



IDG/McGOVERN INSTITUTE
FOR BRAIN RESEARCH AT TSINGHUA

TSINGHUA NEUROSCIENCE **INTERNATIONAL CONFERENCE** **ON BRAIN AND BODY**



CONFERENCE HANDBOOK



🕒 **23-24 October, 2025**

📍 **The Lecture Hall, Main Building, Tsinghua University**

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IDG/McGOVERN INSTITUTE
FOR BRAIN RESEARCH AT TSINGHUA

The IDG/McGovern Institute for Brain Research at Tsinghua University was established in 2011, with the generous support from the McGoverns and IDG Capital. The goal is to build a world leading, new technology driven brain research center. The Institute currently hosts more than 38 outstanding faculty members in the fields of neuroscience and neuroengineering. Scientists working in the IDG/McGovern Institute at Tsinghua University use the latest advances in molecular and cellular biology, imaging, electrophysiology, and behavioral techniques to investigate how brain functions and how to prevent and treat brain disorders.

In 2018, Essential Science Indicators (ESI) announced that the neuroscience discipline at Tsinghua University entered the top 1% of global scientific research institutions for the first time.



23 October, 2025

Session 1: Homeostasis

Session Chair: Bailong Xiao

09:00-09:10	Opening Remarks
09:10-09:20	Group Photo
09:20-10:00	Jeffrey M. Friedman (Rockefeller University) (KEYNOTE) Title: A simple circuit regulating food intake
10:00-10:30	Jing Wang (Shenzhen Bay Laboratory) Title: Gut-brain communication in <i>Drosophila</i> : from nutrient sensing to microbial influence
10:30-11:00	Wenwen Zeng (Tsinghua University) Title: Sympathetic regulation of energy balance
11:00-11:20	Tea Break
11:20-11:50	Bo Li (Westlake University) Title: Dissecting the neural circuitry underlying motivated behaviors
11:50-12:20	Peng Cao (National Institute of Biological Sciences, Beijing) Title: Brainstem opioid peptidergic neurons regulate cough reflexes in mice
12:20-14:00	Lunch Break

Session 2: Somatosensation and Interoception I

Session Chair: Kun Li

14:00-14:30	Zhou-Feng Chen (Shenzhen Bay Laboratory) Title: How pain inhibits itch?
14:30-15:00	Marco Gallio (Northwestern University) Title: Temperature sensing and preference in <i>Drosophila</i> : from molecular receptors, to neural circuits, to the evolution of behavior
15:00-15:30	Jiefu Li (HHMI's Janelia Research Campus) Title: Surfing the cell surface: a deep dive into the 'superficial'

15:30-16:00	Gary Lewin (Max Delbrück Center for Molecular Medicine) Title: Identifying necessary mechanotransduction components for mammalian touch
16:00-16:20	Tea Break
16:20-16:50	Xinzhong Dong (Johns Hopkins, School of Medicine) Title: The roles of Mrgpr GPCR family in itch, pain, and innate immunity
16:50-17:20	Wei Zhang (Tsinghua University) Title: Pandiculation regulates arousal state via proprioceptive pathways
17:20-17:50	Xin Duan (University of California, San Francisco) Title: Translating transcriptomics to connectomics at retinotectal synapses
17:50-20:00	POSTER

24 October, 2025

Session 3: Emotion and Social behavior

Session Chair: Marco Gallio

09:00-09:30	Lisa Stowers (The Scripps Research Institute) Title: Pheromones on tap: active behavior gates and modulates vomeronasal sensation
09:30-10:00	Xiang Yu (Peking University) Title: Using oxytocin to treat autism: insights from animal models
10:00-10:30	Liping Wang (Shenzhen Institute of advanced technology, Chinese academy of sciences) Title: Bone-derived hormone permits visual escape
10:35-10:50	Tea Break
10:50-11:20	Song-Hai Shi (Tsinghua University) Title: Evolutionary emergence of striatal GABAergic interneuron types in mammals modulates behavioural flexibility

11:20-11:50	Kun Li (Tsinghua University) Title: Top-down regulation of adaptive social behaviors: from instinct to impulse
11:50-13:00	Lunch Break
13:00-14:00	Campus Tour

Session 4: Somatosensation and Interoception II

Session Chair: Wei Zhang

14:00-14:40	John Wood (University College London) (KEYNOTE) Title: Sodium channels and pain
14:40-15:10	Nieng Yan (Shenzhen Medical Academy of Research and Translation) Title: Structural Pharmacology of Na _v and Ca _v channels
15:10-15:40	Juanjuan Du (Tsinghua University) Title: Targeting ion channels with ligand-antibody conjugates
15:40-16:00	Tea Break
16:00-16:30	Yulong Li (Peking University) Title: Orphan GPCRs, chronic liver diseases and itch: from bench to bedside
16:30-17:00	Ru-Rong Ji (Duke University Medical Center) Title: Neuroglial network in acute and chronic pain
17:00-17:30	Bailong Xiao (Tsinghua University) Title: Mouse behavioral genomics identifies a gatekeeper of somatosensation
17:30-17:40	Closing Remarks



SESSION 1

Homeostasis





Jeffrey M. Friedman

◆ Rockefeller University

TITLE:

A simple circuit regulating food intake

ABSTRACT:

Leptin and the Regulation of Food Intake and Body Weight. Leptin is an adipose tissue hormone that maintains homeostatic control of adipose tissue mass. This endocrine system thus serves a critical evolutionary function by protecting individuals from the risks associated with being too thin (starvation) or too obese (predation). Mutations in leptin or its receptor cause massive obesity in mice and humans, and leptin can effectively treat obesity in leptin deficient patients. The identification of leptin has thus provided a framework for studying the regulation of feeding behavior and the pathogenesis of obesity. The identification of leptin has also advanced our understanding of the neural mechanisms that control feeding. Current research focuses on the function of specific neural populations in the hypothalamus and other brain regions to control feeding behavior and energy balance. Recent studies have linked arcuate neurons that respond to leptin and a brainstem motor output that coordinates motor programs associated with food consumption. These findings identify the components of a simple subcortical circuit that regulates feeding thus providing a possible framework for elucidating how complex information is processed to generate specific behaviours.

BIOGRAPHY:

Dr. Jeffrey Friedman is a Professor at the Rockefeller University and an Investigator at the Howard Hughes Medical Institute studying the physiologic and genetic mechanisms that regulate food intake and body weight. In 1994, his laboratory isolated the mouse ob gene and showed that it encodes the hormone leptin that reduces food intake in mice. Current research is aimed at understanding the neural and physiological mechanisms by which leptin transmits its weight-reducing signal. He is a member of the National Academy of Science and has won numerous awards including the 2010 Albert Lasker Basic Medical Research Award, the 2019 Wolf Prize in Medicine, the 2009 Shaw Prize, the 2005 Gairdner Award and the 2020 Breakthrough Prize in Life Sciences.





Jing Wang

◆ Shenzhen Bay Laboratory

TITLE:

Gut-brain communication in *Drosophila*: from nutrient sensing to microbial influence

ABSTRACT:

Animals typically display one behavior at a time, switching from one to another at the appropriate moment, governed by evolutionarily selected protocols. In electronic systems, operational protocols are determined by circuit topology, a principle that also applies to biological systems, such as the lac operon, which serves as a protocol for metabolic change. I will first discuss our investigation into the protocol and circuit topology governing feeding and courtship behaviors in *Drosophila*. We found that feeding is initially prioritized in starved males, but the intake of protein-rich food prompts a swift protocol change, prioritizing courtship within minutes. This shift is mediated by a gut-derived neuropeptide. Amino acids in food trigger the release of this neuropeptide, which subsequently excites specific brain neurons. One neuronal population inhibits feeding while another promotes courtship. Our findings illustrate how the gut-brain axis exerts a profound influence on behavioral choice. Furthermore, the gastrointestinal tract is home to a community of microorganisms. I will present a new study that addresses how commensal bacteria in the fly gut influence host food consumption by altering signaling molecules in the gut-brain axis.

BIOGRAPHY:

Dr. Jing Wang received a bachelor's degree in physics and a master's degree in biophysics from Tsinghua University in 1987 and 1990, respectively. He spent 20 years at UC San Diego, initially as Assistant Professor and was promoted to Professor in 2014. He joined Shenzhen Bay Laboratory in January 2025 as Senior Principal Investigator and Director of the Institute of Molecular Physiology. His research focuses on neuromodulation of chemosensory processing. He employs a broad range of cutting-edge technologies to investigate how gut-derived neuropeptides regulate feeding and mating behaviors as well as how internal physiological states modulate interoceptive detection of nutrients. His past work has significantly advanced our understanding of gut-brain communication, metabolic modulation of olfactory circuits, and feeding behavior. During his postdoctoral training with Richard Axel (2004 Nobel Laureate), he pioneered the use of the genetically encoded calcium sensor GCaMP for in vivo cellular imaging. He has continued to develop new tools for neuroscience in his own lab, including the widely used CaLexA activity reporter system and non-invasive three-photon microscopy.





