Picower Institute for Learning and Memory

The Picower Institute for Learning and Memory is a world-class focal point for research and education in the neuroscience of learning and memory. Learning and memory are central to human behavior and the Picower Institute's research aims to understand the mechanisms underlying these cognitive functions at the molecular, cellular, brain circuit, and brain systems levels. The Picower Institute's research also extends to other higher-order cognitive phenomena intimately associated with learning and memory, such as attention, decision making, and consciousness.

Awards and Honors

Li-Huei Tsai was elected as a fellow of the American Association for the Advancement of Science, for "studies of cellular mechanisms of learning and of learning disruptions in Alzheimer's disease."

Mark Bear was invited to address the Congressional Biomedical Research Caucus on March 30, 2011.

Both Time and Forbes magazine featured Bear's work on autism that suggests how a specific class of drug, which is already on the market, could help with fragile X syndrome.

Susumu Tonegawa received the David M. Bonner Lifetime Achievement Award from his alma mater, University of California, San Diego, in November 2010.

Research Breakthroughs

Major research advances in Picower Institute faculty laboratories during the report period are summarized below.

Tonegawa's laboratory discovered a novel electrophysiological mechanism in the mammalian hippocampus (called preplay) by which encoding of the information from a novel experience is aided by patterns of neuronal activity already present in the hippocampus.

Tsai and her colleagues identified HDAC2 as the major histone deacetylase that regulates synaptic plasticity and memory formation.

Troy Littleton's laboratory identified key mechanisms underlying the postsynaptic secretion of retrograde factors that control activity-dependent synaptic growth.

One of the new discoveries in Earl Miller's laboratory includes insights into of neural underpinnings for the well-documented finding that the average person can only retain about four items in working memory at a time.

Elly Nedivi's laboratory found that experience can sculpt mature inhibitory circuits by remodeling dendrites of superficial L2/3 interneurons in an input-specific manner.

Personnel

In addition to 10 faculty members, the Picower Institute consists of other researchers, students, and technical and administrative support personnel. More than 230 community members participated in Picower Institute activities during the report period: 10 faculty members, 12 senior researchers, 10 visiting scientists/scholars, 58 postdoctorates, 58 undergraduates, 21 graduate students, 64 research and technical staff, and 19 administrative and service staff.

Items of note during the academic year included the following:

Kay Tye accepted a junior faculty position within the Picower Institute (and the Department of Brain and Cognitive Sciences) as an assistant professor.

Myriam Heiman started January 16, 2011, as a junior faculty member and continues to hire and set up her lab.

Andrew Devlin, Tsai's assistant, departed the Howard Hughes Medical Institute and the Picower Institute to pursue his passion for film in New York City.

Shawn Hennessey joined the Picower Institute in March2011 as Tsai's new assistant. Shawn brings with him experience as an assistant at Brigham and Women's Hospital.

Resource Development

Resource development continues to be a high priority at the Picower Institute. In FY2011 many Picower Institute faculty gave their time for prospect and donor meetings, both at MIT and in the community.

Several resource development events were held to build awareness and donor interest in the Picower Institute faculty and their work. The Department of Brain and Cognitive Sciences hosted "An Afternoon with MIT's Brains on Brains" symposium featuring Professor Tsai and her work on Alzheimer's disease, as well as Assistant Professor Heiman and her talk titled "Parkinson's Disease: Current State and Hope for the Future." Breakout sessions were also led by Professor Tsai on post-traumatic stress disorder and by Bear on autism and developmental disorders. More than 100 people attended. Follow-up stewardship was conducted for all prospect visits and events.

New leadership gifts to the Picower Institute included individual contributions of $67,000, $60,000, and $20,000 to Tsai's lab for Alzheimer's research; $50,000 to Bear's lab; $30,000 to support Littleton's lab; $10,000 to support Nedivi's lab; and several smaller gifts from new prospects.

Media Recognition

The Picower Institute issued eight MIT press releases during the reporting period. Articles appeared in the Boston Globe, Science Daily, and Technology Review. Picower Institute research breakthroughs were also reported on dailymail.co.uk and the-scientist.com.

