nervous system and discuss how emerging knowledgeis driving creative and novel treatment strategies. We will begin with a briefoverview highlighting shared biological concepts across the repeat expansion diseases.Full presentations will center on the molecular mechanisms underlying the mostcommon cause of FTD/ALS (the C9ORF72 repeat expansion), the basis of selective neuronal vulnerability inHuntington disease, the shared role of ion channel dysfunction in a wide rangeof repeat expansion ataxias including the spinocerebellar ataxias (SCAs), and thecomplex neurobiology driving selective neuronal loss in the polyglutamine disorderSCA1.

S04.01. Chair

H. L. Paulson;

Neurology, University of Michigan, Ann Arbor, MI.

Disclosures:

S04.02. Aberrant synaptic and circuit function in prodromal huntington disease

L. A. Raymond;

Dept Psychiatry and Centre for Brain Health, University of British Columbia, Vancouver, BC, CANADA.

Disclosures: L.A. Raymond: None.

S04.03. Region-specific vulnerabilities in spinocerebellar ataxia type 1

H. Zoghbi;

Jan and Dan Duncan Neurological Research Institute, Baylor College of Medicine, Houston, TX.

Disclosures:

S04.04. Perturbed ion channel function as a shared disease mechanism and therapeutic target in repeat expansion ataxias

V. Shakkottai;

Neurology, and Molecular and Integrative Physiology, UT Southwestern Medical Center, Dallas, TX.

Disclosures: V. Shakkottai: None.

S04.05. Impact and biology of repeat-associated non-ATG (RAN) translation in neurologic disease

L. Ranum;

Molecular Genetics and Microbiology, University of Florida, Gainesville, FL.

Disclosures:

Symposium

S05. Emerging Roles of the Lysosome in Neurodegenerative Disease

Location: Virtual Only

Time: Tuesday, November 9, 2021, 12:00 PM - 1:00 PM

Description: Emerging evidence demonstrates that lysosomes actively coordinate multiple cellular processes through their nutrient- and stress-sensing and nuclear-signaling abilities. Lysosome function declines with aging and contributes to the accumulation of pathological proteins in Alzheimer's disease and other neurodegenerative disorders. This symposium will address novel mechanisms regulating lysosome function and approaches that harness innate lysosomal sensing to promote toxic protein clearance.

Support: NIH R01 NS093652 (HZ)
NIH RF1 AG062257 (HZ)
NIH P01 AG017617 (RN)
NIH R01 AG062376 (RN)
NIH R01 AG057342 (AK)
NIH R01 AG059052 (AK)
NIH R01 NS095257 (AK)

Abstract: Traditionally regarded as a passive organelle for terminal degradation, recent studies demonstrate that the lysosome is positioned at the crossroads of multiple cellular processes, including stress response, nutrient sensing, metabolism and cellular clearance, and these involve coordinated signaling from the lysosome to the nucleus. Genetic and functional evidence establish a critical role of the lysosome in neurodegenerative diseases. This proposal addresses the emerging roles of the lysosome from basic biology to therapeutic targeting across the neurodegeneration spectrum, through the development of cutting-edge lysosomal tracking methods and integration of mouse genetics with patient-derived samples and datasets. Specifically, we will discuss the genetic basis for lysosomal dysregulation in Alzheimer's disease and related disorders, the lysosome-to-nucleus signaling pathways, and the

regulation of lysosomal pH and function in neurodegeneration. Lastly, we will provide therapeutic insight on targeting the nutrient-sensing pathway to promote autophagy-lysosome function in neurodegenerative disease.