Wenwen Zeng

◆ Tsinghua University

TITLE:

Sympathetic regulation of energy balance

ABSTRACT:

The peripheral nervous system serves as a crucial link between the brain and the body. Within this system, the sympathetic nervous system plays an important role in controlling metabolic functions and maintaining energy balance. By unraveling the mechanisms by which the sympathetic nervous system operates, we have gained a deeper understanding of how our bodies adapt to different conditions, leading to improved health outcomes. Through examining the neural connections, we have uncovered that the sympathetic nerves are densely present in metabolic organs. We have further demonstrated their essential role in regulating lipid and glucose metabolism. Through communication with adrenergic receptors, the sympathetic input can alter energy expenditure and influence hormone secretion. As a result, the sympathetic nervous system can affect cellular activities in various organs and influence metabolic homeostasis. Overall, our research has unraveled the functions of the sympathetic nervous system, offering insights into how the body adjusts and reacts to various stimuli, ultimately impacting our overall health.

BIOGRAPHY:

Dr. Wenwen Zeng is a professor at the Institute for Immunology and the School of Basic Medical Sciences, Tsinghua University, in Beijing. She earned her B.S. degree from Tsinghua University and her Ph.D. from the University of Texas Southwestern Medical Center in Dallas. She completed postdoctoral training at Genentech Inc. and Rockefeller University. Her research group at Tsinghua University focuses on the peripheral nervous system, particularly its role in connecting the brain to organs and its associated pathophysiology.

Dr. Zeng has received the Youth Technology Prize, the National Science Fund for Distinguished Young Scholars, the National Science Fund for Excellent Young Scholars, the Outstanding Young Scholar Award from the Qiu-Shi Science & Technologies Foundation, the Young Investigator Award from the Lipid Metabolism and Bioenergetics Subociety of the Biophysical Society of China, and the National Young Investigator Fund. She serves as an associate editor for AJP-Endo, Endocrinology, and Molecular Immunology, and is on the editorial boards of Protein & Cell, Science Bulletin, Advanced Science, Journal of Genetics and Genomics, and Oxford Open Immunology.





Bo Li

◆ Westlake University

TITLE:

Dissecting the neural circuitry underlying motivated behaviors

ABSTRACT:

The amygdala and basal ganglia circuits have important roles in learning and executing behaviors motivated by either appetitive or aversive stimuli. How exactly these circuits contribute to the generation of divergent behavioral responses remains elusive. Our recent studies indicate that learning driven by reward or punishment induces distinct plastic changes in discrete circuits in the basal ganglia and the amygdala, and reveal how these learning-induced changes participate in guiding flexible behaviors. Interestingly, neurons in these circuits can also convey information about the nutritional properties of foods and the metabolic status of animals, and furthermore control energy utilization and weight gain. An emerging picture is that these circuits are used to regulate different aspects of motivated behaviors as well as energy homeostasis.

BIOGRAPHY:

Dr. Bo Li is Chair Professor of Neuroscience at Westlake University. He received his Ph.D. in Neuroscience from The University of British Columbia in 2003. He did his postdoctoral research from 2003 to 2008, first at Cold Spring Harbor Laboratory (CSHL) and then at University of California, San Diego. He was recruited back to CSHL as an Assistant Professor in 2008, and was promoted to Associate Professor in 2013 and Professor in 2017. He was the Robert Lourie Endowed Professor at CSHL before joining Westlake University in August 2023. Research in Dr. Bo Li's laboratory focuses on the cellular and circuit mechanisms underlying aspects of motivated behaviors – including motivation, valence processing and learning – as well as cellular and circuit dysfunctions that may underlie mental disorders, including depression, anxiety disorders, autism, and drug addiction. More recently, Dr. Li's team has embarked on studying the interactions between the brain and peripheral metabolism in the contexts of cancer progression and obesity. His research integrates in vitro and in vivo electrophysiology, imaging, molecular, genetic, optogenetic, chemogenetic and RNA sequencing technologies to probe and manipulate the functions of specific neural circuits in the rodent brain, and to determine their roles in adaptive/maladaptive behavioral and metabolic responses.





Peng Cao

◆ National Institute of Biological Sciences, Beijing

TITLE:

Brainstem opioid peptidergic neurons regulate cough reflexes in mice

ABSTRACT:

Cough is a vital defensive reflex for expelling harmful substances from the airway. The sensory afferents for the cough reflex have been intensively studied. However, the brain mechanisms underlying the cough reflex remain poorly understood. Here, we developed a paradigm to quantitatively measure cough-like reflexes in mice. Using this paradigm, we found that prodynorphin-expressing (*Pdyn+*) neurons in the nucleus of the solitary tract (NTS) are critical for capsaicin-induced cough-like reflexes. These neurons receive cough-related neural signals from *Trpv1+* vagal sensory neurons. The activation of *Pdyn+* NTS neurons triggered respiratory responses resembling cough-like reflexes. Among the divergent projections of *Pdyn+* NTS neurons, a glutamatergic pathway projecting to the caudal ventral respiratory group (cVRG), the canonical cough center, was necessary and sufficient for capsaicin-induced cough-like reflexes. These results reveal that *Pdyn+* NTS neurons, as a key neuronal population at the entry point of the vagus nerve to the brainstem, initiate cough-like reflexes in mice.

BIOGRAPHY:

Dr Peng Cao is a principal investigator from National Institute of Biological Sciences in Beijing. His laboratory investigates the brain-body interface for detecting pathogens/toxins and initiating defensive responses in the gut and airway.





SESSION 2

Somatosensation and Interoception I





Zhou-Feng Chen

◆ Shenzhen Bay Laboratory

TITLE:
How pain inhibits itch?

ABSTRACT:

Itch and pain are distinct sensory modalities, encoded and transmitted from the skin to the brain through separate pathways. Yet, they also interact antagonistically, with pain notably inhibiting itch. In this talk, I will describe the organizational principle underlying pain-mediated inhibition of itch at the spinal level. Our findings demonstrate that neuropeptides in primary afferents encode modality-specific information, conveyed through functionally distinct labeled lines or microcircuits defined by non-overlapping GPCR expression in the dorsal spinal cord. Although pain and itch circuits are distinct, pain can inhibit itch by activating a dedicated inhibitory circuit defined by a unique GPCR expression and a specific neuropeptide-receptor interaction. The cross-modality inhibition highlights the intricate architecture of dorsal horn circuitry, characterized by high-fidelity specificity shaped through evolution.

BIOGRAPHY:

Zhou-Feng Chen received his B.S. at Wuhan University, his Ph.D. at the University of Texas Health Science Center at Houston, and his postdoctoral training at Caltech. He subsequently joined Washington University School of Medicine, where he founded the Center for the Study of Itch and Sensory Disorder and held the Russell D. and Mary B. Shelden Professorship. He later joined Shenzhen Bay Laboratory and SMART as a senior investigator. His research focused on the coding mechanisms of somatosensation, and more recently expanded to emotional circuits underlying pleasure, depression, and anxiety.





Marco Gallio

◆ Northwestern University

TITLE:

Temperature sensing and preference in *Drosophila*: from molecular receptors, to neural circuits, to the evolution of behavior

ABSTRACT:

How temperature preference evolves during the colonization of new environments is not known. Here, we show that at least two distinct neurobiological mechanisms drive the evolution of temperature preference in *Drosophila*. Fly species from mild climates (*D. melanogaster*, *D. persimilis* etc.) avoid heat, and we show that this can be fully explained by differences in the activation threshold of the peripheral hot receptor neurons. Desert-dwelling *D. mojavensis* are instead attracted to heat. We demonstrate that this is due to a valence switch, from aversive to attractive, in how the brain processes input from the peripheral receptors. Although insects are ubiquitous, few species inhabit thermal extremes. Our findings illustrate how adaptation to desert life in *Drosophila* involved a remarkable rewiring of the thermosensory system.

BIOGRAPHY:

Dr. Gallio is a Professor at the department of Neurobiology, Northwestern University, the Soretta and Henry Shapiro Research Professor in Molecular Biology, a member of the National Institute for Theory and Mathematics in Biology, and the Director of the Northwestern University Master of Science in Neurobiology program. He received a BS from the University of Pavia (Pavia, Italy) and a PhD from the Karolinska Institute (Stockholm, Sweden). He carried out postdoctoral research at University of California, San Diego and at Columbia University as an HHMI postdoctoral fellow before joining Northwestern University in 2012. His work has been funded by grants from the National Institutes of Health, Pew Charitable Trust, the McKnight Foundation, the Paula M. Trienens Institute for Sustainability and Energy and the NSF-Simons National Institute for Theory and Mathematics in Biology. Gallio lab research has been published in high profile journals such as *Nature*, *Nature Neuroscience*, *Nature Communications* and *Current Biology*.





Jiefu Li

◆ HHMI's Janelia Research Campus

TITLE:

Surfing the cell surface: a deep dive into the 'superficial'

ABSTRACT:

Hundreds of millions of years ago, single cells began sticking together, gaining powerful evolutionary advantages. These early alliances ignited the rise of multicellular life. At the heart of this shift lies cell-surface signaling—controlling how cells connect, communicate, and shape the form and function of complex organisms. We developed a generalizable, wet-to-dry method suite for in situ cell-surface proteomics in native tissue environment—spanning chemical and transgenic tools to an intuitive, user-friendly data analysis platform. Applying this approach to the mouse brain and internal organs revealed how neuronal and vascular surface milieu changes across development, maturation, and aging, and uncovered unexpected regulators of neural circuit formation and vascular barrier integrity.