Programs and Activities

Collaboration among disciplines is an integral component of the Picower Institute's research philosophy. To facilitate these interactions, the Picower Institute follows a rigorous calendar of formal lectures, conferences, and workshops, in addition to hosting informal events. Activities are designed to bring Picower researchers and the MIT neuroscience community together with other neuroscientists and practitioners from public and private sectors to exchange research findings, facilitate cross-disciplinary collaborations, and continue to explore the potential that advances about learning and memory mechanisms offer to science and society. Ongoing programs and activities are described below.

Held annually, the Picower Lecture was named to honor and recognize the Picower Foundation's generous support of the neurosciences at MIT. Each lecture features the work of a current leader in brain research. This year's lecturer was Dr. Richard Huganir from The Johns Hopkins University in Baltimore, MD. His talk, "Receptors, Synapses, and Memories," was delivered May 19, 2011.

The Picower Institute colloquia bring together the highest caliber of learning and memory researchers from universities throughout the world to share their findings and experiences with the MIT community and to create working relationships with members of the Picower Institute. During the past year, colloquia speakers were: Dr. Morgan Sheng of Genentech, Drs. Alfredo Kirkwood and Ed Connor of The Johns Hopkins University, Dr. Marla Feller of University of California Berkley, Drs. Azad Bonni and Beth Stevens of Harvard University.

In the language of neuroscience, "plasticity" refers to the minute but crucial physical changes that take place in our synapses every time we learn, experience, or remember anything new. At the Picower Institute, "Plastic Lunch" refers to a biweekly series of informal talks during the academic year that gives postdoctorates and graduate students from across the Picower Institute a chance to share their latest, often prepublished, research with colleagues. The Plastic Lunch series provided an opportunity for participants to improve their presentation skills and also fostered collaboration and built new relationships across disciplines and between laboratories.

An endeavor targeted to the Picower Institute's postdoctorate community provided resources to support activities that build community and enrich interactions between postdoctoral colleagues and future associates. The postdoctorates convened a series of informal talks and social events and continued to maintain a website detailing their research interests and community activities.

A monthly faculty lunch, known as Picower Power Lunch, allowed faculty and guest speakers to informally relate recent research findings or present a new idea.

After the close of each academic year, the Picower Institute hosts an annual retreat for its community members. The fourth annual Dana and Betty Fisher Retreat of the Picower Institute for Learning and Memory was held June 15 and 16, 2011. More than 130 researchers attended the event in South Yarmouth, MA. The retreat included eight

laboratory research presentations, a highly interactive poster session (24 submissions), and a keynote address by renowned neurobiologist Michael E. Greenberg of Harvard University.

Research Initiatives

The induced pluripotent stem cell (iPS) core facility within the Brain and Cognitive Sciences complex was completed in November2010. Tak Ko, a researcher from Michigan, with years of iPS research knowledge, was successfully recruited to manage the new iPS facility in May 2011. This core facility will integrate the various research goals of members of the Picower Institute, the McGovern Institute for Brain Research, and the Department of Brain and Cognitive Sciences. Members' expertise with different experimental protocols, when combined collaboratively to study human cells, will result in accelerated progress in this novel, dynamic, and competitive field. This exciting resource is now available to MIT researchers.

The fate of human fibroblast cells can be changed by introducing some genes of interest, such as OCT4 (Octamer-4), NANOG (Nanog homeobox), SOX2 (SRY-related HMG-box gene 2), cMYC (proto-oncogene myc), and KLF4 (Kruppel-like factor 4), into pluripotent stem cells that can be called induced pluripotent stem cells. These iPS cells resemble embryonic stem cells. Various patient-derived skin primary fibroblast cells are being used to make iPS cells. These patient-specific iPS cells allow researchers to examine a variety of fundamental questions about human disease, which cannot easily be done with embryonic stem (ES) cell technology because the health status of ES cells from a given embryo is generally unknown. Therefore, this iPS cell technology creates a powerful research tool that will enable researchers to study disease processes to which they previously had limited access. The iPS cells allow for the creation of cell lines that are genetically customized to a patient; thus, potentially overcoming the issue of immune rejection. The iPS cells can be used to conduct patient-specific novel therapeutic drug screening and to study the mechanism of multiple neuropsychiatric and neurodegenerative disorders.

The Viral Vector Core Facility was launched in the fall of 2008 by the Picower Institute, in partnership with the McGovern Institute and with the support of an anonymous donor. Dr. Rachael Neve serves as facility director. Designed to become a self-supporting service facility in three years, the facility is a resource for MIT's neuroscience community, and it is now available to others across MIT and externally.