Abstract: Traditionally regarded as a passive organelle for terminal degradation, recent studies demonstrate that the lysosome is positioned at the crossroads of multiple cellular processes, including stress response, nutrient sensing, metabolism and cellular clearance, and these involve coordinated signaling from the lysosome to the nucleus. Genetic and functional evidence establish a critical role of the lysosome in neurodegenerative diseases. This proposal addresses the emerging roles of the lysosome from basic biology to therapeutic targeting across the neurodegeneration spectrum, through the development of cutting-edge lysosomal tracking methods and integration of mouse genetics with patient-derived samples and datasets. Specifically, we will discuss the genetic basis for lysosomal dysregulation in Alzheimer's disease and related disorders, the lysosome-to-nucleus signaling pathways, and the regulation of lysosomal pH and function in neurodegeneration. Lastly, we will provide therapeutic insight on targeting the nutrient-sensing pathway to promote autophagy-lysosome function in neurodegenerative disease.

S05.01. Chair

H. Zheng;

Huffington Ctr Aging, Baylor College of Medicine, Houston, TX.

Disclosures:

S05.02. Co Chair

R. Nixon;

Center of Dementia Research, Nathan S. Kline Institute, Orangeburg, NY.

Disclosures:

S05.03. TFEB-mediated lysosome regulation and signaling in Alzheimer's disease

H. Zheng;

Huffington Ctr Aging, Baylor Col Med, Houston, TX.

Disclosures:

S05.04. Speaker

R. A. Nixon;

Psychiatry, Nathan S Kline Institute, Orangeburg, NY.

Disclosures:

S05.05. Dynamic regulation of lysosomal cathepsins and their contribution to aging and neurodegeneration

A. Kao;

Neurology, University of California, San Francisco, San Francisco, CA.

Disclosures:

S05.06. . Speaker

A. R. La Spada;

Duke University, Durham, NC.

Disclosures:

Symposium

S06. Neuroscience of Sensing and Controlling Bladder Function

Location: Virtual Only

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

Description: This symposium will describe the advances in understanding bladder afferent and efferent innervation, as well as emerging technologies that monitor bladder state. Discussions will highlight principles relating nerve activity patterns to organ state and function using novel engineered pressure, volume and nerve activity sensors. In addition, the identification of potential peripheral nerve targets and development of novel neuromodulation therapies for lower urinary tract dysfunction will be discussed.

Support: 3OT2OD023872
1U01DK101029
NIH U18EB021760
NIH OT2OD023873
NIH OT2OD024907
R01 NS088184
OT2 OD025297

Abstract: Urinary incontinence and voiding dysfunction is endemic among the elderly. Restoration of bladder function consistently ranks as a top priority for individuals with neurological injury and disorders such as spinal cord injury or multiple sclerosis. Much of this dysfunction is a result of deficits in sensory and motor systems that sense and transmit bladder and lower urinary tract function via spinal and brain networks that coordinate activity with other pelvic organs and somatic structures (e.g. pelvic floor). State of the art methods of sensing lower urinary tract dysfunction are leading to improved therapies for urinary incontinence. This session will highlight state of the art emerging neuroscience, technologies, mathematical modeling and strategies for potential therapeutic mechanisms to improve urinary continence and voiding function. Many of these solutions are driven by emerging results of molecular, cellular, and functional studies exploring peripheral innervation patterns and activity that control and responds to lower urinary tract function. Discussions will focus on: visualizations at mesoscopic and

microscopic scales of neural tracts and neural phenotyping for key components of peripheral and spinal circuits; comparative studies between rodent and human innervation patterns; computational methods for estimating bladder pressure based on sensory fiber and dorsal root ganglion activity patterns; real-time closed-loop bladder control in awake, behaving large animal models; optogenetic approaches for lower urinary tract control and functional recovery after spinal cord injury; phenotypic differences in anatomically defined bladder afferent populations and their implications for bladder sensation and function, and stimulation of spinal sensory pathways to control bladder function. The outputs of these studies will provide new maps, atlases, and establishment of proofs of principle for controlling bladder function. These results demonstrate critical foundations for the broader neuroscience community in the development of neuromodulation strategies for controlling and modeling organ function.