BIOGRAPHY:

Jiefu Li is a Group Leader of 4D Cellular Physiology at the Janelia Research Campus of the Howard Hughes Medical Institute. His laboratory develops chemical, genetic, and computational methods to advance spatial proteomics and volume electron microscopy. By integrating these cutting-edge technologies, the Li lab investigates how cell-surface signaling governs cellular organization in the nervous and vascular systems. Before joining Janelia in 2022, Jiefu Li earned his B.S. from Shanghai Jiao Tong University in 2014 and his Ph.D. from Stanford University in 2020.





Gary Lewin

◆ Max Delbrück Center for Molecular Medicine

TITLE:

Identifying necessary mechanotransduction components for mammalian touch

ABSTRACT:

A gentle touch or a painful pinch are relayed to the brain and spinal cord by rapidly conducting myelinated sensory neurons. Many sensory neurons, especially vibration sensitive mechanoreceptors that innervate Meissner's or Pacinian corpuscles, are exquisitely sensitive to mechanical force, being activated by sub-micrometer displacements. Such mechanoreceptors possess mechanically-activated ion channels, like PIEZO2 and ELKIN1 that can be gated by small movements of the substrate^{1,2}. The PIEZO2-related ion channel PIEZO1 can be opened directly by membrane stretch, but it is still unclear whether PIEZO2 is stretch-activated. Both ELKIN1 and PIEZO2 are genetically required in mice for normal touch sensation. The question remains how are these channels gated *in vivo* to initiate touch sensation. We have provided evidence that extracellular tether proteins may transfer force from the matrix to native mechanotransduction channels, data which conforms to the so-called force from tether model³. The molecular nature of such tethers has, however, remained elusive. Here we identify the type II membrane protein TENM4 as an essential component for mechanosensory transduction in the vast majority of cutaneous mechanoreceptors, including fast conducting nociceptors. TENM4 mostly consists of a large dimeric extracellular domain which can interact with other extracellular proteins. Sensory neuron specific genetic ablation of *Tenm4* in mice led to profound touch insensitivity. Additionally, we devised a strategy to rapidly and reversibly disassemble the TENM4 protein at sensory endings, demonstrating its direct involvement in sensory transduction. TENM4 has been primarily studied in the context of neural development, but here we show an unexpected role in transferring force to mechanically activated channels like PIEZO2 and ELKIN1, with which it associates. We previously identified large (~100 nm long) extracellular filamentous structures in sensory neurons whose presence was associated with transduction³. Using immunogold labelling combined with transmission electron microscopy we provide evidence that such tethers contain TENM4 dimers. The identification of TENM4 as an essential component of fast somatic sensation including fast mechanical nociception is a major step towards identifying treatments for sensory disorders including pain. Our data also raise the intriguing possibility that the function of this protein may involve interactions with mechanically activated ion channels, also during brain development.





BIOGRAPHY:

Gary is Manx and grew up in Douglas on the Isle of Man. He received his first degree in Physiology and Pharmacology from Sheffield University in 1986, then worked on his doctoral thesis in Stephen B. McMahon's lab at St. Thomas's Hospital Medical school in London. He received his Ph.D. in February of 1990. He then moved to the lab of Professor Lorne Mendell in New York at Stony Brook. He worked in Lorne's lab for three years and in the last year he was appointed Research Assistant Professor. It was in Lorne Mendell's laboratory that Gary discovered that NGF is a critical mediator of hyperalgesia and pain. These findings formed the mechanistic basis of anti-NGF medication, which is now used to treat arthritis pain in dogs and cats. Human treatments exploiting the same biology are under development. In 1993 he received a von Humboldt Fellowship to work in the department of Neurobiochemistry at the Max-Planck Institute for Psychiatry in Munich under the directorship of Professor Yves-Alain Barde. In February of 1996 he took up an appointment as an independent Group Leader at the MDC in Berlin. The projects in his lab first focused on the molecular basis of sensory neuron mechanotransduction and sensory ion channels. In 2003 Gary obtained a joint appointment at the Charité University Medical Faculty as a full Professor. His lab has pioneered the study of sensory mechanotransduction and has identified critical molecules that are necessary for mammalian touch including the scaffold protein STOML3 and the mechanically gated ion channel ELKIN1. Most recently his lab has identified novel tether proteins necessary for mammalian sensory mechanotransduction. The lab is also developing small drug-like molecules that can interfere with touch sensation and can be used to treat pain. In addition to his work on mechanotransduction, his lab has pioneered the molecular exploitation of extreme physiology observed in the naked mole-rat and other African rodents. The lab has broadened its outlook and now looks at molecules that may promote metabolic health inspired by the phenomenal fitness and longevity of the naked mole-rat.





Xinzhong Dong

◆ Johns Hopkins, School of Medicine

TITLE:

The roles of Mrgpr GPCR family in itch, pain, and innate immunity

ABSTRACT:

We have taken a multidisciplinary approach to characterize a large family of G protein-coupled receptors (GPCRs) called Mrgprs. Many Mrgprs are specifically expressed in primary sensory neurons in dorsal root ganglia (DRG) and function as itch receptors by directly sensing variety of itchy substances including peptides, drugs, amino acids, lipids, and proteases. Besides sensory neuron-expressing Mrgprs, we found that several Mrgprs are present in innate immune cells like mast cells and neutrophils in barrier tissues. For example, MrgprX2 in humans and MrgprB2 in mice are exclusively expressed in mast cells and play an essential role in IgE-independent mast cell activation. We have shown the mast cell specific receptors play key roles in innate immunity at barriers, neurogenic inflammation, and various inflammatory diseases.

BIOGRAPHY:

Dr. Dong is a Professor in the Departments of Neuroscience, Dermatology, Neurosurgery at Johns Hopkins University School of Medicine. He is also an Investigator at Howard Hughes Medical Institute. He completed his PhD degree in Biochemistry at UCLA and his postdoc training at Caltech. His laboratory has taken a multidisciplinary approach to understand the cellular and molecular mechanisms of pain and itch. His lab has identified and characterized a novel family of G protein-coupled receptors (GPCRs) called Mrgprs which are expressed in primary sensory neurons and innate immune cells and function as novel itch receptors. Dr. Dong has published over 160 papers, book chapters, reviews on his research and trained numerous successful PhD and MD/PhD students and postdoctoral fellows. He received Javits Neuroscience Investigator Award from NIH/NINDS in 2021 and a member of AAAS. He is also a scientific founder of two companies: Escient Pharmaceuticals in San Diego, CA and SereNeuro Therapeutics in Baltimore, MD.





Wei Zhang

◆ Tsinghua University

TITLE:

Pandiculation regulates arousal state via proprioceptive pathways

ABSTRACT:

Pandiculation, the involuntary act of stretching and yawning when waking up or feeling tired, is a conserved behavior that occurs in many vertebrates. In the past a few decades, scientists explored the neuropharmacology and biological functions of yawning and stretching. However, the neuronal circuit underlying pandiculation remains unknown. Medullary center for yawning has not yet been identified. Finding medullary center for yawning would help us to perform controlled experiments based on various hypothesis about functions of yawning. Here, we found that CF-1 and C57 mice exhibit yawning behavior, most of which accompanied by head rising and body stretch. And we established vibration-wake-yawn paradigm. Using fluorogold and pseudorabies virus retrograde tracing method, we found the Acc7 motoneuron of digastric muscles and upstream brain areas of Acc7 might be involved in the control of pandiculation. We then identified a group of sensory neurons that innervate the facial area that are activated by yawning and project to the pons to regulate brain state. After identifying medullary center for yawning, we could measure different kinds of physiological parameters before and after artificially induced yawning, providing clues for biological functions of yawning and paving the way for contagious yawning study.

BIOGRAPHY:

Dr. Wei Zhang is an Associate Professor at the School of Life Sciences, Tsinghua University. He also serves as a principal investigator at the IDG/McGovern Institute for Brain Research and Tsinghua-Peking Center for Life Sciences. He has established his independent research group since joining the School of Life Sciences at Tsinghua University in 2017. His lab adopts the bottom-up pipeline of "Biophysics of Receptors - Cell Biology of Neurons - Functionality of Neural Circuits - Animal Behaviors" to explore the molecular and neural mechanisms of interoception. He also conducts multidimensional research on bidirectional nerve-organ communication and physiological regulation.





