

# NEURO TECHNOLOGIES

*Shaping the Future of Brain Research*

## Neuroscience News



WINTER 2017



**THE PICOWER  
INSTITUTE**  
FOR LEARNING AND MEMORY



## DIRECTOR'S MESSAGE

Dear Friends,

We are looking to the future. We are looking into ways in which technologies can improve our health and our ability to learn. We are also developing technologies to help us better understand the brain and its complex features like consciousness, thought, and memory. Some can be invasive or non-invasive, and some are for direct diagnostic or therapeutic benefit in humans. All are necessary if we are to push the boundaries of what we know about our brain and come up with new solutions for brain illness.

The brain furnishes us with the capacities that underpin our existence. Neurotechnologies involve interventions in the brain that are liable to affect our lives in the most intimate and fundamental ways. Now, more than ever, we have a more sophisticated understanding of how we can make this possible.

In this issue, we are excited to share several recent discoveries that tap into some of the most innovative brain therapeutic systems and technologies and detail some of what we are doing to explore the full range of their capabilities. We share work on using artificial intelligence techniques to decode dreams, a way to flicker light to slow Alzheimer's disease, and how genetic engineering is used to study the circuitry of behavior.

We also share news of our newest Catalyst program, which is transforming the way research gets funded, and paying our respects to beloved friend and MIT professor, Susan Lindquist, whose passing has affected not only our community, but many people around the world.

I hope you will enjoy reading this issue and come to appreciate that with new technology, we have the potential to improve countless lives who are suffering from the most debilitating and painful diseases. We are looking forward to where tomorrow will take us. We are on our way and we hope that you will join us on this amazing journey.

LI-HUEI TSAI, DIRECTOR

Gamma Light Therapy – One day light therapy could be used to help combat Alzheimer's disease.

# How Neurotechnologies Are Transforming How We Understand Ourselves

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TECHNOLOGY IS THE SCIENCE OF CRAFTSMANSHIP, and there are few places on Earth where crafting new tools to investigate the complexities of nature is as ambitious as The Massachusetts Institute of Technology.

The Picower Institute for Learning and Memory embraces technological development and views it as the essence of brain and mind research. In this newsletter we will explore some of the exciting and powerful tools being created by faculty members, which are turning science fiction into science fact. These include genetic engineering tools to help understand the way neural circuits compute behavior, new programming code that utilizes artificial intelligence to analyze the dreaming brain, and a technique to hack the brain, enabling it to potentially cure itself from Alzheimer's disease.

Front Cover - A physiology rig designed to turn on or off very specific sub-circuits of the brain by activating neurons with light sensitive channels. By using laser light neuroscientists can identify the neural circuitry that underlies associative learning, emotional memory, and drug addiction.

Indeed, the Latin motto of MIT *Mens et Manus* — mind and hand — is one embodied by the Picower Institute as our researchers seek to translate abstract concepts into practical applications.

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## Flashing Light Activates the Brain's Ability to Rid Itself of Alzheimer's Disease

As humankind entered the 21st century, and celebrated the grand technological leaps that have pushed life expectancy in the United States from an average age of 49 years in 1906 to 79 years in 2016, unexpected consequences have arisen.

The sharp rise in dementia and the difficulties associated with the aging brain have come into sharp focus. Alzheimer's disease -- the most common form of dementia -- is characterized by severe degeneration, and the development of clusters of proteins between neurons known as senile plaque.

Nearly 30 years ago it was discovered that senile plaques consisted of an overproduction and accumulation of a protein called amyloid-beta, which was due, in part, to a genetic mutation in the amyloid gene.

Since then, research institutions and pharmaceutical companies have spent billions of dollars in their quest to develop therapeutic drugs that target amyloid-beta. However, clinical trials have so far had a 99.6% failure rate.

Picower Director and Professor Li-Huei Tsai has developed a new approach to tame Alzheimer's disease. She invented a non-invasive technology that has been shown to successfully clear amyloid-beta from the brains of mice with Alzheimer's disease.

When targeted with an LED light that flashes on and off at 40Hz -- a frequency that is barely perceptible -- mice with Alzheimer's disease showed a reduction in amyloid-beta levels by half in the brain's primary visual center.

In addition, the decrease in amyloid-beta occurred with increased levels of a brain rhythm called gamma, which is known to be impaired in those with Alzheimer's disease.

To understand how increased gamma rhythm might be connected to reduced levels of amyloid-beta, researchers in the Tsai lab found that gamma rhythms transformed microglia and enhanced their ability to clear away pathogens such as amyloid-beta.

Professor Tsai's light therapy technique boosts the brain's natural ability to repair itself and reduce pathogenic proteins implicated in the development of Alzheimer's disease.

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## Using the Methods of Artificial Intelligence to Decode Our Dreams

Sleeping problems are more likely to affect people with a mental illness. Disrupted sleep also increases the risk of developing a mental illness, and hinders learning and memory.