S06.01. Chair

M. S. Damaser;

Dept of Biomedical Engineering, Cleveland Clinic Lerner Research Institute, Cleveland, OH.

Disclosures:

S06.02. Co Chair

T. Best;

Stimulating Peripheral Activity to Relieve Conditions (SPARC), National Institute of Health, Washington, DC.

Disclosures:

S06.03. Controlling bladder function through electrical stimulation of the sensory system

R. A. Gaunt;

Physical Medicine and Rehabilitation, University of Pittsburgh, Pittsburgh, PA.

Disclosures: R.A. Gaunt: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Braingrade GmbH. F. Consulting Fees (e.g., advisory boards); Neurowired LLC.

S06.04. Mapping pudendal nerve function in humans with intraoperative electrophysiology, pelvis imaging, and computational modeling

T. M. Bruns;

Biomedical Engineering, University of Michigan, Ann Arbor, MI.

Disclosures: T.M. Bruns: 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.; Medtronic.

S06.05. Regulation of bladder function by sensory afferent populations

J. DeBerry;

Department of Anesthesiology, University of Alabama, Birmingham, AL.

Disclosures: J. DeBerry: None.

S06.06. Defining peripheral and spinal components of the bladder connectome

J. Keast;

University of Melbourne, Parkville, AUSTRALIA.

Disclosures: J. Keast: None.

Symposium

S07. Structural Basis of Sensation

Location: Virtual Only

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

Description: Our sensory systems provide us with some of life's greatest pleasures and, when they go awry, our most harrowing experiences. This symposium will explore some of the lesser-understood senses of touch, taste, and hearing. The speakers will describe the discovery and function of molecules that serve as sensory receptors or sensory transducers, including their clinical relevance.

S07.01. Chair

E. R. Liman;

Section of Neurobiology, University of Southern California, Los Angeles, CA.

Disclosures:

S07.02. Deciphering touch and feeling force in living animals

M. B. Goodman;

Molecular and Cellular Physiology, Stanford University, Stanford, CA.

Disclosures:

S07.03. A proton channel that tastes sour

E. R. Liman;

Section of Neurobiology, Univ. of Southern California (USC), Los Angeles, CA.

Disclosures: E.R. Liman: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Patent application (pending).

S07.04. My channel got a million hits: how hearing helps!

J. R. Holt;

Harvard Medical School / Boston Children's Hospital, Boston, MA.

Disclosures: J.R. Holt: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Scientific Founder, Audition Therapeutics. F. Consulting Fees (e.g., advisory boards); SAB, Audition Therapeutics.

S07.05. The molecules of mechanosensation and their roles in nociceptor sensitization

G. R. Lewin;

Dept of Neuroscience, Max-Delbruck-Center for Molecular Medicine, Berlin, GERMANY.

Disclosures:

S07.06. Co Chair

M. Goodman;

Stanford University, Stanford, CA.

Disclosures:

Symposium

S08. Spinal Interneurons and Neuroplasticity After Injury or Disease

Location: Virtual Only

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

Description: As interest in neuroplasticity increases and novel treatments are being developed to harness its potential, spinal interneurons have been identified as a key therapeutic target in the treatment of spinal cord injury and degenerative disease. This symposium will highlight our current understanding of spinal interneuron heterogeneity, their contribution to control and modulation of motor and sensory functions, how that role might change after traumatic spinal cord injury, and how treatments can be used to optimize their contribution to functional improvement.

Abstract: The central nervous system is not a static, hard-wired organ. Examples of neuroplasticity, whether at the level of the synapse, the cell, within and between circuits can be found during development, throughout the progression of disease or after injury. One essential component of the molecular, anatomical and functional changes associated with neuroplasticity is the spinal interneuron. This dedicated session will highlight advances in our understanding of the beneficial - and at times detrimental - roles that spinal interneurons play in regulation and modulation of motor, sensory and autonomic functions in the intact, developing and injured mammalian spinal cords. As our appreciation for the diversity and functional capacity of spinal interneurons continues to grow, they are also being identified as therapeutic targets for

promoting anatomical plasticity and functional recovery after traumatic injury or degenerative disease.