Xin Duan

◆ University of California, San Francisco

TITLE:

Translating transcriptomics to connectomics
at retinotectal synapses

ABSTRACT:

Different visual features are initially decoded by various types of retinal ganglion cells (RGCs), which act as conduits from the eyes to the brain. With the help of mouse genetic markers and viral tools, mechanistic studies have mapped intra-retinal circuits, such as those responsible for direction selectivity, revealing the computational mechanisms underlying these circuits. In contrast, once RGC axons leave the retina and enter the brain, the types of cells they innervate and the computations they drive remain poorly understood. Clarifying the wiring diagrams from the retina to the brain, starting from defined RGC types, is crucial for achieving a comprehensive understanding of how visual information is processed in the brain. To achieve this goal, we integrated a suite of circuit tracing, spatial transcriptomics, genetics, and imaging toolsets to map the circuitry immediately downstream of genetically defined RGC types in the mouse superior colliculus (SC). First, we combined Multiplexed Error-robust Fluorescence In Situ Hybridization (MERFISH) with an anterograde trans- neuronal tracer (mWGA-mCherry, PMID: 35524141), termed TransA-MERFISH. This technique enables high-throughput neuron capturing in the adult brain while preserving the spatial distribution of individual neurons. By introducing mWGA-mCherry into genetically defined RGCs, such as alpha-RGCs or direction-selective RGCs, as starters, we then registered retino-recipient neurons onto the MERFISH-based SC neuron type atlas, respectively. This high-throughput, imaging-based method delineates comprehensive wiring diagrams from retinal cells to retinorecipient neurons in the SC with single-neuron-type resolution. Second, to validate circuit predictions from the TransA-MERFISH experiments, we initiated a new set of genetic, optogenetic, electrophysiological, and retrograde tracing experiments to investigate whether selective RGC-SC neuronal pairs are specified at the retinotectal synapses. Third, building upon the molecules identified through spatial transcriptomics, such as Type II Cadherins (Cdh7, Cdh6, for example), we examined the roles of various cell-adhesive molecules in wiring the selective circuits identified here, such as Direction-Selective Circuits from the retina to the brain. These findings help establish a molecularly and genetically tractable model in the mammalian brain, addressing a series of long-standing questions regarding the assembly and function of the early visual pathway.





BIOGRAPHY:

Dr. Xin Duan graduated with the highest honors from Tsinghua University. He earned his PhD from Johns Hopkins University and completed postdoctoral training at Harvard University. Dr. Duan's research program focuses on the visual system, aiming to develop new tools to understand how its components are interconnected for specific visual features. The simplicity of the retina provides fundamental principles that underpin circuit assembly and neuronal repair in the mammalian brain. Dr. Duan and his team have employed innovative genetic and genomic engineering techniques to classify and study various subtypes of retinal neurons, particularly retinal ganglion cells, in order to comprehend their connectivity (Duan et al., 2014, *Neuron*; Duan et al., *Neuron*, 2018; Tsai et al., *Nature Nsci*, 2022). Using this same set of genetic tools in an optic nerve crush model, Dr. Duan also extended the studies to retinal neuron degeneration and regeneration (Duan et al., *Neuron*, 2015; Tsai et al., *Neuron*, 2022; Varadarajan et al., *Cell Reports*, 2023; Zhao et al., *Cell Reports*, 2023). Most recently, Dr. Duan's laboratory has initiated a new series of studies on neuron-guided vascular patterning (Toma et al., *Cell*, 2024; Nimkar et al., *Neuron*, 2025). Dr. Duan's work has been recognized by Stein Innovation Award, Klingenstein Neuroscience Fellowship, Whitehall Award, Ziegler Award, Catalyst for a Cure Team Award, among numerous other awards and honors.





SESSION 3

Emotion and Social behavior





Lisa Stowers

◆ The Scripps Research Institute

TITLE:

Pheromones on tap: active behavior gates and modulates vomeronasal sensation

ABSTRACT:

During natural behavior, sensation is a dynamic and directed process. How primary sensory neurons encode the environment is extensively studied in most sensory systems. However, how sensory inputs are used to meet the flexible goals of the animal has remained difficult to study. Here we are exploiting a uniquely tractable sensory system, the Accessory Olfactory system, to characterize primary sensation during free solo and social behavior. The Accessory Olfactory system is a chemical sense, mediating the detection of pheromones which evoke natural social behavior in the mouse. When and how animals use the Accessory Olfactory system during freely moving behavior is not known, so we established an approach to image primary sensory neural activity in the glomeruli of the accessory olfactory bulb in awake, freely moving mice using mini-endoscopes. This approach reveals that vomeronasal sensation is sparse, actively generated, and employed strategically to scale the persistence of sensory inputs. This selective use of pheromone sensation is expected to shed new light on how behavior is evoked and response dynamics of social circuits.

BIOGRAPHY:

Dr. Stowers obtained her PhD at Harvard University in 1997. She then joined Catherine Dulac's lab as a Howard Hughes Medical Institute Post-doctoral Fellow to undertake the study of how the brain determines fundamental behavior and emotion. During this training she completed experiments identifying a new subset of odor sensing neurons that detect cues that promote social behavior. In 2002 she began independent work at Scripps Research where she remains today as a Professor of Neuroscience. Her lab aims to understand basic rules of how the brain uses sensory cues from both the environment and internal body state to decide upon an appropriate behavior. The Stowers Lab focuses on natural, innate animal social behavior that is highly conserved across species such as courtship, rage, fear, and newborn-parent interactions. In this platform, the state of the mind of another is more readily interpretable from overt actions and the brain mechanisms and function are accessible to scientific testing. Moreover, this research strategy provides a window into most aspects of brain function because the display of social behavior requires multiple parallel computations such as learning and memory, sensory coding, motivation, sexual dimorphism, age-related changes, and integrate internal state needs such as hunger or sleepiness. Her work has been accelerated by using tools and methods in freely moving individuals because the state of one's own body colors many aspects of brain function creating a feedback loop in which one's behavior guides neural activity and decisions – which in turn guides subsequent behavior. Focusing on natural social behaviors in this context enables understanding of general principles about basic circuit and mechanistic function. She has been a finalist for the Eppendorf & Science Prize for Neurobiology; and named a Pew Scholar and Ellison Medical Foundation Senior Scholar in Neuroscience.





Xiang Yu

◆ Peking University

TITLE:

Using oxytocin to treat autism: insights from animal models

ABSTRACT:

Oxytocin plays diverse functional roles, contributing to regulation of socio-emotional and socio-sexual behaviors, sensory processing, learning and memory, modulation of stress and pain systems, as well as homeostatic, metabolic and autonomic responses. Reduction in oxytocin level, as well as SNPs in oxytocin receptor, are associated with autism. Oxytocin nasal spray has been clinically tested for autism treatment in a number of trials, with mixed results. Further mechanistic insight from animal studies can contribute towards more effective treatment strategies.

In previous work, we show that pleasant touch can increase the firing of oxytocin neurons in the paraventricular hypothalamus (PVH), and promoted social interactions (Yu et al., *Neuron*, 2022, PMID: 35045339). This effect was blocked in oxytocin knockout mice, and can be mimicked by chemogenetic activation of PVH oxytocin neurons. We also 3D reconstructed the complete axon, dendrite, and soma morphologies of individual PVH oxytocin neurons at submicron resolution, and showed extensive arborizations co-projecting to many brain regions, including multiple neuromodulatory regions (Li et al., *Neuron*, 2024, PMID: 38290516).

In ongoing work, identified reduced endogenous oxytocin level in the 15q dup mouse model of autism, and showed that i.p. injection of oxytocin, as well as elevating the firing of PVH oxytocin neurons can rescue both sensory and social deficits in the 15q dup model. We are currently exploring the circuit mechanisms through which oxytocin mediates its effects, as well as developing non-invasive approaches for elevating endogenous oxytocin levels.

BIOGRAPHY:

Xiang Yu is Professor in the School of Life Sciences at Peking University, and Director of the Autism Research Center of Peking University Health Science Center. She received her Bachelor's degree from Trinity College, University of Cambridge, completed her Ph.D at the MRC Laboratory of Molecular Biology in Cambridge, and carried out her post-doctoral work at Stanford University. Between 2005 and 2019, she was Principle Investigator at the Institute of Neuroscience, Chinese Academy of Sciences. Work from her laboratory has identified several key molecules and mechanisms regulating experience-dependent neural circuit development. The long-term goal of her laboratory is to understand which early neural circuit wiring mechanisms go awry in neurological disorders such as autism, and to develop targeted rescue strategies.





Liping Wang

- ◆ Shenzhen Institute of advanced technology,
Chinese academy of sciences

TITLE:

Bone-derived hormone permits visual escape

ABSTRACT:

Innate fear is an evolutionarily conserved adaptive mechanism that enables organisms to respond to threatening environmental stimuli. My lab has systematically deciphered the subcortical circuitry governing instinctive fear and its behavioral strategy selection, revealing how it is modulated by multiple factors including: environmental cues, positive contextual signals, negative threatening conditions, species-specific differences, and developmental stages. However, the neurobiological mechanisms underlying individual variability in instinctive fear responses, as well as how peripheral hormones such as osteocalcin regulate visually evoked escape behaviors, remain poorly understood. This presentation will highlight recent discoveries from my lab on the neural principles governing individual differences in innate defensive responses and elucidate how osteocalcin which is a bone-derived hormone, facilitates visually triggered flight behavior. Collectively, these findings provide critical structural and functional evidence for the existence of conserved "survival circuits" in the brain, demonstrating that the nervous system employs evolutionarily refined and universal strategies to coordinate emotional behaviors and maintain organismal homeostasis.

