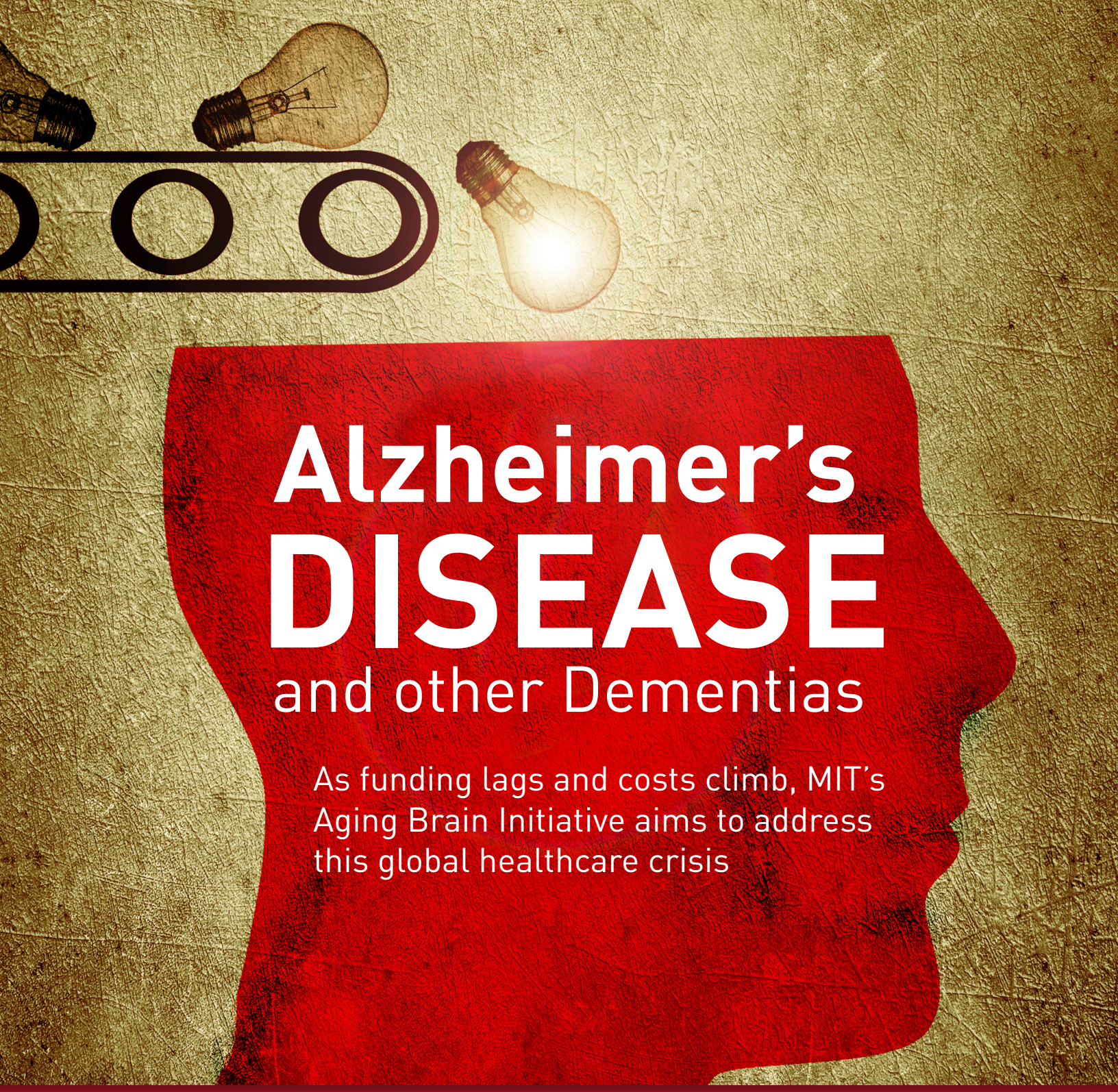


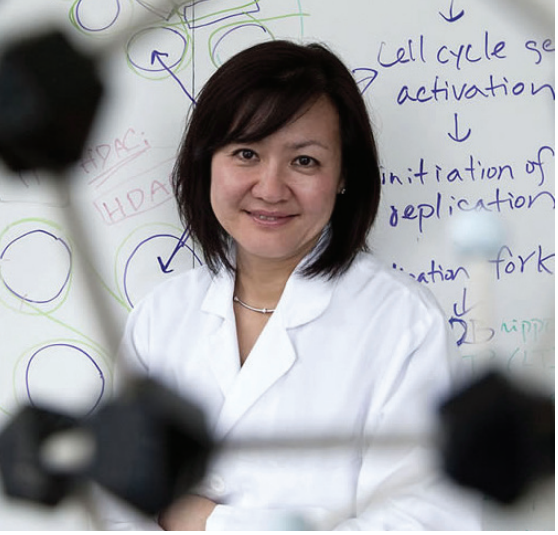
Alzheimer's DISEASE
and other Dementias

As funding lags and costs climb, MIT's Aging Brain Initiative aims to address this global healthcare crisis