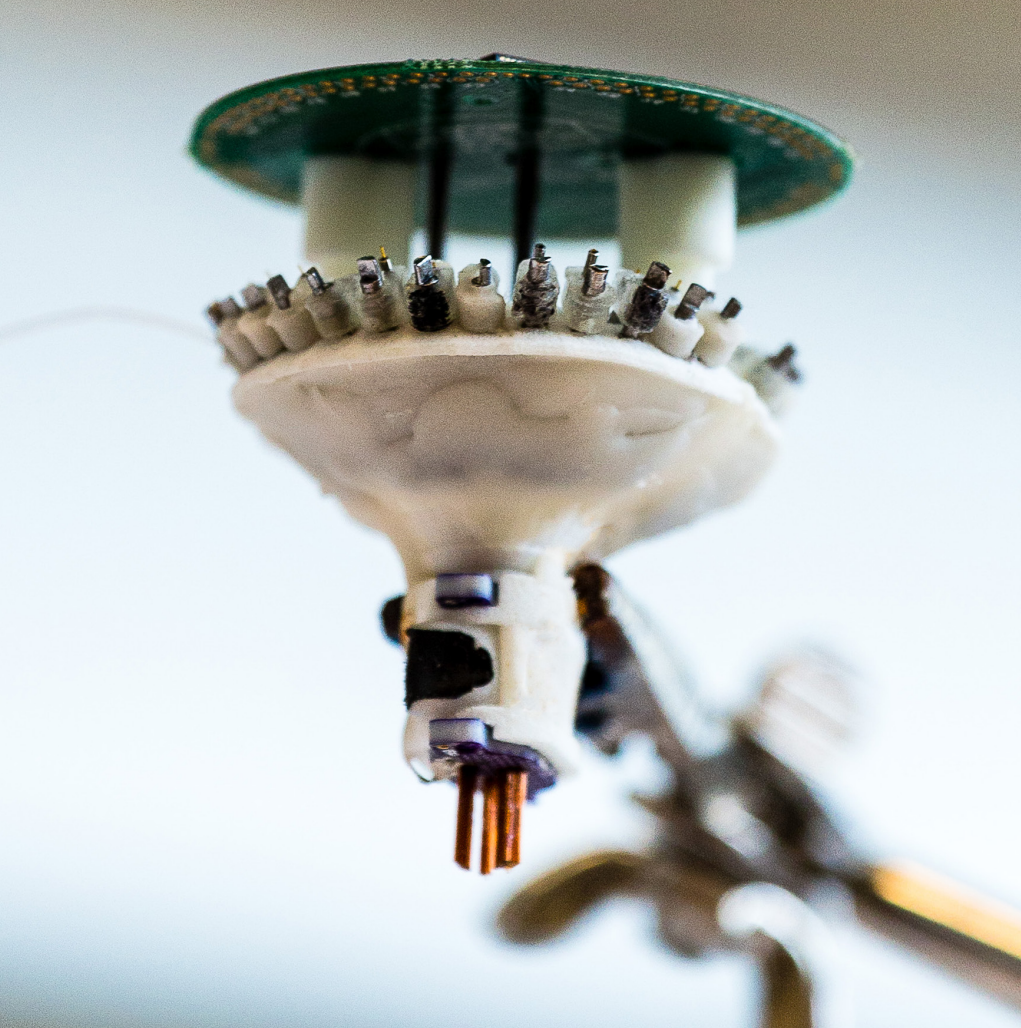
However, the neural computations of the sleeping brain are remarkably different to those of the awake brain.

Professor Mathew Wilson and his lab study how the activity of a brain region called the hippocampus — which is critical for spatial memories and memories of specific events — relates to memory formation and problem solving.

The researchers create software tools that are used by artificial intelligence (AI), software that learns from the data it is given, to decode the language of the sleeping brain. They hope this will also help further our understanding of the language of the awake brain.

Neuroscientists have known for decades that memory is enhanced when an animal is asleep. So Wilson uses specially developed programming tools to show that the activity of the sleeping brain is a crucial, if not a primary, driver of behavioral performance during learning and memory tasks.

*Mice with  
Alzheimer's  
disease  
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visual center.*



Microdrive Array – Dozens of electrodes are used to collect the electrical data from the brains of rodents. The neurological data is used to understand the language of the awake and sleeping brain.

IMAGE: JOSHUA SARIÑANA

In effect, Wilson and his lab decode dreams using the same approach as AI software to predict how the brain learns. His work will give greater insight into how we understand mental illness and further ties together the inherent functions of the brain to artificial intelligence.

## Genetic Engineering Dissects the Circuits of Behavior

All animals exhibit a natural ebb and flow of behavior, whether it is a bird singing during a summer night, or a bee dancing to share the location of nectar-yielding plants with other members of its colony in the springtime.

Such behaviors emerge from neural circuits. But how do properties of neural circuits sustain and change over time, from

singing to chirping, or dancing to flying?

Picower Professor Steven Flavell seeks to answer these questions. More specifically, he is interested in the genetic basis of neural circuit function in nematodes -- a round worm the width of a pencil tip -- which only have 302 neurons.

Flavell genetically dissects nematodes by deleting specific genes to see how they dictate shifts from one behavior to another. He seeks to reveal the fundamental principles of how neurotransmitters regulate circuit activity across the nervous system, and to discover how physiological and environmental inputs impact this process.

Flavell has discovered that neuromodulators like serotonin organize circuit activity over time, and has defined the architecture of serotonergic circuits that control ongoing behaviors.

A deeper understanding of neuronal circuit functionality should help neuroscientists understand why some people experience extended periods of depression and anxiety. If researchers can pinpoint specific deficiencies in the brains of those who suffer from these disorders, drug therapies could be developed to target faulty circuits.