BIOGRAPHY:

Liping Wang received his Ph.D. in medical neuroscience from Charité -Universitätsmedizin Berlin and completed postdoctoral training at the Department of Bioengineering, Stanford University. He joined SIAT in 2009, and has been the founding director of BCBDI at SIAT since 2014. He holds the position as the vice president of the Chinese Neuroscience Society, and was supported by "National Science Fund for Distinguished Young Scholars" (2014). Dr. Liping Wang's research contributions focus on: 1) Pioneering the dissection of subcortical neural circuit and cellular mechanisms governing innate behaviors such as fear, feeding, and sleep, by integrating advanced animal behavior analysis techniques and optogenetics-based neural modulation tools developed by his team. 2) Elucidating structural and functional interactions between the central nervous system and peripheral systems under physiological and pathological conditions, particularly their relevance to mental disorder comorbidities. These discoveries provide critical structural and functional evidence for the "survival circuitry" in organisms, demonstrating that the brain employs conserved and universal strategies to coordinate emotional behavioral outputs and maintain systemic homeostasis. From an interdisciplinary perspective bridging neurobiology and integrative physiology, his findings offer novel insights into the brain-body interaction in a holistic view. To date, he has published over 130 research papers in journals including Nature (2007), Nature Methods (2006), Nature Reviews Neuroscience (2023), Neuron (2025, 2023, 2022, 2019), JCI (2020), and Nature Communications (2021, 2016a, 2016b, 2015a, 2015b, 2014).





Song-Hai Shi

◆ Tsinghua University

TITLE:

Evolutionary emergence of striatal GABAergic interneuron types in mammals modulates behavioural flexibility

ABSTRACT:

Unlike the dorsal pallium that exhibits drastic evolutionary changes in cell number, type, and organization across vertebrates, the ventral subpallium is considered to be highly conserved for motor, cognitive, and emotional control. Here, we report the evolutionary emergence of specific striatal interneuron types – Parvalbumin-expressing (PVALB)⁺ and Thyrotropin-releasing hormone-expressing (TRH)⁺ – from spatiotemporally and molecularly defined progenitors in mammals to modulate flexible behaviour. In-depth comparative single-cell transcriptomics analyses revealed the existence of specific striatal GABAergic neurons in mice, but not lizards or salamanders. Spatial transcriptomics, lineage tracing, and genetic analyses delineated the unique progenitor origins and developmental trajectories of these interneurons specifically corresponding to PVALB⁺ and TRH⁺ interneurons in the striatum. Their disruption led to reduced synaptic inhibition, abnormal repetitive behaviour, impaired sensorimotor gating, and heightened anxiety, core phenotypes reminiscent of obsessive-compulsive disorder. These findings suggest that the mammalian striatum acquires specialized GABAergic interneurons to enhance inhibitory synaptic modulation and behavioural flexibility.

BIOGRAPHY:

Song-Hai Shi, Ph.D., received his undergraduate training at Tsinghua University in 1996 and obtained his Ph.D. degree on Genetics from Cold Spring Harbor Laboratory & State University of New York at Stony Brook, New York in 2001. He then completed his postdoctoral training at University of California at San Francisco in 2006. From 2006 to 2019, he served as Assistant Member/Professor, Associate Member/Professor and Full Member/Professor at the Memorial Sloan Kettering Cancer Center and the Weill Medical College of Cornell University in New York City. He is currently a professor at Tsinghua University and serves as the Dean of the School of Life Sciences and the Director of the IDG/McGovern Institute for Brain Research. Prof. Shi was elected as an academican of the Chinese Academy of Sciences in 2023. Dr. Shi has focused his research on identifying the common commodities of brain circuits at both the structural and functional levels, and linking them with animal behaviors, with the ultimate goal of arriving at a circuit- and system-level understanding of brain operation and function under normal and disease conditions. He has published numerous peer-reviewed articles in journals including Nature, Science, Cell, Nature Neuroscience, Neuron and National Review Science. His expertise and achievement have been recognized by a number of awards and supports, including the Science Breakthrough of the Year (1999), the Amersham Biosciences and Science Grand Prize for Young Life Scientists (2001), the Helen Hay Whitney Fellowship (2002), the Klingenstein Fellowship (2007), the McKnight Scholar Award (2010), the Blavatnik Award for Young Scientists (2010), the Howard Hughes Medical Institute (HHMI) Faculty Scholar Award (2016), the Beijing Outstanding Young Scientist (2019), the New Cornerstone Investigator (2022), and the Beijing Scholar (2023).





Kun Li

◆ Tsinghua University

TITLE:

Top-down regulation of adaptive social behaviors:
from instinct to impulse

ABSTRACT:

Social behavior is essential for survival and reproduction, but how internal states shape social interactions is not well understood. Our work shows that the prefrontal cortex plays a central role in this process. First, we identified *Cacna1h*⁺ neurons in the medial prefrontal cortex that track female estrous state. By integrating ovarian hormones with male social cues, these neurons drive estrus-dependent sociosexual behaviors through hypothalamic outputs, enabling females to adapt their preferences across the cycle. Second, using a new social threat paradigm, we found that the lateral orbitofrontal cortex transforms anxiety signal into maladaptive aggression. Excitatory ORBI neurons integrate anxiogenic thalamic inputs and recruit striatal outputs to drive stress-evoked attacks, whereas inhibitory ORBI neurons constrain aggression without affecting anxiety. Together, these findings establish prefrontal circuits as hubs that integrate hormonal and emotional states with social cues, providing mechanistic insight into how internal states flexibly shape social behavior and how their disruption may contribute to psychiatric disorders.

BIOGRAPHY:

Dr. Kun Li is a principal investigator at the IDG/McGovern Institute for Brain Research, School of Life Sciences at Tsinghua University, and Tsinghua-Peking Joint Center for Life Sciences. She received her Ph.D. from the Institute of Neuroscience, Chinese Academy of Sciences, China, under the mentorship of Dr. Hailan Hu. She then completed her postdoctoral training at The Rockefeller University with Dr. Nathaniel Heintz. Her lab focuses on understanding the neural basis of sex differences in social and emotional behaviors, particularly how hormones and emotional regulation interact with brain circuits to shape adaptive and maladaptive social behaviors in males and females.





SESSION 4

Somatosensation and Interoception II





John Wood

◆ University College London

TITLE:
Sodium channels and pain

ABSTRACT:

Pain is a massive problem, particularly amongst the elderly. The revolution in molecular genetics in the late twentieth century coupled with recent advances in protein structure determination has provided powerful insights into how pain works, as well as identifying analgesic drug targets. Peripheral sodium channels are plausible candidates. Today I will discuss recent successes with drugs targetting a voltage-gated sodium channel (Nav1.8), found only in the peripheral nervous system. Nav1.8 mutations have been linked to both pain and cardiovascular problems - even sudden death. The discovery of a role for a small C-terminal fragment of Nav1.8 in cardiac function enables drugs that avoid actions on the heart to be developed. A new orally active drug named Journavx or Suzetrigine is now in the clinic and promises to improve present pain treatment.

In terms of human validation, the sodium channel Nav1.7 is a more compelling target. Humans lacking Nav1.7 are apparently normal but pain-free. However, embryonic pain-free humans and mice lacking Nav1.7 show a distinct mechanism of analgesia from adult knock-out animals. Intriguingly, embryonic Nav1.7 gene deletion enhances endogenous opioid signalling in peripheral neurons, resulting in diminished neurotransmitter release. In contrast, adult gene deletion or channel blocking drugs diminish excitability. Nav1.7 is expressed broadly within the central nervous system, as well as in the autonomic nervous system and some non-neuronal tissues such as the pancreas. Small molecule drug side effects are always a problem, and the broad role of Nav1.7 particularly in the autonomic nervous system means that antagonists of Nav1.7 are toxic. In contrast, the interaction with the opioid system in embryonic nulls presents a fascinating potential new route to pain treatment.

As well as targetting ion channels, the cell populations expressing particular ion channels can be useful analgesic targets. Chemogenetic silencing of neurons expressing Nav1.8 is a highly effective route to causing analgesia in preclinical studies. In addition, neuro-immune interactions can be interrogated through studies of neuron-depleted mice.





BIOGRAPHY :

Professor John N. Wood graduated from Leeds University and studied Virology at Warwick University. He moved to the Institut Pasteur in 1976 to work as a postdoctoral fellow with Luc Montagnier on Interferons, before transitioning to Neuroscience thanks to meeting Tom Jessell. He worked for 12 years in Industry (Wellcome Foundation and Sandoz Institute) joining University College London (UCL) in 1996. He identified a number of new analgesic targets in studies of transgenic mice. An ATP-gated channel (P2X3) is the target of a drug called Gefapixant in honour of Geoff Burnstock. With Armen Akopian, he also cloned a sodium channel named Nav1.8 that is important for human pain. Vertex have produced an orally active channel blocker - Journavx or Suzetrigine that is a useful analgesic. John founded Ionix Pharmaceuticals, to make Nav1.8 channel blockers in 2002 without success. In 2009 he was elected to the Royal Society and won the Grand Prix Scientifique of the Institut de France. More recently, he and others showed that Nav1.7, another sodium channel, plays a key role in human pain, and explained the mechanism behind this, but the genetic basis of pain loss (with compensatory expression of other channels) does not translate into useful therapeutics because of bad side effects. His recent work focusses on chemogenetic studies of cancer pain.