S08.01. Chair

M. A. Lane;

Neurobiology, Drexel University College of Medicine, Philadelphia, PA.

Disclosures:

S08.02. Co Chair

L. V. Zholudeva;

Gladstone Institutes, San Francisco, CA.

Disclosures:

S08.03. Spinal sensorimotor circuits: from development to function

M. D. Goulding;

MNL-G, Salk Institute, La Jolla, CA.

Disclosures:

S08.04. Wired for touch: the neurons and circuits of the somatosensory system

V. E. Abraira;

Cell Biology and Neuroscience, Rutgers University, Piscataway, NJ.

Disclosures:

S08.05. Reverse engineering spinal cord networks: revealing new roles for long propriospinal neurons

D. S. Magnuson;

Neurological Surgery, University of Louisville, Louisville, KY.

Disclosures: D.S. Magnuson: None.

S08.06. Spinal interneurons contributing to plasticity and recovery after spinal cord injury

K. Satkunendrarajah;

Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI.

Disclosures:

Symposium

S09. Neurorehabilitation in the 21st Century — Managing Plasticity

Location: Virtual Only

Time: Monday, November 8, 2021, 12:00 PM - 1:00 PM

Description: Neurorehabilitation, one of the most vibrant areas of neuroscience, lacks a comprehensive strategy. This symposium will show how new insights into how the central nervous system (CNS) acquires and maintains skills lead to the new concepts of Heksors and the Negotiated Equilibrium of CNS properties that Heksors create. The concepts provide two powerful new strategies for initiating and guiding widespread beneficial plasticity. Speakers will review the science underlying the strategies, the exciting results of their first human trials, and computer modeling to design new treatment protocols.

Support: Wolpaw: P41 EB018783(NIH/NIBIB); R01 NS110577(NIH/NINDS); I01CX001812-01 (VA); DOH01-C33279GG-3450000 (NY State SCIRB)
Thompson: NS069551(NIH/NINDS); C023685 & C029131(NY State SCIRB); SCIRF#2014 R01 (South Carolina SCIRF); P41 EB018783(NIH/NIBIB); P2C HD086844(NIH/NICHD)
Perez: NS090622(NIH/NINDS); I01BX007080(VA); The Walkabout Foundation.
Norman: NSF Graduate Research Fellowship Grant #2012143485; P41 EB018783(NIH/NIBIB); HD062744 (NIH/NICHD); UL1TR000153 (NIH/NCATS)

Abstract: From cortex to spinal cord, the CNS changes through life. New therapies seek to engage this plasticity to restore skills lost to injury or disease. How to do this, how to choose among many possible protocols, is a difficult problem, especially given the time and effort needed to test even one. Recent basic science insights into the mechanisms that enable the healthy CNS to acquire and maintain the many skills that share its neurons and synapses have led to the *negotiated equilibrium model*. The model elucidates the process of skill acquisition and maintenance and provides a comprehensive new therapeutic strategy. According to the negotiated equilibrium model, the CNS substrate of a motor skill is a distributed network of brain and spinal plasticity; the plasticity in brain induces and maintains the plasticity in spinal cord. When the skill (e.g., walking) is performed, errors in its key features (e.g., right/left symmetry) guide corrective changes in its substrate. The aggregate process is a negotiation among the skills. *They negotiate the properties of the neurons and synapses that they all use.* The ongoing negotiation maintains CNS properties in an equilibrium that serves all the skills. The negotiated equilibrium model (J Physiol 2018;596.16:3469-91) provides a therapeutic strategy that begins with *targeted neuroplasticity (TNP)*. A TNP method targets beneficial plasticity to a crucial CNS site (e.g., a spinal or corticospinal pathway used by many skills). By doing so, it improves the negotiation among skills. This enables CNS properties to escape a suboptimal local minimum and reach a superior new equilibrium. For example, a TNP method that reduces triceps surae hyperreflexia after spinal cord injury allows better gait, which produces beneficial plasticity at many sites and thus further improves gait. TNP methods lie at one end of a spectrum. At the other end are permissive methods (e.g., epidural spinal stimulation; skill practice) that restore to relevant CNS areas the ability to participate in the improved negotiation. Recent studies indicate that the most effective protocols combine a TNP method that improves a crucial site with a permissive method that enables the functional benefit of the targeted plasticity to initiate much