The history of the Picower Institute is woven into a deep culture of technological development at one of the most innovative institutes in the most inventive state in the US. Creating new analytical tools that are based on AI allow us to decipher the cryptic language of the brain, while engineering genetic code can enhance our understanding of the neural circuits responsible for behavior, and the use of sensory tools can fool the brain into ridding itself of mutated proteins implicated in Alzheimer's disease. Technological advancement is fueling a renaissance in neuroscience research where the only hindrance is having enough hands and minds to further the understanding of our brains and ourselves.

- JOSHUA SARIÑANA, PHD



Picower Institute Professor Li-Huei Tsai

# Visual Stimulation Treats Alzheimer's Disease

A NON-INVASIVE TECHNIQUE TO TREAT patients with Alzheimer's disease has been developed by researchers at MIT.

The brain functions, in part, by synchronizing its activity across different regions. This synchrony enhances the brain's ability to recall memories, pay closer attention to the surroundings, and learn. Recent studies suggest that Alzheimer's disease disrupts brain signaling and a particular type of neuronal synchrony called the gamma rhythm.

Altered gamma rhythms in Alzheimer's disease are partly due to the toxic accumulation of a protein called amyloid-beta, resulting in fewer neurons firing in synchrony. Research from the lab of **Picower Institute Director Li-Huei Tsai** has sought to understand the relationship between Alzheimer's disease and gamma rhythms in the brain.

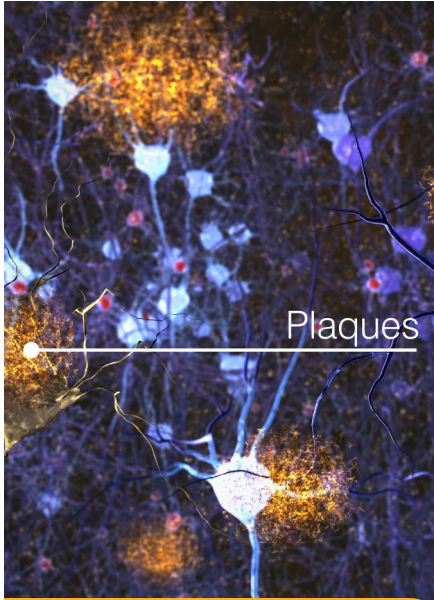
Dr. Tsai's team established that the gamma rhythm amplitude at the 40Hz range was reduced in mice with

Alzheimer's disease, called 5XFAD mice. More specifically, the gamma rhythm was significantly decreased in the hippocampus, a brain region crucial for learning and memory. Gamma rhythm in the 5XFAD mice was diminished with the accumulation of amyloid-beta.

Remarkably, Dr. Tsai's lab developed a completely non-invasive approach to using LED light to make neurons activate at the gamma rhythm to reduce the high levels of amyloid-beta

5XFAD Alzheimer's mice were exposed to 40Hz flickering light, which caused enhanced gamma rhythm neuronal activity and reduced amyloid-beta levels. In addition, the 40Hz flickering light treatment caused microglia – the immune cells that keep the brain healthy -- in the Alzheimer's mice to become more active, and dramatically increase in size by engulfing amyloid-beta.

This technique is a significant step forward in finding new and effective treatments for Alzheimer's disease.



Plaques – Neurons are surrounded by plaques, which consists of clumped mutated proteins of amyloid-beta.

IMAGE: SPUTNIK ANIMATION

## Linking Balanced Gene Expression to Autism

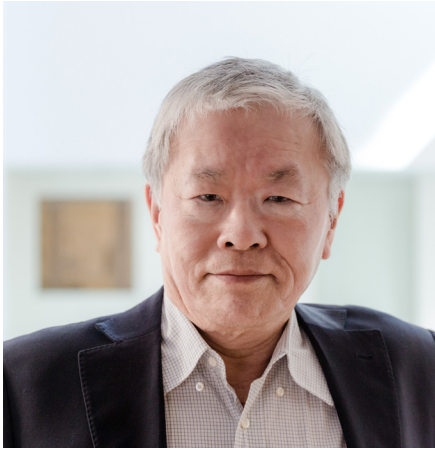
MUTATIONS IN THE GENE CHD8 ARE linked to developmental delay, cognitive impairment, and autism spectrum disorder (ASD).

Although the link between CHD8 and ASD is well known, little is understood about how mutations in the gene directly affect brain development. **Picower Institute Director Li-Huei Tsai's** laboratory depleted CHD8 in mice, in a bid to study the effects of this gene on brain development, and gain a greater insight into the genetic basis of ASD.