Viral gene delivery is a powerful adjunct to the use of transgenic mice for sophisticated manipulations of neuronal function. The facility initially offered modified herpes simplex virus (HSV) for delivery of genes into neurons in the brain, a resource not available anywhere else in the world. Two additional viral vectors with features that complement those of the HSV vector were developed by Dr. Neve, and a fourth is in the pipeline. This technology allows research laboratories to answer, in a uniquely direct way, basic questions about how specific types of neurons contribute to brain function and behavior.

The facility has attracted many users from MIT with diverse research interests; all are members or affiliates of the Picower Institute or the McGovern Institute. More than three different genes for investigators in the McGovern Institute and over 10 different genes for investigators in the Picower Institute have been sub-cloned into the three viral vectors currently available and packaged into virus. The use of these viruses to understand memory and cognition will provide the basis for new treatments of neurological and psychiatric disorders.

Faculty Research Summaries

Li-Huei Tsai's laboratory uses a combination of molecular/cellular, genetic, and behavioral approaches to study Alzheimer's disease and psychiatric and developmental disorders. Tsai's lab developed an innovative mouse model exhibiting the onset of Alzheimer's symptoms in a fraction of the time previously possible. Using this model, she has explored novel therapeutic approaches to combat cognitive impairment as the consequence of neurodegeneration. Tsai and colleagues reported a remarkable recovery of long-term memories by housing the mice in an enriched environment or treating them with nonselective histone deacetylase (HDAC) inhibitors that induce chromatin remodeling. She and colleagues identified HDAC2 as the major histone deacetylase that regulates synaptic plasticity and memory formation. Further experiments suggested that HDAC2 serves as the major target for the nonselective HDAC inhibitors in facilitating learning and memory and that HDAC2 expression may be correlated with cognitive impairment in Alzheimer's disease. The Tsai group also has found that HDAC2 down-regulation is by itself sufficient to restore cognition in a mouse model of Alzheimer's disease. These studies pinpoint HDAC2 as a promising target for therapeutic intervention.

Tsai also studies another class of deacetylases known as the sirtuins. She reported a role for sirtuin 1 (SIRT1) in modulating cognition. This finding supplements her earlier work, which showed a role for SIRT1 in neuroprotection, and uncovered a novel mechanism by which SIRT1 influences synaptic plasticity and memory through regulation of a microRNA, miR134. In her continuing effort to elucidate the pathways underlying neurodegeneration, Tsai has also revealed an important functional interaction between HDAC1 and SIRT1 in the maintenance of genomic integrity following neurotoxic insults such as oxidative stress and amyloid pathology.

Tsai has also made major advancements in understanding the biology of neuropsychiatric disorders. She found an interaction between the schizophrenia candidate gene DISC1 and Wnt signaling in the regulation of neural progenitor proliferation that provides fundamental insights into the role of brain development in schizophrenia. In collaboration with Chris Moore at MIT and Karl Deisseroth at Stanford University, Tsai used optogenetics to drive the activation of parvalbumin-positive interneurons in the somatosensory cortex, and provided the first causal evidence for the induction of distinct network activity states by activation of a specific cell type in the brain. Her work continues to incorporate in vivo optogenetics as her lab attempts to restore memory deficits resulting from neurodegeneration.

In Mark Bear's laboratory, work relates to the proposition that modification of synapses by neural activity is the substrate for experience-dependent brain development, learning, and recovery of visual function after brain injury. The effectiveness or "strength" of synaptic transmission can be persistently modified in response to defined patterns of pre- and postsynaptic activity. Well-studied examples of this type of synaptic plasticity are long-term potentiation and long-term depression. Can we exploit the current understanding of these mechanisms in order to strengthen brain connections that may have been weakened or impaired by sensory deprivation, disease, or injury? Theoretically motivated research in the visual cortex has suggested ways to promote synaptic potentiation. The theoretical concept is that the type and extent of synaptic plasticity caused by patterns of activity depend critically on the recent prior history of synaptic or cellular activity. Studies in visual cortex strongly support this concept and have suggested a mechanism for "metaplasticity"—the plasticity of synaptic plasticity—based on activity-dependent modification of N-methyl-d-aspartate (NMDA) receptor structure and function. The knowledge gained by these studies suggests ways in which recovery of function can be promoted, which are now being tested in the Bear lab.