wider beneficial plasticity. Talk 1 presents the relevant basic science, the negotiated equilibrium model, and the comprehensive therapeutic strategy that the model provides. Talks 2 & 3 describe new noninvasive therapies using the strategy and the exciting results of their first clinical trials. Talk 4 discusses the use of computational modeling to select and parameterize promising therapeutic protocols prior to animal and human studies.

S09.01. Chair

J. R. Wolpaw;

National Center for Adaptive Neurotechnologies, Stratton Veterans Administration Medical Center, Albany, NY.

Disclosures:

S09.02. Co Chair

A. K. Thompson;

Health Sciences and Research, College of Health Professions, Medical University of South Carolina, Charleston, SC.

Disclosures:

S09.03. Targeted neuroplasticity enhances functional recovery after spinal cord injury

M. A. Perez;

Shirley Ryan AbilityLab, Shirley Ryan AbilityLab, Chicago, IL.

Disclosures: M.A. Perez: None.

S09.04. Operant conditioning of EMG evoked responses to improve impaired motor function in people with CNS disorders

A. K. Thompson;

Health Sciences and Research, College of Health Professions, Medical University of South Carolina, Charleston, SC.

Disclosures: A.K. Thompson: None.

S09.05. Targeted neuroplasticity and its functional impact after stroke: a computational study

S. L. Norman;

Andersen Lab, California Institute of Technology, Pasadena, CA.

Disclosures: S.L. Norman: None.

S09.06. Inducing and guiding CNS plasticity to restore function: the promise, the problem, the science, and the strategy

J. R. Wolpaw;

National Center for Adaptive Neurotechnologies, Wadsworth Center, NY State Department of Health, Albany, NY.

Disclosures:

Symposium

S10. Memory in the Light of Single Neuron Recordings in Humans

Location: Virtual Only

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

Description: Intracranial recordings from patients with epilepsy provide an unprecedented opportunity to study the human memory system at the level of individual neurons and networks thereof. This symposium will provide new insights into the building blocks of episodic memory (including concept cells); single-trial learning; the ways by which associations are encoded; the ways by which memories are retrieved and used for decisions; and the role of persistent activity in the medial temporal lobe in working memory.

Support: U01NS103792
R01MH110831

Abstract: Memories are essential for human cognition, but we are only beginning to understand the underlying neural mechanisms. New insights into the mechanisms of human memory have recently been gained from utilizing in-vivo recordings from single neurons in humans undergoing treatment for drug resistant epilepsy. While relatively rare, technological and organizational advances have made such recordings more widely used. In this session, we highlight recent scientific results on human episodic and working memory gained with this technique. Our focus will be on new scientific insights gained from single-neuron and local field potential recordings in the human medial temporal and medial frontal lobes. We will discuss recent work on the representation of familiarity/novelty by memory selective cells and their contribution to confidence judgments in recognition memory, the role of memory-choice neurons in medial frontal cortex in recognition memory, the role of persistently active neurons in the medial temporal lobe in supporting working memory maintenance, the encoding of associations, and semantically invariant concept cells as building blocks for episodic memories. Together, these results begin to provide a circuit-level understanding of human memory.

S10.01. Chair

U. Rutishauser;

Department of Neurosurgery, Cedars-Sinai Medical Center, Los Angeles, CA.