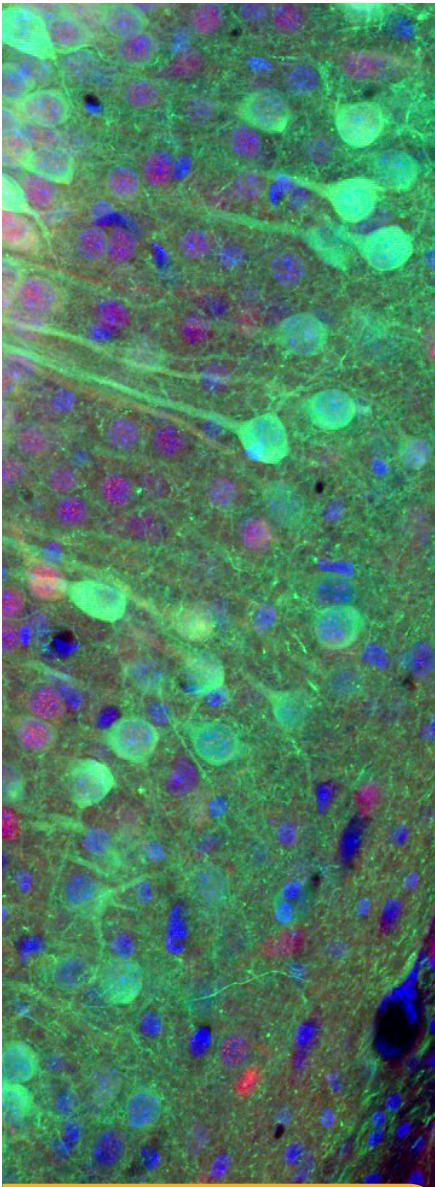
They found mice with depleted CHD8 showed a decrease in neurogenesis, or the process of creating new neurons that make

up the brain. To gain a deeper insight into how CHD8 alters neurogenesis, the researchers then conducted a global analysis of genes affected by CHD8 depletion. The results showed thousands of genes to be affected, which were broadly categorized into being either upregulated or downregulated. Upregulated genes were important for neuronal differentiation, while downregulated genes were important for brain size and development.

Professor Tsai's research suggests that CHD8 is important for balancing the genetic upregulation and downregulation critical for creating new neurons and proper brain development.



Picower Institute Professor Susumu Tonegawa



Hippocampus – Pyramidal cells (green) in area CA1 of the ventral hippocampus, which store memories of familiar individuals.

IMAGE: TERUHIRO OKUYAMA

## Connecting Social Memories

MEMORY OF FAMILIAR PEOPLE HAS BEEN shown to be stored in the brain region known as the hippocampus.

In mice, the hippocampus is shaped like a tiny cashew, and each end of this nut-shaped region has different functions. Although neuroscientists know that the upper portion is critical for event memories, the purpose of the lower section -- also known as the ventral hippocampus -- is less well understood.

**Picower professor Susumu Tonegawa's** lab has shown that the ventral hippocampus stores social memories. Researchers in his lab used optogenetics to identify and alter the activity of neurons that were active when a test mouse was introduced to a familiar or novel mouse. Optogenetics is a tool that allows neuroscientists to activate or inhibit very specific regions of the brain. When the researchers inhibited the ventral CA1 region of the hippocampus, the test mouse treated a familiar mouse as though it was novel.

The researchers then sought to determine where social memories from the ventral CA1 were sent to in the brain. They discovered that social memories were transferred to the nucleus accumbens, which is a brain region important for emotional processing. When they inhibited ventral CA1 inputs into the nucleus accumbens, they again found that it inhibited the ability of test mice to remember familiar mice.

When the ventral CA1 was re-activated in the test mice that had forgotten a once familiar mouse, they were again able to remember the mouse. In addition, an artificial memory was created, which linked the memory of a familiar mouse to either a positive or negative emotion.

The Tonegawa lab has shown that the connection between the ventral CA1 and the nucleus accumbens is necessary for social memories. These findings may have implications in finding genetic links to autism and social interaction.

## Blocking Bad Memories with Good Experiences

THE AMYGDALA IS A REGION OF THE brain that processes positive and negative emotions.

However, it is not yet known how the amygdala separates the tasks of positive and negative emotional processing. Do the same neurons process both positive and negative emotional states -- also known as valence -- or are there two distinct populations that each either process positive or negative emotional states?

Researchers in **Picower professor Susumu Tonegawa's** lab genetically identified distinct populations of neurons, and found that they each process only

positive or negative valence. In addition, those neurons that process positive emotional valence directly inhibit neurons that process negative valence, and vice versa.

Using optogenetics, the researchers were able to demonstrate that when positive valence neurons are inhibited, animals are unable to form positive memories. Similarly, when neurons that process negative valence are inhibited, the animals are unable to form fear memories.

Analyzing how the brain processes valence is crucial for understanding how brain activity gives rise to anxiety, depression, addiction, and other mental illnesses.



Picower Institute Professor Mark Bear

## Blowfish Toxin Restores Lazy Eye

AMBLYOPIA, ALSO KNOWN AS LAZY EYE, IS a common form of visual disability in children, which affects up to 5% of the world's population.

During a child's development, the visual system and the connections between the eyes and brain grow. However, such development can be hindered if the two eyes are misaligned, or if an eye is blocked, by a cataract for example, causing impaired vision

Now **Picower professor Mark Bear's** lab has developed a novel technique for the treatment of amblyopia, in which the activity of the retina is manipulated using toxin from the blowfish, known as tetrodotoxin (TTX).

Researchers in the Bear lab artificially induced amblyopia, using a technique called monocular deprivation (MD) during early life. This causes functional depression of the visual cortex, which degrades visual acuity in the deprived eye.

When the researchers temporarily silenced the retinal activity of the eye by injecting TTX, they found it reversed this deprivation-driven modification of the visual cortex. Indeed, just one week after TTX injection, the activity of the visual cortex had returned to normal.

Retinal silencing may provide an exciting potential treatment for a common visual disorder.