Nieng Yan

◆ Shenzhen Medical Academy of Research and Translation

TITLE:

Structural Pharmacology of Na_v and Ca_v channels

ABSTRACT:

Voltage-gated sodium (Na_v) and calcium (Ca_v) channels are responsible for the initiation of electrical signaling. Being associated with a variety of disorders, Na_v and Ca_v channels are targeted by multiple pharmaceutical drugs and natural toxins. Taking advantage of the resolution revolution of single particle cryo-EM, we have determined the structures of different Na_v and Ca_v subtypes from human and other eukaryotes. These structures, alone or in complex with distinct auxiliary subunits, toxins, and drugs, not only afford unprecedented insights into the working and disease mechanism of these channels, but also reveal novel pharmacological sites. In light of these structural advances, we proposed a structure-based nomenclature for ligand binding sites on Na_v and Ca_v channels, which may facilitate rational drug design and optimization.

BIOGRAPHY:

Dr. Nieng Yan received her B.S. degree from the Department of Biological Sciences & Biotechnology, Tsinghua University, Beijing, China, in 2000. She then pursued her PhD in the Department of Molecular Biology at Princeton University under the supervision of Prof. Yigong Shi between 2000 and 2004. She was the regional winner of the Young Scientist Award (North America) co-sponsored by Science/AAAS and GE Healthcare in 2005 for her thesis on the structural and mechanistic study of programmed cell death. She continued her postdoctoral training at Princeton University, focusing on the structural characterization of intramembrane proteases. In 2007, she joined the faculty of School of Medicine, Tsinghua University. Her lab has been mainly focusing on the structural and functional study of membrane transport proteins. In 2012 and 2013, she was promoted to tenured professor and Bayer endowed chair professor, respectively. She returned to Princeton University as the founding Shirley M. Tilghman Professor of Molecular Biology in 2017. Five years later, she resigned from Princeton University and took on the post of the Founding President of Shenzhen Medical Academy of Research & Translation (SMART) as of December 1st, 2022. Meanwhile, she re-established her research program in School of Life Sciences in Tsinghua University as a University Professor. In March 2023, she was appointed the Director of Shenzhen Bay Laboratory.





BIOGRAPHY :

Dr. Yan's primary research interest has been in the structural and mechanistic investigation of membrane transport proteins that are of tremendous physiological, pathophysiological, and pharmaceutical significance. She reported the first structures of the human glucose transporters GLUT1 and GLUT3, the eukaryotic voltage-gated sodium and calcium channels, and a number of proteins involved in sterol metabolism. Her present research program focuses on structure-guided mechanistic understanding and drug discovery for pain relief.

Dr Yan's achievements have won her numerous accolades. She was an HHMI international early career scientist in 2012-2017, the recipient of the 2015 Protein Society Young Investigator Award, the 2015 Beverley & Raymond Sackler International Prize in Biophysics, the Alexander M. Cruickshank Award at the GRC on membrane transport proteins in 2016, the 2018 FAOBMB Award for Research Excellence, the 2019 Weizmann Women & Science Award, the 2021 Anatrache Membrane Protein Award by the Biophysical Society, and 2024 L'Oreal-UNESCO Award for Women in Science. Dr. Yan was elected as an International Member of the US National Academy of Sciences in 2019, an International Honorary Member of the American Academy of Arts and Sciences in 2021, a member of the Chinese Academy of Medical Sciences in 2022, an associate member of EMBO in 2023, and a member of the Chinese Academy of Sciences in 2023.





Juanjuan Du

◆ Tsinghua University

TITLE:

Targeting ion channels with
ligand-antibody conjugates

ABSTRACT:

Crucial for cell activities, ion channels are key drug discovery targets. Although small-molecule and peptide modulators dominate ion channel drug discovery, antibodies are emerging as an alternative modality. However, challenges persist in generating potent antibodies, especially for channels with limited extracellular epitopes. To address this challenge, we innovatively integrate the small molecule and antibody binding modes, thereby creating antibody derivatives capable of targeting both flat surfaces and deep pockets. Our initial endeavor revolved around leveraging antibody avidity to enhance ion channel targeting. Conventional monoclonal antibodies rely on intermolecular bivalent binding for avidity, but this diminishes with low receptor densities, especially for ion channels. To address this limitation, we rationalized that bi-epitopic intramolecular crosslinking of ion channels could yield antibody derivatives with enhanced avidity for these sparsely distributed membrane proteins. Extending the concept of integrating ligand and antibody for ion channel drug discovery, we further introduced the ligand into the antigen-binding domains of antibodies. Such strategy expands the chemical space of antigen-binding regions, thereby offering new opportunities to modulate difficult targets, such as ion channels. Overall, our strategies to integrate small molecule and antibody binding modes have the potential to modulate the function of ion channels, offering a promising avenue for addressing unmet medical needs.

BIOGRAPHY:

Professor DU Juanjuan got her Bachelor degree from the College of Chemistry and Molecular Engineering at Peking University and earned her Ph.D. from the University of California, Los Angeles (UCLA). She subsequently conducted postdoctoral research with Professor Peter G. Schultz at The Scripps Research Institute (TSRI) and California Institute for Biomedical Research (CALIBR). In 2016, she joined the School of Pharmaceutical Science at Tsinghua University, where her primary research focus is on pioneering the frontier of next-generation antibodies. By integrating bioconjugation techniques with protein engineering, she is dedicated to develop innovative chemical and protein engineering technologies to modulate the function, clustering state, and endocytosis of cell surface proteins, thereby providing new opportunities to generate new modalities for therapeutic applications.





Yulong Li

◆ Peking University

TITLE:

Orphan GPCRs, chronic liver diseases and itch:
from bench to bedside

ABSTRACT:

Orphan G protein-coupled receptors (GPCRs) have emerged as critical players in various physiological and pathological processes, yet many remain poorly characterized. Chronic liver diseases, such as intrahepatic cholestasis of pregnancy (ICP), often manifest with persistent itching that severely impacts patients' quality of life. Our recent studies identified human MRGPRX4 (hX4)—originally described as an orphan GPCR—as a key itch receptor that mediates cholestasis-associated pruritus. Through a comprehensive metabolite library screen, we discovered that progesterone sulfates selectively activate hX4 in vitro and in vivo, triggering ICP-related itching. Furthermore, elevated progesterone sulfate levels not only correlate with pruritus severity but also accurately predict ICP onset months before clinical diagnosis. These findings highlight hX4 as actionable therapeutic target in cholestatic and other chronic liver disorders accompanied by pruritus. From mechanistic insights at the bench to biomarker-driven interventions at the bedside, this work underscores the potential of orphan GPCR research to transform the diagnosis, early prediction, and treatment of chronic liver diseases.

BIOGRAPHY:

Dr. Yulong Li, a professor at the School of Life Sciences, Peking University, is a researcher at the PKU-THU Center for Life Sciences, IDG/McGovern Institute for Brain Research at PKU, New Cornerstone Science Laboratory. He got his B.S. from Peking University and his Ph.D. in Neurobiology from Duke University, followed by postdoctoral research at Stanford University. Since 2012, he established his lab at Peking University. His research centers on the 'synapse', the fundamental unit for the communication between neurons. He carries two layers of research: first, he develops cutting-edge research tools, namely advanced imaging probes, to untangle the complexity of the nervous system in space and in time; second, capitalizing on the advancement of research toolkits, he studies the regulation of synaptic transmission, focusing on the modulation of presynaptic transmitter release in health and disease conditions. His research group has successfully developed a series of novel genetically encoded optical probes called GPCR Activation-Based (GRAB) sensors for imaging neuromodulators such as acetylcholine, monoamines, purines, lipids, and neuropeptides. These probes have allowed, probably for the first time, rapid, chemical- and cell-specific in vivo detection in multiple organisms ranging from flies, zebrafish, and mice to songbirds.





Ru-Rong Ji

◆ Duke University Medical Center

TITLE:

Neuroglial network in acute and chronic pain

ABSTRACT:

Pain is essential for survival, serving as a protective response to external noxious stimuli and as a potential indicator of internal physiological states. However, chronic pain affects approximately 20% of the global population, and current treatments remain limited in both efficacy and safety. Over the past two decades, research has highlighted the critical role of glial cells in maintaining nervous system homeostasis, with accumulating evidence showing that glial dysregulation contributes to the development and persistence of chronic pain. In this presentation, I will present evidence that glial cells—such as astrocytes in the central nervous system (e.g., spinal cord) and satellite glial cells in the peripheral nervous system (e.g., dorsal root ganglia)—form dynamic networks with nociceptive neurons, exerting profound influence on neuronal activity and pain states. I will also discuss emerging non-opioid therapeutics for managing acute and chronic pain, with a focus on targeting GPCRs selectively expressed in glial cells and nociceptive neurons.

BIOGRAPHY:

Dr. Ji is William Maixner Professor of Anesthesiology and the Director of the Center for Translational Pain Medicine at Duke Medical Center. He earned his PhD from Shanghai Institute of Physiology, Chinese Academy of Sciences and had postdoctoral training in Peking University, Karolinska Institute, and Johns Hopkins University. Before joining Duke University, Dr. Ji served on the faculty at Harvard Medical School for over a decade. He has authored over 270 papers in leading scientific journals. Over the past 7 years, Dr. Ji has been recognized in the global list of Highly Cited Researchers by Clarivate, with an H-index of 124 and a total citation count of 52,000 citations on Google Scholar. Dr. Ji has mentored more than 100 scientists, many of whom now lead independent labs. He serves on the editorial boards of Journal of Neuroscience, Anesthesiology, Pain, JCI, and Physiological Reviews and is Co-Chief Editor of Neuroscience Bulletin. A globally recognized leader in pain and anesthesiology research, Dr. Ji has made pioneering contributions to our understanding of how glial cells regulate acute and chronic pain through neuro-glial interactions. His work has profoundly shaped current concepts of pain mechanisms and identified numerous therapeutic targets, paving the way for the development of novel pain treatments.