Disclosures:

S10.02. Co Chair

J. Sarnthein;

Klinik für Neurochirurgie, University Hospital of Zürich, Zurich, SWITZERLAND.

Disclosures:

S10.03. Semantically invariant concept neurons in the human temporal lobe as building blocks of episodic memory

F. Mormann;

University of Bonn, Bonn, GERMANY.

Disclosures:

S10.04. A single-neuron perspective on human memory

U. Rutishauser;

Department of Neurosurgery, Cedars-Sinai Medical Center, Los Angeles, CA.

Disclosures:

S10.05. Single neurons in the human hippocampus encode associations between related stimuli

L. Reddy;

CNRS-Centre de Recherche Cerveau et Cognition, Toulouse, FRANCE.

Disclosures: L. Reddy: None.

S10.06. Verbal working memory integrates persistent firing of hippocampal neurons into cortico-hippocampal network activity

J. Sarnthein;

Klinik für Neurochirurgie, Universitätsspital Zurich, Zurich, SWITZERLAND.

Disclosures: J. Sarnthein: None.

Symposium

S11. Internal Brain States and the Dynamics of Behavior

Location: Virtual Only

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

Description: Internal brain states are the evolving prism through which external information and inner goals shape animal behavior. This prism has been difficult to study because it cannot be observed from outward behavior alone. New work that leverages quantitative behavioral analysis, computational modeling, and population neural recordings is beginning to shed light on the nature of dynamic internal states. The four speakers are leaders in this research area and will share their latest findings.

Abstract: Internal brain states are the evolving prism through which external information and inner goals shape animal behavior. This prism has been difficult to study because it cannot be observed from outward behavior alone. New work that leverages quantitative behavioral analysis, computational modeling, and population neural recordings, is beginning to shed light on the nature of dynamic internal states. The four speakers are leaders in this research area and will share their latest findings.

S11.01. Chair

M. Murthy;

Neuroscience Institute, Princeton University, Princeton, NJ.

Disclosures:

S11.02. Behavioral state tuning in the striatum

B. Olveczky;

OEB, Harvard University, Cambridge, MA.

Disclosures:

S11.03. What does a flexible navigational goal look like in the brain?

G. Maimon;

Rockefeller University and HHMI, NY, NY.

Disclosures:

S11.04. Internal states and the dynamics of social behavior

M. Murthy;

Neuroscience Institute, Princeton University, Princeton, NJ.

Disclosures:

S11.05. Rich behavioral dynamics during decision-making and their impact on cortical activity

A. K. Churchland;

UCLA, Los Angeles, CA.

Disclosures: A.K. Churchland: None.

Symposium

S12. Mitochondrial and Metabolic Substrates of Cellular, Circuit, and Complex Behavioral Responses to Stress

Location: Virtual Only

Time: Monday, November 8, 2021, 10:45 AM - 11:45 AM

Description: There is high mechanistic overlap between metabolic and behavioral dysfunction. This symposium will present data that demonstrate that altering metabolism via environmental manipulations changes neural circuits important in regulation of social behavior, mood, and cognition. Speakers will show how environmental stressors, which increase susceptibility to mood disorders, impact peripheral and neural metabolic processes at the cellular level. Particular focus will be placed on mitochondria as a neurometabolic hub.

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R01MH119336
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NCCR Synapsy 51NF40-158776
R01NR014886
R01MH113512

Abstract: Neuropsychiatric and neurodegenerative disorders are increasingly recognized to involve disrupted cellular metabolism. Energetic defects within mitochondria, a nexus for multiple factors to impact cellular and circuit function, have also been described. We will show how environmental stressors, which increase susceptibility to mood disorders, impact peripheral and neural metabolic processes at the cellular level. First, we will show how repeated social defeat stress affects mitochondrial function, with a particular focus on how mitochondrial phenotypes differ between brain regions. Second, we will explore how nutritional interventions can help reverse deficiencies in both motivated behaviors and striatal metabolism. Third, we will demonstrate causative links between dietary or psychosocial stressors during development and mitochondrial function in the brains of adults. Finally, we will show how disruption of sleep and circadian rhythms, perhaps the most common quality of life complaint in many psychiatric disorders, affects neurometabolic processes. In summary, this symposium will highlight the importance of brain-body interactions in the healthy function of brain circuits and behavioral outputs, by probing metabolic processes using both bottom-up and top-down approaches.