Myriad mechanisms have been suggested to account for the full richness of visual cortical plasticity. In collaboration with Mriganka Sur's lab, the Bear lab previously found that visual cortex lacking the protein Arc is impervious to the effects of deprivation or experience. The remarkable new view that emerges from these studies of visual cortex is that by adolescence, excitatory synapses are rendered essentially immutable by experience or deprivation if Arc is not expressed in their postsynaptic target. Despite this profound defect in acquired properties, the innate organization and levels of visual responsiveness appear to be normal in Arc knockout mice. It appears that a requirement for Arc paints a bright line that separates the contributions of "nurture" (those dependent on the quality of sensory experience) from "nature" (those dependent on genetic instructions alone) on the development of glutamatergic synaptic connections in the cortex.

Fragile X syndrome is the leading inherited cause of mental retardation and autism. The Bear group's advances in mechanistic understanding of the disease have led to the identification of the metabotropic glutamate receptor (mGluR) as a therapeutic target for the disease. These studies have revealed that core defects in multiple animal models can be corrected by down-regulation of mGluR5 signaling. Although it remains to be seen whether mGluR5 antagonists or related approaches will succeed in humans with fragile X, the progress in this area stands as a strong testament to the power of applying knowledge of basic neurobiology to understand pathophysiology in a genetically validated model of human psychiatric disease.

Since arriving in January 2011, Myriam Heiman has been building her research group at the Picower Institute. Her new laboratory uses molecular, cellular, and behavioral approaches to study the molecular mechanisms underlying neurodegeneration. Among the tools she uses are transgenic mice that allow molecular studies of individual nerve cell populations. She has begun projects aimed at understanding the basis of cell-type-specific vulnerability in Huntington's disease. Working with two distinct mouse models, she has identified molecules that are enriched in the most vulnerable cells in

Huntington's disease and has begun to test their role in the pathophysiology of the disease. Additionally, she has been continuing collaborative work focused on elucidating the molecular alterations underlying the development of dyskinesias in Parkinson's disease, a collaborative project between the research groups of Dr. Paul Greengard (The Rockefeller University), Dr. James Surmeier (Northwestern University), and Dr. Angela Cenci (Lund University).

Troy Littleton's laboratory studies the mechanisms by which neurons form synaptic connections, how synapses transmit information, and how synapses change during learning and memory. The lab also studies how alterations in neuronal signaling underlie several neurological diseases, including epilepsy, autism, and Huntington's disease. Recently, the lab has identified key mechanisms underlying the postsynaptic secretion of retrograde factors that control activity-dependent synaptic growth. In addition, the lab has characterized how endosomal trafficking of activated synaptic growth receptors in the presynaptic terminal controls synaptic wiring downstream of retrograde signals. New synapse formation and synaptic rewiring are key elements of plasticity in the developing and adult brain. Similar to many species, modulation of synapse formation in Drosophila has been implicated in learning and memory. Previous work has suggested that several known autism-causing mutations identified in humans alter endosomal trafficking, implicating this pathway in human disease. The lab is characterizing one of the autism-linked endosomal trafficking regulators to define how dysfunction of endosomal processing of synaptic growth signals may link to autism. Together, these studies are beginning to define the molecular mechanisms by which neuronal activity modifies synaptic connections and how this process is disrupted in neurodevelopmental diseases.

The overarching goal of Earl K. Miller's laboratory is to understand cognitive functions in a broader context, as a product of interactions between networks and circuits of neurons, brain areas, and systems. To this end, the Miller lab has developed (and shared) technology and techniques for recording from many separately movable, acutely inserted electrodes, which allows for bridging the gap between the global scope of human brain imaging and the spatiotemporal precision of single neuron physiology. It also allows examination of precise timing relationships and interactions between neuronal populations. The lab couples this with the kind of sophisticated, flexible, rule-based behaviors at which humans and monkeys are so adept.

In the past year, the Miller laboratory has made a number of discoveries, including insights into the neural underpinnings for the well-documented finding that the average person can only retain about four items in working memory at a time. Researchers simultaneously recorded from neurons in the prefrontal and the parietal cortex of monkeys trained on a human capacity limitation task. Like humans, monkeys could only hold an average of about four objects in working memory. But neural recordings revealed that the monkeys had two independent capacities of two objects each in the right versus left half of vision, and that the limiting factor occurred during encoding, not during memory retrieval, suggesting that brain's two cerebral hemispheres have independent pools of neural information. This finding has implications for brain games to improve cognition and design of heads-up displays.