Lazy Eye – Picower scientists have created a novel way to help treat Amblyopia or lazy eye. IMAGE: JOSHUA SARIÑANA



Picower Institute Professor Emery N. Brown

## Regaining Consciousness is Spurred by Dopamine

GENERAL ANESTHESIA IS A STATE IN which patients are rendered completely insensate so that they can tolerate surgeries or invasive diagnostic procedures. General anesthesia is maintained by continuously administering drugs. To wake the patient up, the drugs are turned off and their effects are allowed to wear off. This process of allowing the drug effects to wear off likely contributes to brain dysfunction after general anesthesia that is commonly seen in elderly patients.

**Professor Emery N. Brown**, an anesthesiologist, a statistician and a neuroscientist, has been studying ways to actively turn the brain back on following general anesthesia. Brown has

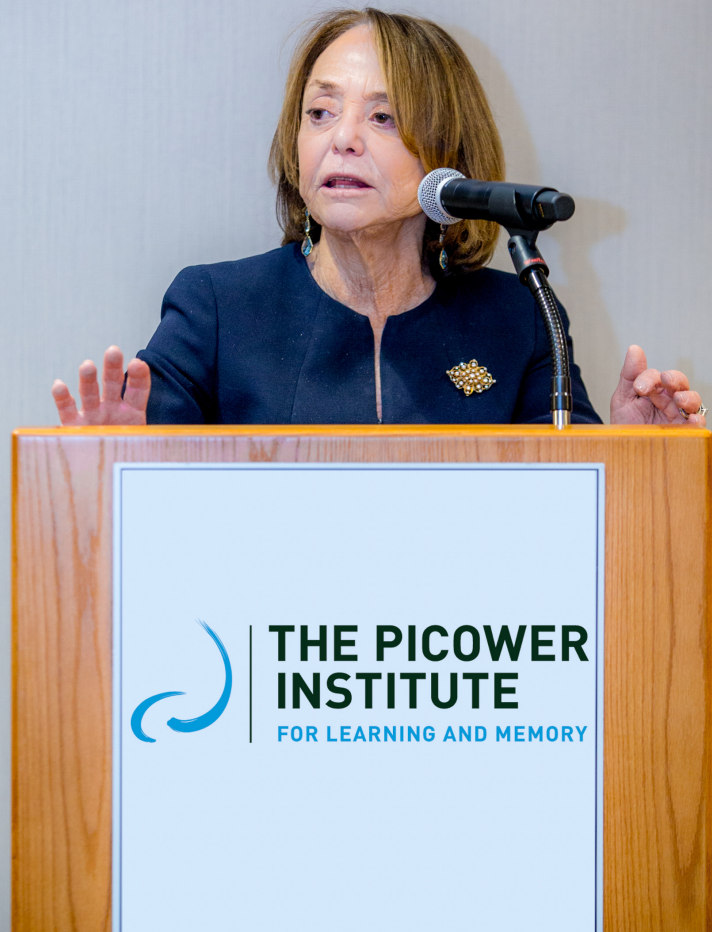
recently shown that dopamine is an important neurotransmitter that helps promote arousal from general anesthesia in animals.

Researchers in his laboratory inserted light-activated channels into neurons in a region of the brain called the ventral tegmental area, which produces dopamine. When the researchers activate the channels with light from a fiber optic probe, which in turn activates the dopamine neurons, they found it switched anesthetized animals from an unconscious state to an awake state. These findings lay important groundwork for developing dopaminergic drugs, which can be used to awake patients from general anesthesia.



Anesthesia – A woman is given anesthesia before surgery.

# A New Catalyst for Research



Barbara Picower, President of the JPB Foundation.

*The goal is to catalyze an increase in funding applications to directly enable the Picower Institute's mission.*

A NEW PROGRAM SUPPORTED BY THE JPB Foundation could radically transform the way research is typically funded.