Bailong Xiao

◆ Tsinghua University

TITLE:

Mouse behavioral genomics identifies a gatekeeper of somatosensation

ABSTRACT:

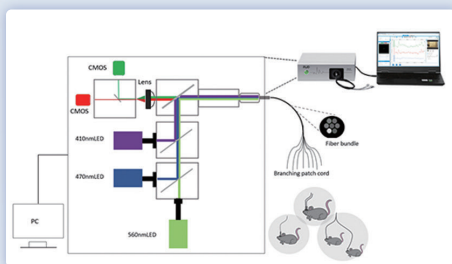
Somatosensation enables the perception of touch, temperature, pain, and itch. These sensory modalities are mediated by primary sensory neurons in the dorsal root ganglion (DRG), which convert physical and chemical stimuli into electrical signals using specialized molecular sensors—such as the touch sensor PIEZO2 and the temperature sensor TRPV1—along with downstream voltage-gated sodium channels like Nav1.7. However, the full repertoire of molecular components involved in somatosensory processing remains incompletely identified. Here, we developed an efficient postnatal CRISPR-Cas9 knockout platform to screen DRG-expressed genes via AAV9-sgRNA delivery. Combining this approach with behavioral assays of somatosensory responses, we validated the roles of PIEZO2 and TRPV1 in sensing gentle touch and noxious heat, respectively, and revealed previously uncovered broad involvement of Nav1.7 in distinct somatosensory modalities. Remarkably, a targeted screen of DRG-expressed genes identified a master regulator of somatosensation. Either sgRNA-mediated postnatal knockout or tamoxifen-induced Cre-mediated deletion in DRG neurons resulted in profound behavioral deficits in touch, temperature, pain, and itch perception, while its overexpression enhanced touch and thermal responses. Mechanistically, it functions as a novel auxiliary regulator of voltage-gated sodium channels for controlling the excitability of DRG neurons. Together, this work establishes a robust postnatal screening platform for somatosensory genomics, identifies a master regulator of somatosensory function, and provides novel therapeutic strategy for pain and itch treatment.

BIOGRAPHY:

Dr. Bailong Xiao is a Professor at the School of Pharmaceutical Sciences in Tsinghua University, as well as a Principal Investigator at the Tsinghua-Peking Center for Life Sciences, the IDG/McGovern Institute for Brain Research, and the Beijing Advanced Innovation Center for Structural Biology. His lab has established a multidisciplinary research program to advance the understanding of the structure-function relationships as well as physiological and pharmacological regulations of the mechanically activated PIEZO channels; and also aimed to identify novel receptors and regulators involved in somatosensation.



Multichannel Fiber Photometry Promotes Neuroscience Research



Fiber photometry system characterizes the change of neuronal population activity by recording the fluorescence intensity change of population neurons in specific brain regions, which can be used to monitor the population neurons of freely moving animals stably for a long period of time, and it is an effective technical means to understand the correlation between neuronal activity and animal behavior. Fiber photometry system has been published more than 7000 articles, and is currently rising at a rate of more than 500 new articles per year, favored by more and more researchers.

Technological Advantages

Cell Specificity

Sensors are expressed or imported into specific cell populations to record type-specific neuronal population activity.

Sensor Diversity

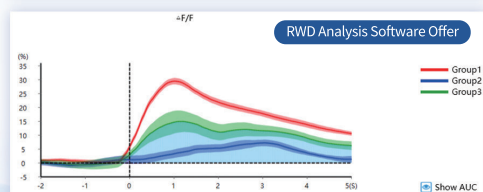
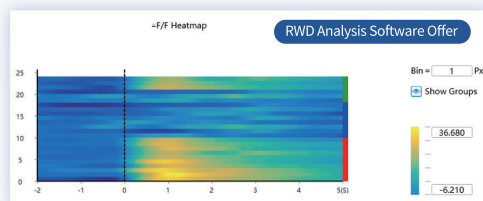
Various types of sensors are available, which can be freely selected according to the needs of experimental design.

Multi-site Synchronous Recording

Meet the synchronous recording of multiple brain regions or animals to enhance the efficiency of experiments.

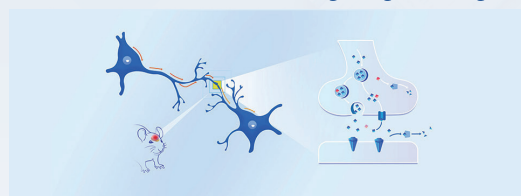
Long-term Stable Recording

TCSPC-based optical fibers are ultrasensitive tools for detecting emitted light, meeting the requirement that fluorescence can be detected at low intensities of excitation light, reducing photobleaching and extending recording time.

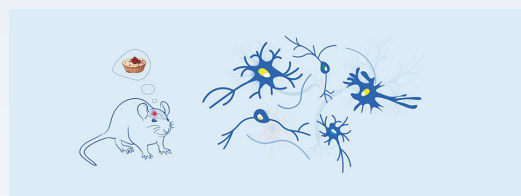


Application Scenarios

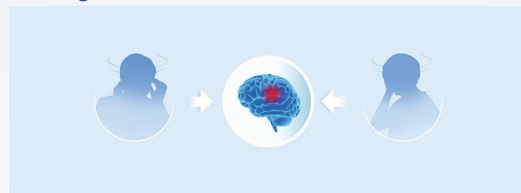
Calcium Ion and Neurotransmitter Signaling Recordings



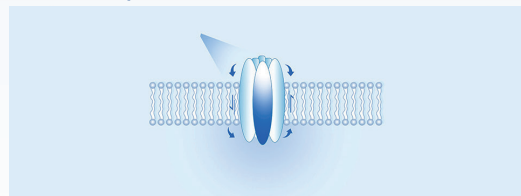
Neural Circuit Research



Neurological Disease Research



Probe Development Detection



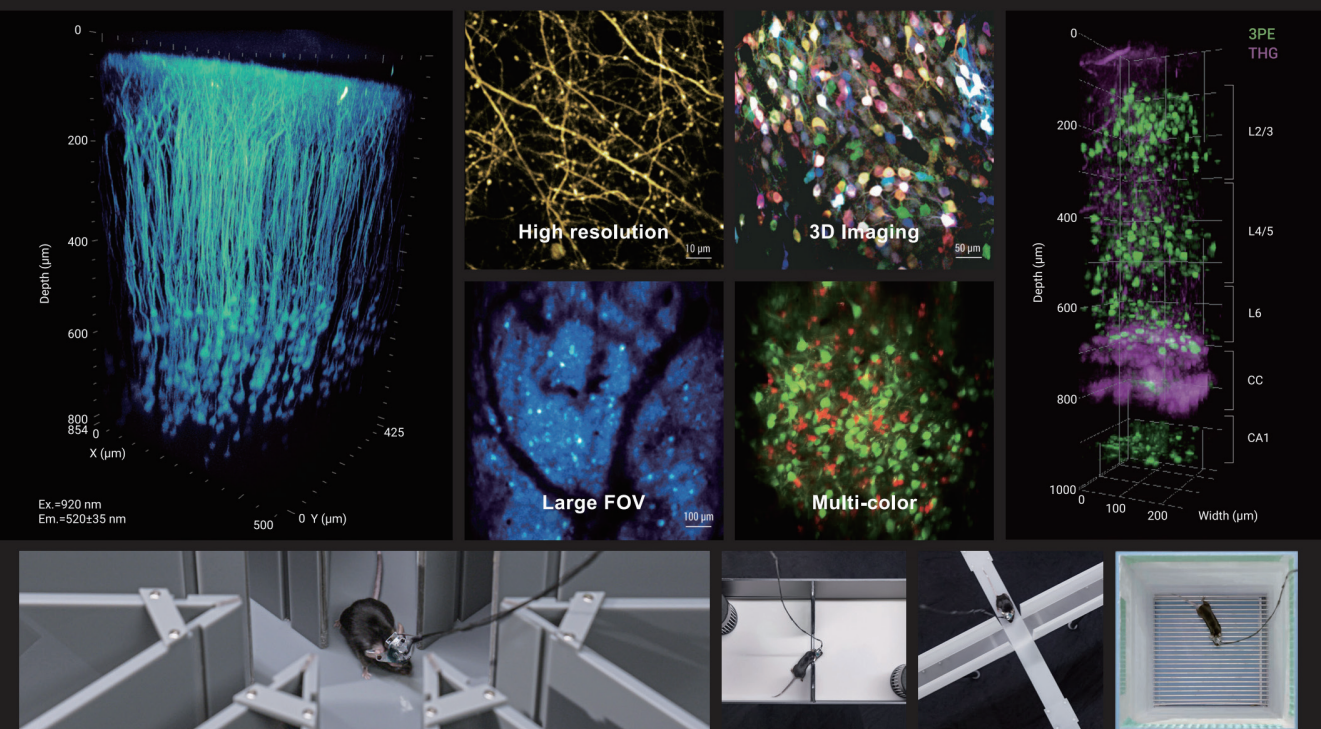
◀ Scan Code for Free Trial



「神经科学多光子显微成像解决方案」

超维景生物科技有限公司是一家专注于高端生物医学成像设备研发、生产和销售的国家高新技术企业。始创于2016年，公司核心力量来自北京大学院士创建和领导的多学科交叉团队。团队攻克了多项关键核心技术，于2017年成功研制探头仅重2.2g的超高时空分辨率微型化双光子显微镜，国际获取了小鼠在自由行为过程中大脑皮层神经元和神经突触清晰、稳定的动态图像。随后，超维景公司成功完成了显微镜的产品化，解决了包括探头、特种光纤、飞秒激光器、微型化物镜等核心技术部件的国产化瓶颈，实现了自主生产一系列微型化双光子显微成像系统，广泛应用于国内外脑科学的研究。