Elly Nedivi's laboratory studies plasticity of neural connections in the adult brain using molecular tools in combination with in vivo imaging of neuronal structure. Using multiphoton microscopy, they were able to visualize and reconstruct entire dendritic arbors of layer 2/3 neurons in visual cortex and monitor their dynamics over weeks and months. They found that experience can sculpt mature inhibitory circuits by remodeling dendrites of superficial L2/3 interneurons in an input-specific manner. Sensory deprivation leads to a retraction of inhibitory interneuron dendrites, resulting in a reduced inhibitory tone permissive for further structural and functional adaptation. Global disinhibition by the antidepressant fluoxetine can provide a permissive environment similar to that afforded by sensory deprivation, allowing for enhanced structural plasticity. Consequently, ocular dominance plasticity in the adult can occur in response to a brief monocular deprivation such as in the juvenile brain. These findings suggest that therapeutic approaches that reduce cortical inhibition, when combined with an instructive stimulus, could prove effective in enhancing cognitive abilities and restoring function to fully developed circuits impaired by neurological damage or disease.

Mriganka Sur's laboratory uses cutting-edge technologies for imaging cells and molecules in the intact brain in order to reveal their roles in synaptic plasticity and cortical function. Combined with novel probes, these methods have revealed unexpected mechanisms of cortical plasticity, the role of specific cell classes in cortical circuits, and mechanisms of brain disorders. In the past year, his lab identified a subset of microRNAs whose expression is differentially regulated by visual experience and showed that inhibition of one of the miRNAs, miR-132, impaired visual cortex plasticity. His lab demonstrated that specific inhibitory interneuron classes have specific response features and particular roles in the cortical processing of visual signals. By analyzing mice that are deficient in specific genes of autism and examining the effects on signaling molecules at synapses, his lab has proposed a therapeutic for Rett syndrome that has entered clinical trials. This discovery points to an exciting breakthrough in autism research.

Susumu Tonegawa's laboratory seeks to understand the brain mechanism underlying memory and its disorders. Among the laboratory's major discoveries is identifying a protein and neuronal circuits in the hippocampus that prevent a memory from remaining at the "tip of the tongue," a common memory recall deficit pronounced by normal aging and by Alzheimer's disease. Tonegawa's laboratory combines the cutting-edge technologies of genetic engineering, electrophysiology, and behavioral methods. Using a genetic technology it developed, Tonegawa's research team created mouse strains in which 1 of about 30,000 mouse genes and the protein it creates is knocked out only in a particular type of neuron of a highly restricted part of the brain. By observing the physiological and behavioral deficits of these mice, the Tonegawa laboratory, in collaboration with Matthew A. Wilson's laboratory, discovered that a single gene encoding a neurotransmitter receptor—the NMDA receptor in the tiny hippocampal area CA3—is critical for two major memory functions: the ability to rapidly form memories of episodes or events in day-to-day life and the ability to recall the details of the memory previously formed with scant information as recalling cues (a phenomenon called "pattern completion"). Most recently, using an analogous approach, Tonegawa's laboratory discovered that the NMDA receptor in another part of the hippocampus,

called the dentate gyrus, plays a crucial role in the animal's ability to acquire similar events as distinct memories (a phenomenon called "pattern separation"). This discovery also provides an intriguing explanation for the phenomenon of déjà vu and has been widely publicized in both professional journals and the popular press (e.g., Time magazine).