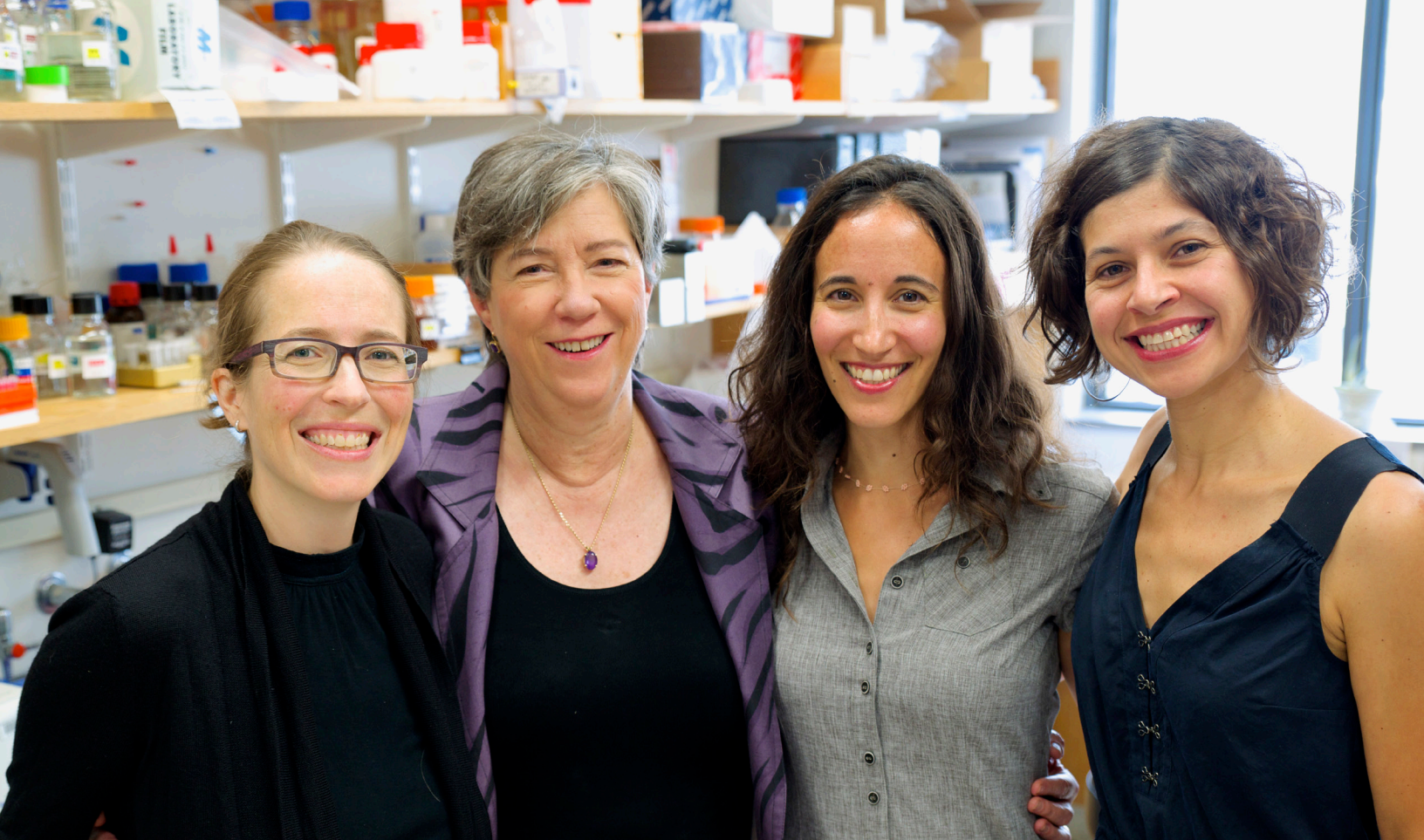
For years, research at MIT has been widely supported by the federal government. Given the current economic climate there has been a recent trend of decreased funding, broader distribution of federal budget funds to accommodate higher application rates, and other financial challenges. Because of these factors, MIT faculty have been encouraged to apply for funding from other sources. Although these sources are vital to our researchers, many of these groups chose not to pay for the full cost of research. Instead, they provided limited or no funding to cover the costs of overhead. This obstacle has proven to be an increased burden on our faculty and the Institute.

The Picower Institute “Catalyst” Program (PICP) is a new program, supported by the JPB Foundation to help fund those indirect costs. The goal is to catalyze an increase in funding applications to

directly enable the Picower Institute’s mission. With more PILM investigators submitting applications the expectation is that more secured research support will be awarded to understanding of how the brain learns and encodes memories in sickness and health.

We are thankful to the JPB Foundation for their commitment and foresight to launch this program and deal head-on with this challenge. This is a truly transformative gift to forge new territory, providing one of the first major funds specifically directed to support the true costs of research. This type of comprehensive commitment is rare, yet addressing a very real and important challenge to researchers. If you would like to join the JPB Foundation and Picower Institute to tackle this established problem in the scientific community, you may make a contribution online or by contacting Dr. Asha Bhakar, Director of Development at the Picower Institute for Learning and Memory at [abhakar@mit.edu](mailto:abhakar@mit.edu).





Susan Lindquist, second from the left.

## *Remembering* SUSAN LINDQUIST

IT IS WITH PROFOUND SADNESS THAT WE share the news of the passing of Professor Susan Lindquist. Sue was a friend to the MIT community and to many at the Picower Institute for Learning and Memory.

After earning her PhD in biology from Harvard, Sue spent 23 extraordinarily productive years at the University of Chicago. She joined the Department of Biology and MIT's affiliated Whitehead Institute in 2001, infusing both communities with her scientific fearlessness, creativity and drive, and her great personal warmth. Sue collaborated widely and was a founding member of MIT's Aging Brain Initiative, which seeks to find an end to the dementias of aging.

Professor Li-Huei Tsai, Director of the Picower Institute and lead investigator of the Aging Brain Initiative, remembers an insightful leader and dear colleague. "Sue's bold strategies and unique ideas to understand neurodegenerative disease were recognized by her peers and supported by generous partners, including the JPB Foundation and the Belfer Family Foundation," said Professor Tsai. Sue was a pioneer in the study of protein folding and a genuine luminary, appreciated for her candor, friendship, thoughtful behavior and superb communication skills. She led by example and her work and influence will continue to accelerate the fight against diseases such as Parkinson's and Alzheimer's, as well as inspire and educate upcoming generations of scientific leaders.

A Howard Hughes Medical Institute investigator, Sue was a member of the American Academy of Arts and Sciences, the National Academy of Sciences, the National Academy of Medicine and the Royal Society. An esteemed member of Johnson & Johnson's corporate board, she also played a key role in several start-ups. For her scholarly achievements, she received, among many honors, the National Medal of Science, the nation's highest award in the sciences; for her brilliance and devotion as a teacher and mentor, she won the love and admiration of hundreds of young scientists, many now themselves rising scientific stars.

This heartbreaking loss is felt throughout MIT's campus and has certainly left a deep void within our community. Her life was meaningful to so many. It is important to remember, however, that her passion, determination, and heart will leave lasting impressions in scientific communities around the world and for generations to come.