S12.01. Chair

I. N. Karatsoreos;

Psychological and Brain Sciences, University of Massachusetts, Amherst, Amherst, MA.

Disclosures:

S12.02. Co Chair

G. N. Neigh;

Anatomy, Virginia Commonwealth University, Richmond, VA.

Disclosures:

S12.03. Profiling brain mitochondrial recalibrations in response to psychological and neuroendocrine stress

M. Picard;

Psychiatry, Columbia University Medical Center, New York City, NY.

Disclosures:

S12.04. Chronic stress and low-level inflammation alter cognition and synaptosomal respiration

G. N. Neigh;

Anatomy, Virginia Commonwealth University, Richmond, VA.

Disclosures: G.N. Neigh: None.

S12.05. Consequences of disrupted circadian rhythms on sleep and neurometabolism

I. N. Karatsoreos;

Psychological and Brain Sciences, University of Massachusetts, Amherst, Amherst, MA.

Disclosures: I.N. Karatsoreos: None.

S12.06. Brain mitochondria and metabolism in motivated behaviors - regulation by early life stress and nutritional interventions

C. Sandi;

Brain Mind Institute, EPFL, Lausanne, SWITZERLAND.

Disclosures: C. Sandi: 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 S.A.. C. Other Research Support (receipt of drugs, supplies, equipment or other in-kind support); Nestle S.A.. E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Amazentis S.A.. F. Consulting Fees (e.g., advisory boards); Amazentis S.A..

Symposium

S13. Estrogens Across the Adult Female Lifespan — Rapid and Chronic Influences of Estrogens on Structural Plasticity, Memory, and Disease

Location: Virtual Only

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

Description: Estrogens influence neural function in critical ways throughout the adult female lifespan that can significantly impact cognition, affect, and recovery from brain injury. Of note, reproductive experience and age can drastically affect the response to estrogens. This symposium will review recent evidence that estradiol can act both rapidly and chronically to affect brain function, memory, and affective behaviors in young and aging female rodents.

Support: National Institute of Mental Health R01MH107886 to Karyn Frick

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Alzheimer's Association SAGA-17-419092 to Karyn Frick
Natural Sciences and Engineering Research Council of Canada RGPIN-2018-04699 to Elena Choleris
Natural Science and Engineering Research Council of Canada RGPIN-2018-04301 to Liisa Galea
Canadian Institutes for Health Research MOP 142308, PJT-148662 to Liisa Galea
National Institutes of Health AG042189 and NS074895 to Farida Sohrabji.

Abstract: This symposium will discuss rapid and sustained effects of estrogens on brain function and cognition that change during the adult female lifespan. Estrogens are associated with depression and anxiety disorders in women, and age-related estrogen loss increases risks of cognitive decline, Alzheimer's disease, and depression. Yet too little is known about how brain estrogen levels during the reproductive cycle, pregnancy, postpartum, and menopausal transitions influence cognitive and affective function. Understanding the mechanisms in the brain through which estrogens modulate neural function throughout the female lifespan, including the response to aging, stress, and stroke may provide new avenues for the development of therapies that mimic the benefits of estradiol without side effects. This symposium features four talks that address multiple aspects of estradiol's effects on cognitive and neural function throughout the adult female lifespan. The first will illustrate how rapid estradiol-induced changes in hippocampal structure and function are necessary for memory consolidation in young adult mice of both sexes. Brain regions involved in rapid action of estrogens and their receptors on social cognition will then be discussed. The next talk will describe the impact of estrogens across the lifespan on neurogenesis in the hippocampus and stress resilience. Finally, the impact of reproductive age and estrogen treatment on stroke recovery will be addressed.