这项技术进一步提升和拓展，目前已应用于人体皮肤细胞在体检测，并通过与传统内窥镜相结合，可以进一步提升细胞成像功能，形成多模态内窥诊断系统。超维景以科技创新为己任，研精致思，奋力为全球生命科学和临床医学在体成像的高速发展做出卓越贡献。



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E-mail: info@tvscope.cn
Website: www.tv-scope.com



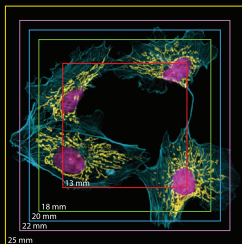
超维景官方



超维景科研解决方案

Data that delivers

In the age of big data, it is important that key laboratory instruments such as the confocal microscope keep pace with experimental demand. Here, we discuss factors to consider that can maximize the throughput of your confocal instrument.



Actin (cyan), mitochondria (yellow), and nuclei (magenta).

Field of view (FOV)

A critical factor in confocal imaging throughput is the area of a single image field. A lower-magnification objective can image a larger field, but generally has lower resolving power, as measured by its numerical aperture (NA).

Optical systems have a magnification-independent, maximum field of view (FOV). Beyond this, the image quality rapidly degrades. The image to the left compares the FOV of different confocal instruments.



25-mm FOV



Danio rerio 2-day old embryo

Dorsal view of bones and scales (blue) and lymphatic vessels (orange) in a juvenile zebrafish.

GLOSSARY

Deconvolution: the use of an iterative algorithm for reassigning out-of-focus light in a 3D dataset back to the correct plane/location to improve the signal-to-noise ratio (SNR) and available resolution.

Numerical aperture (NA): a measure of the resolving power of a lens, calculated using the formula $NA = n \sin \theta$, where n is the refractive index of the immersion medium and θ is half the angular aperture of the lens.

Nyquist sampling criterion: requires that an analog signal (the image) be sampled at a rate at least twice the highest frequency present (twice the resolution limit of the microscope). In practice, at least $\sim 2.3\times$ sampling is recommended.

Peak quantum efficiency (QE): QE is the percentage of incident photons that are successfully converted into photoelectrons by the detector. It is spectrally dependent, so manufacturers usually report the peak QE at a single value.

Point-scanning confocal: the confocal mode that scans the focus of a laser beam in the x-y plane, imaging each point in the field. Emission light from each point is decoupled and passes through a pinhole aperture in a conjugate image plane prior to reaching the detector. Out-of-focus light is mostly blocked by the pinhole, thus providing optical sectioning.

Point-spread function (PSF): the 3D diffraction pattern obtained by imaging an infinitely small (subresolution) object. The PSF can be thought of as a blurring function—its dimensions limit the colorable resolution.

Rayleigh criterion: defines two objects as being optically resolved when the center of the diffraction pattern of one object overlaps with the first minimum of the other.

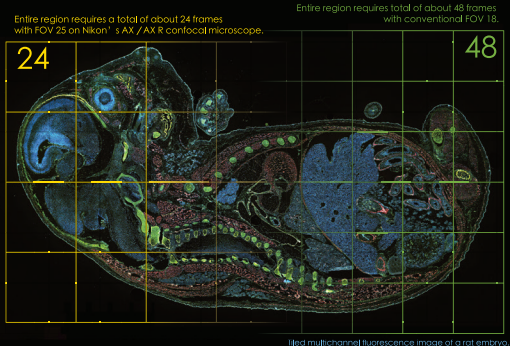
Resonant scanners: point-scanning confocal systems that use a resonant scanning mirror for the fast scan direction. Resonant scanning mirrors vibrate at a fixed, high-frequency rate. Galvano scanning mirrors are driven by an adjustable sawtooth signal, and scan speed is more limited by inertia.

Signal-to-noise ratio (SNR): the ratio of the detected signal to noise. Noise may originate either from quantum fluctuations in the number of emitted photons (which follows a Poisson distribution) or from the detector.

Spinning disk confocal: instead of scanning a single point, these systems use an array of confocal pinholes imaged onto the sample. The pinholes are positioned in a disk in a conjugate image plane—rotating the disk many times within a single camera exposure allows for the entire field to be evenly sampled by the pinholes, providing optical sectioning.

Tiled acquisition

When the entire region of interest cannot be captured in a single image field, overlapping, “tiled” images can be acquired and merged into a single file. The FOV determines the number of tiled images needed to capture the entire specimen. Decreasing the number of locations imaged can significantly shorten the overall acquisition time.



Tiled multichannel fluorescence image of a rat embryo.

Optimizing resolution across the FOV

To realize optimal performance at full resolution, it is important to achieve a pixel density that meets the Nyquist sampling criterion. Sufficient sampling can be an issue with lower-magnification objectives. The FOV divided by the total magnification gives the usable field size (at the specimen plane). Once an objective is selected, the theoretical resolution limit can be estimated using its NA and a metric such as the Rayleigh criterion.

The table shows how well a point-scanning system with a 25-mm FOV satisfies the Nyquist criterion with various low-magnification objectives (8,192 × 8,192-pixel resolution, 25-mm FOV, no scan zoom applied).

The NA determines the lens resolving power, calculated using the Rayleigh criterion (row 3). The back-projected virtual pixel size (last row) for the stated conditions should be less than or equal to the Nyquist sampling value in order to be within the range for imaging with the full resolving power (NA) provided by the lens.

Objective Magnification (dry)	1X	2X	4X	10X	20X	40X
Numerical Aperture (NA)	0.04	0.1	0.2	0.45	0.75	0.95
Rayleigh Resolution Limit (nm, $\lambda = 546$ nm)	8.33 μ m	3.33 μ m	1.67 μ m	0.74 μ m	0.44 μ m	0.35 μ m
Nyquist Sampling (2.3x, $\lambda = 546$ nm)	3.62 μ m	1.45 μ m	0.72 μ m	0.32 μ m	0.19 μ m	0.15 μ m
Minimum Effective Pixel Size (25-mm FOV)	2.16 μ m	1.08 μ m	0.54 μ m	0.22 μ m	0.11 μ m	0.05 μ m

EXAMPLE

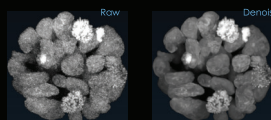
Sensitivity

The signal collected from a specimen and the sensitivity of the detector impact the practical acquisition rate more than the maximum possible scan speed or camera frame rate do. Pairing a spinning disk system with a scientific complementary metal-oxide semiconductor (sCMOS) detector—the latest feature peak quantum efficiency (QE) values of $\sim 95\%$ —can achieve close to single-photon sensitivity. Point-scanning instruments rely on single-element detectors known as “photomultiplier tubes” which generally have a QE lower than that of the latest sCMOS cameras.

Camera images contain three principal noise sources: the camera readout, dark current, and shot noise. The signal-to-noise ratio (SNR)

can be improved with post-acquisition analyses such as denoising and deconvolution. Nikon’s unique Denoise AI artificial intelligence software uses a pretrained neural network to rapidly denoise resonant-scanning confocal data, which is mostly affected by shot noise.

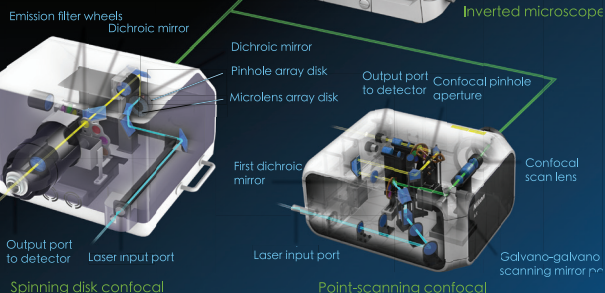
Deconvolution uses knowledge of the system’s point-spread function to reassign out-of-focus light to its correct location. This can significantly improve SNR, even with confocal data where most out-of-focus light is eliminated by the pinhole aperture(s). Data for deconvolution can also be prefiltered using denoising analysis.



Raw versus denoised (using Nikon’s Denoise AI) resonant confocal image of Caco-2 cells stably expressing H2B-GFP, cultured in Matrigel.

Optical path

Learn more about Nikon’s range of confocal microscopes



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