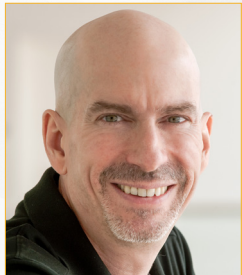
# PICOWER Accomplishments



Kwanghun Chung



Troy Littleton



Earl Miller



Elly Nedivi



Li-Huei Tsai



Kay Tye

## Kwanghun Chung

Kwanghun Chung was selected to receive a 2016 NIH New Innovator Award for his project, *Proteome-Driven Holistic Reconstruction of Organ-Wide Multi-Scale Networks*. Chung incorporates new tissue processing technologies with genetic labeling techniques to create maps of the brain with nanoscopic resolution.

## Troy Littleton

Troy Littleton received the Menicon Professor in Neuroscience chair. Littleton works to understand the mechanisms by which neurons form synaptic connections, how synapses transmit information, and how synapses change during learning and memory, as well as how alterations in neuronal signaling underlie several neurological diseases, including epilepsy, autism and Huntington's disease. He also received the Department of Brain and Cognitive Science Award for Excellence in Graduate Teaching.

## Earl Miller

The Brain and Behavior Research Foundation awarded Earl Miller the Goldman-Rakic Prize for Outstanding Achievement in Cognitive Neuroscience for his work on the neural circuitry of cognition. The award celebrates the transformative power of neuroscience and psychiatric research to improve the lives of people with mental illness, which affects one in five people.

## Elly Nedivi

Elly Nedivi was elected as a 2016 AAAS Fellow and recognized for distinguished contributions to the field of neuroscience, particularly for defining novel cellular and molecular mechanisms underlying activity-dependent synaptic plasticity.

## Li-Huei Tsai

Li-Huei Tsai was named the recipient of the Society for Neuroscience Mika Salpeter Lifetime Achievement Award. This Mika Salpeter Lifetime Award recognizes an individual neuroscientist with distinguished achievements in neuroscience that actively promotes the advancement of women in neuroscience.

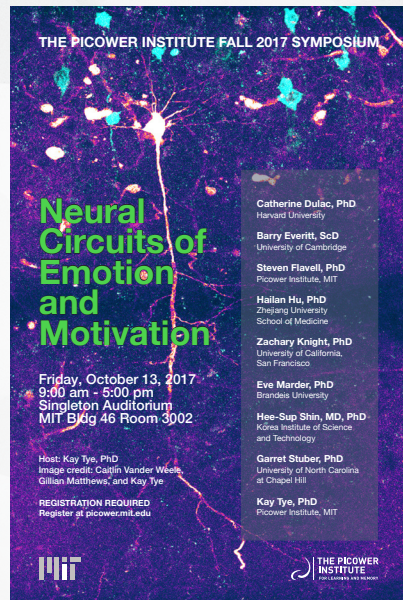
## Kay Tye

The Society for Neuroscience named Kay M. Tye the recipient of its Young Investigator Award, which recognizes outstanding achievements and contributions by a young neuroscientist. Tye also received the Brain and Behavior Research Foundation's annual Freedman Prizes for her project *Identifying Unique Neural Circuits for Anxiety Control*.

# Upcoming EVENTS

For a list of ongoing scientific lectures, colloquia, and workshops, please go to: [picower.mit.edu](http://picower.mit.edu)

## Fall Symposium 10.13.17



**Catherine Dulac, PhD** is the Higgins Professor of Molecular and Cellular Biology and chair of the Department of Molecular and Cellular Biology at Harvard University. Her lab investigates the architecture and functional logic of neuronal circuits underlying pheromone signaling and the phenomenon of genomic imprinting in the brain, and the role of this mode of epigenetic modification in brain development and adult brain function.

**Barry Everitt, ScD** is a Professor and Director of Research, and Provost of the Gates Cambridge Trust, at the University of Cambridge. His research is concerned with the neural and psychological mechanisms underlying learning, memory, motivation and reward especially related to drug addiction.

**Steven Flavell, PhD** is an Assistant Professor in the Brain and Cognitive Sciences Department and the Picower Institute for Learning and Memory at MIT. His lab's goal is to understand how neural circuits generate sustained behavioral states, and how physiological and environmental information is integrated into these circuits.

**Hailan Hu, PhD** is a Professor and Senior Principal Investigator at the Zhejiang Uni-

versity Interdisciplinary Institute of Neuroscience and Technology (ZIINT) and School of Medicine at Zhejiang University. Her lab aims to understand how emotional and social behaviors are encoded in the brain, with a main focus on the neural circuitry underlying depression and dominance hierarchy.

**Zachary Knight, PhD** is an Assistant Professor in the Department of Physiology at the University of California, San Francisco and a Robertson Neuroscience Investigator of the New York Stem Cell Foundation. His lab studies neural circuits in the mouse that control feeding and other motivated behaviors central to survival. Their goal is to understand how these circuits are able to sense the needs of the body and then generate specific behavioral responses that restore homeostasis.

**Eve Marder, PhD** is the Victor and Gwendolyn Beinfeld Professor of Neuroscience in the Biology Department of Brandeis University. She studies the dynamics of small neuronal networks, and her work was instrumental in demonstrating that neuronal circuits are not "hard-wired" but can be reconfigured by neuromodulatory neurons and substances to produce a variety of outputs.