Also widely publicized is another recent discovery made in Tonegawa's laboratory that described a novel method to cure fragile X mental retardation and some forms of autism in a mouse model. In addition, Tonegawa's laboratory has recently invented a novel mouse genetic engineering technology that permits a blockade of neurotransmitter release from a specific type of brain cell. Applying this technique to the major pathway within the hippocampus (CA3 to CA1), Tonegawa's laboratory demonstrated that this hippocampal pathway plays a crucial role in a rapid acquisition of fear-associated memory but is dispensable for slow acquisition of spatial memory by repeated exposures. The group also demonstrated that the consolidation of fear memory into a long-lasting form requires repeated activation of relevant CA1 neurons during slow-wave sleep. This genetic technology (dubbed DICE-K) promises to be powerful in the dissection of the functions of neural circuits—neural circuit genetics. This past year, Tonegawa's laboratory has continued to study brain circuit mechanisms underlying memory. A widely held memory consolidation theory posits that memory of events and space is initially stored in the hippocampus (HPC) in a time-limited manner and is consolidated in the neocortex for permanent storage. Although studies have demonstrated that post-training HPC lesions result in temporally graded amnesia, the precise HPC circuits and mechanisms involved in remote memory storage remain poorly understood. To investigate the role of the trisynaptic pathway, one of the two major excitatory circuits of the HPC, in the consolidation process they employed the CA3-TeTX transgenic mouse, in which CA3 output can be specifically and inducibly controlled. Tonegawa's laboratory found that post-training blockade of CA3 output for up to four weeks impairs the consolidation of contextual fear memory. Moreover, in vivo hippocampal recordings revealed reductions in the intrinsic frequency of CA1 ripples and a significant decrease in the experience-dependent enhancement of the ripple-associated coordinated reactivation of CA1 cell pairs during post-run slow-wave sleep or awake quiescent periods in the mutant mice. Collectively, these results suggest that the post-training integrity of the trisynaptic pathway and the ripple-associated reactivation of hippocampal memory engram are crucial for memory consolidation.

Work in Matthew A. Wilson's laboratory continues to focus on the hippocampus's role in the formation, maintenance, and use of memory in the mammalian nervous system during awake and sleep states. Recent experiments found that while animals stop briefly on a maze they rapidly replay, or "think," about both past and future paths that they have taken or might take in a manner that is very similar to the reactivation of memories seen during sleep. This finding suggests that the mechanisms of thinking and the mechanisms of dreaming may be directly related and is consistent with recent evidence in humans that suggests that the hippocampus is involved in both processing memories of the past as well as imagining future events. Current work seeks to characterize the detailed structure of brain activity as rats navigate and contemplate such mazes, and the lab has successfully demonstrated the ability to reconstruct the content of this activity, providing a potential window into the process of thought itself.

Weifeng Xu's laboratory aims to elucidate the molecular mechanisms that underlie activity-dependent modification of neuronal properties. The work focuses on the regulation of ionotropic glutamatergic receptors, which convey the fast, feed-forward excitatory synaptic transmission in the central nervous system, and the regulation of calcium homeostasis of projection neurons in the brain. These processes are fundamental for the development of the neuronal circuitry and experience-dependent behavioral plasticity, including learning and memory. Many genes involved in these processes, including the genes of interest, have been shown to be associated with neurodevelopmental and neuropsychiatric disorders including autism spectrum disorders, schizophrenia, nonsyndromic X-linked mental retardation, and obsessive-compulsive disorder. The Xu group uses a combination of newly developed molecular approaches to manipulate genes and test the functional outcome using electrophysiological, cell biological, and biochemical approaches.

In one line of research, the Xu lab studies postsynaptic scaffold proteins in conveying signaling specificity during plasticity events. Glutamatergic receptors are embedded in the postsynaptic density (PSD), a well-organized protein-protein interaction network. The Xu group's hypothesis is that PSD scaffold proteins orchestrate normal synaptic function at glutamatergic synapses, and its aims are to analyze the functional significance of PSD scaffolds in regulating glutamatergic synaptic transmission and plasticity and to understand the functional diversity among different members of PSD families.

A second area of inquiry is the role of neurogranin in regulating neuronal calcium homeostasis and neuronal properties. Neurogranin is a small neuronal calmodulin-binding protein and is thought to regulate the calcium/calmodulin dynamics crucial for neuronal functions, including synaptic plasticity. Xu lab data suggest that the levels of neurogranin are dynamically regulated by neuronal activity and consequently influence the excitability of neurons. The Xu group aims to further determine the function of neurogranin in regulating the strength and direction of synaptic plasticity and to study the molecular substrates for the transcriptional and translational regulation of neurogranin.

The Picower Institute has a distinguished international reputation as a leader in neuroscience research. The scholarly excellence of our faculty is reflected in a distinguished publication record. During the reporting period, Picower Institute faculty published articles in 41 peer-reviewed publications, including 11 articles in hallmark science journals, such as Science, Neuron, Cell, Nature, Nature Neuroscience, and the Journal of Neuroscience.

Li-Huei Tsai
Director
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