S13.01. Chair

K. M. Frick;

Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI.

Disclosures:

S13.02. Brain regions involved in the rapid action of estrogens and their receptors on social cognition

E. Choleris;

Psychology, University of Guelph, Guelph, ON, CANADA.

Disclosures: E. Choleris: None.

S13.03. Impact of reproductive age and estrogen treatment on stroke recovery

F. Sohrabji;

Neuroscience and Exp Therapeutics, Texas A and M University System, Bryan, TX.

Disclosures:

S13.04. Neural mechanisms underlying estradiol's rapid effects on memory consolidation and dendritic spine morphology

K. M. Frick;

Department of Psychology, Univ. of Wisconsin-Milwaukee, Milwaukee, WI.

Disclosures: K.M. Frick: E. Ownership Interest (stock, stock options, royalty, receipt of intellectual property rights/patent holder, excluding diversified mutual funds); Co-founder and stock holder of Estrigenix Therapeutics, Inc..

S13.05. The good, the bad and the confusing: effects of estrogens on hippocampal neurogenesis and stress resilience

L. A. Galea;

Djavad Mowafaghian Centre for Brain Health, Psychology, University of British Columbia, Vancouver, BC, CANADA.

Disclosures: L.A. Galea: None.

Symposium**S14. Noncanonical Hippocampal Memory Circuits**

Location: Virtual Only

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

Description: Memory research has long focused on the excitatory connections linking the hippocampus and entorhinal cortex. Recent work has begun to consider noncanonical circuits, including some long-ignored subcortical inputs and outputs, that have the ability to shape hippocampal function and bias circuit output. This symposium will highlight current work along these lines and touch on common motifs that shape memory.

Support: JSPS Kakenhi 19H05646 (T.J.M)
JSPS Kakenhi 17H05591 (T.J.M)
JSPS Kakenhi 19H05233 (T.J.M)
Medical Research Council UK Programme MC_UU_12024/3 (D.D)
Hungarian Brain Research Program 2017-1.2.1-NKP-2017-00002 (G.N)
NIH MH119391 (S.M)

Abstract: The mnemonic functions of the hippocampal circuit have long been viewed through the classic model of sequential processing along the trisynaptic loop; the large number and sparse activity of granule cells in the dentate gyrus (DG) providing orthogonalization of similar inputs from the entorhinal cortex, the DG providing input to the recurrent CA3 network to facilitate autoassociation and finally, CA1 broadcasting the results back to the cortex. However, recent advances have led to a new consideration of noncanonical circuits impinging on and

radiating from the hippocampus, including some long ignored subcortical inputs and outputs that have the ability to shape hippocampal function and bias circuit output. This symposium will bring together speakers combining physiological, behavioral and genetic approaches to better understand how these circuits influence memory and to motivate discussion of the larger hippocampal network.

S14.01. Chair

T. J. McHugh;

Lab for Circuit & Behavioral Physiology, RIKEN Center for Brain Science, Wakoshi, JAPAN.

Disclosures:

S14.02. Hippocampal involvement in decision making neurocircuitry

S. J. Mizumori;

University of Washington, Seattle, WA.

Disclosures:

S14.03. Hypothalamic modulation of hippocampal memory

T. J. McHugh;

Lab for Circuit & Behavioral Physiology, RIKEN Center for Brain Science, Wako-shi, JAPAN.

Disclosures: T.J. McHugh: None.

S14.04. Brainstem control of memory formation.

G. Nyiri;

Institute of Experimental Medicine, Budapest, HUNGARY.

Disclosures: G. Nyiri: None.