**Hee-Sup Shin, MD, PhD** is the Director of the Center for Cognition and Sociality Institute for Basic Science and a Principal Research Scientist at the Korea Institute of Science and Technology. His work is aimed at understanding how changes in calcium dynamics in nerve cells regulate brain functions.

**Garret Stuber, PhD** is an Assistant Professor in the Department of Cell Biology and Physiology at the University of North Carolina at Chapel Hill. His primary research goal is to further delineate the synaptic mechanisms and functional neural circuitry that underlie motivated behavioral processes that are perturbed in neuropsychiatric disorders such as addiction, depression, and eating disorders.

**Kay Tye, PhD** is an Assistant Professor in the Brain and Cognitive Sciences Department and the Picower Institute for Learning and Memory at MIT. Her lab employs an interdisciplinary approach including optogenetics, electrophysiology, pharmacology and imaging techniques to find a mechanistic explanation for how emotional and motivational states can influence learning and behavior, in both health and disease.



**05.17.17 Patrick Purdon, PhD** is an Associate Professor of Anesthesia at Massachusetts General Hospital. He develops and applies novel methods in neuroimaging and biomedical signal processing to study the systems neuroscience of general anesthesia and other altered states of consciousness.

**10.17.17 Scott A. Small, MD** is the Director of the Alzheimer's Disease Research Center at Columbia University, where he is the Boris and Rose Katz Professor of Neurology. With an expertise in Alzheimer's disease and cognitive aging, Dr. Small's research focuses on the hippocampus, a circuit in the brain targeted by these and other disorders, notably schizophrenia. He has pioneered the development and application of high-resolution functional MRI techniques that can pinpoint parts of the hippocampus most affected by aging and disease. His lab then uses this information to try to identify causes of these disorders.

**11.01.17 Richard Ransohoff, MD** is Vice President and Senior Research Fellow in Neuroimmunology at Biogen. He is a leading neuroimmunologist whose research has focused on the functions of chemokines and chemokine receptors in the development and pathology of the central nervous system.

**12.06.17 Bruce Yankner, MD, PhD** is Professor of Genetics and Neurology at Harvard Medical School, Director of the Harvard Neurodegeneration Training Program, and Co-Director of the Paul F. Glenn Center for the Biology of Aging. His work has contributed to understanding pathogenic mechanisms in Alzheimer's disease, Down's syndrome and Parkinson's disease, beginning with the initial observation that amyloid beta protein is a toxic molecule, and later with investigations into the roles of presenilin proteins, Notch and Wnt in neuronal signaling and pathology.

## Neuroscience News // Winter 2017



### OUR VISION

The Picower Institute is a community of scientists focused on a common question: How is the brain modified by experience?

To answer this question we use multiple levels of analysis, ranging from molecular to behavioral, and exploit the tools of modern molecular biology and genetics to dissect the contributions of specific molecules, synapses, cells and circuits to behavior.

We work to understand the pathophysiological mechanisms underlying complex disorders of the brain that affect emotion and cognition.

### SUPPORT THE PICOWER INSTITUTE

For more information on our research or how to make a gift to the Picower Institute for Learning and Memory, please contact: Asha Bhakar, PhD, [abhakar@mit.edu](mailto:abhakar@mit.edu), Tel: 617-258-0759.

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**TOP ROW:** **Mark F. Bear**, Picower Professor of Neuroscience, Department of Brain and Cognitive Sciences, Investigator, Howard Hughes Medical Institute (HHMI); **Emery Brown**, Edward Hood Taplin Professor of Computational Neuroscience and Health Sciences & Technology, The Picower Institute for Learning and Memory, Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology; **Kwanghun Chung**, Assistant Professor, Departments of Chemical Engineering and Brain and Cognitive Sciences, Institute of Medical Engineering and Science core faculty; **Steven Flavell**, Assistant Professor of Neuroscience, The Picower Institute for Learning and Memory, Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology; **Myriam Heiman**, Assistant Professor of Neuroscience, Department of Brain and Cognitive Sciences, Broad Institute core member; **Troy Littleton**, Picower Professor of Biology and Neuroscience, Departments of Biology and Brain and Cognitive Sciences; **Earl Miller**, Picower Professor of Neuroscience, Department of Brain and Cognitive Sciences.

**BOTTOM ROW:** **Elly Nedivi**, Professor, Departments of Brain and Cognitive Sciences and Biology; **Mriganka Sur**, Paul E. Newton Professor of Neuroscience, Director of The Simons Center for the Social Brain; **Susumu Tonegawa**, Picower Professor of Biology and Neuroscience, Departments of Brain and Cognitive Sciences and Biology, Investigator, Howard Hughes Medical Institute, Investigator and Director of the RIKEN-MIT Center for Neural Circuit Genetics; **Li-Huei Tsai**, Picower Professor of Neuroscience, Department of Brain and Cognitive Sciences, Director, The Picower Institute for Learning and Memory; **Kay Tye**, Assistant Professor of Neuroscience, Department of Brain and Cognitive Sciences; **Matthew Wilson**, Sherman Fairchild Professor in Neurobiology, Departments of Brain and Cognitive Sciences and Biology, Associate Director, The Picower Institute for Learning and Memory; **Weifeng Xu**, Assistant Professor of Neuroscience, Department of Brain and Cognitive Sciences.