Neuroscience News

Spring 2015

THE PICOWER INSTITUTE
FOR LEARNING AND MEMORY



Director's **Message**

Dear Friend,

The ailments of the mind and mysteries of the brain are what unite and inspire us here. That's why I'm proud to announce the Aging Brain Initiative, a far-reaching, highly collaborative effort that we're mounting at the Picower Institute together with the MIT School of Science and our colleagues in Neurosciences, Bioengineering, Biology, Computer Science, Artificial Intelligence, Medicine, Health Economics, and Health Policy.

There's no more urgent health challenge today than Alzheimer's disease and the dementias of aging. In this issue you'll read how the Aging Brain Initiative will help us understand the neurological bases of the aging brain in health and cognitive decline. You'll also read about the urgent need for fundamental research into how the brain ages -- and for the funds to support this research.

I'd also like to share recent scientific breakthroughs made at the Picower Institute as we work toward solving some of the world's most pressing health problems. You'll read about recent discoveries on Alzheimer's Disease, addiction and Huntington's Disease, as well as new work on Down syndrome, autism spectrum disorders and PTSD. And we share new insights on how brain waves contribute to learning, how we habituate to new information, and how we're developing new technologies to better understand how neurons communicate in the brain.

Finally, I want to thank two respected donors whose gifts testify to the transformative power of private philanthropy. Thank you to the JPB Foundation for its generous gift to support high-risk, high-reward fundamental research, and to Brazil's Alana Foundation for its foresighted support of an important collaborative study of the links between Down syndrome and Alzheimer's disease.

We hope you'll enjoy reading about the incredibly varied and enormously productive basic research conducted here: research that lays the foundation for the therapies of the future.

- Li-Huei Tsai, Director

New Research Initiative Targets the **Aging Brain**

Our most serious healthcare challenge is in a funding crisis.

Sadly, the funds to support the research we need to conquer Alzheimer's disease are simply not there. Last year the NIH spent $5.4 billion on cancer research, $3 billion on HIV/AIDS, $1.2 billion on heart disease...and just $566 million on AD.

And while deaths from other chronic diseases are declining — along with their impact on costs of care — AD and AD-related Medicare and Medicaid expenses are becoming our largest healthcare cost. The Alzheimer's Association tells us that 5.2 million Americans now have AD. By 2050 the number will rise to 13.8 million. No one has ever recovered from the disease. There is no cure. There's not even an effective treatment to slow its spread.

There's never been a greater need — or a greater urgency — to address this crisis. And the necessary first step is to unravel the foundational biology of the aging brain and the co-morbidities it spawns. That's the mission of a new research effort spearheaded by **Li-Huei Tsai**, director of the Picower Institute, and **Michael Sipser**, Dean of the MIT School of Science.

The Aging Brain Initiative is a far-reaching, highly collaborative effort involving the Picower Institute, the MIT School of Science and our colleagues in the Neurosciences, Bioengineering, Biology, Computer Science, Artificial Intelligence, Medicine, Health Economics and Health Policy.

Its aim is to focus a broad range of research talent on a single goal: improving quality of life through fundamental research into how the brain ages in health and in decline. For until we know more about what causes brain functions to change with age, we will be no closer to a cure or a disease-modifying therapy.

In the past two years the FDA has approved 19 new cancer drugs, with more in the pipeline. In contrast, not a single new AD drug has been approved in the past decade. The reason is simple: more than 40 years of major investment in basic cancer research has made the disease much better understood than AD. And this understanding has led to successful translational research. Today's oncology drugs could not have been developed without this foundational knowledge and the financial commitment of the War on Cancer.

in this **issue**

Picower researchers target Down Syndrome, Alzheimer's disease, Type II diabetes, obesity, autism spectrum disorders, Huntington's disease, PTSD, schizophrenia... plus the many ways we learn and remember

While the task facing the Aging Brain Initiative is daunting, the opportunities are great. "We're steps away from new models of neurological disease, mining big genomic data sets and applying engineered technologies that will lead to novel treatments for the co-morbidities of aging," says Li-Huei Tsai.

The Initiative founding members represent the best in fundamental aging brain research and innovation. In addition to **Li-Huei Tsai**, they include MIT Professors **Edward Boyden, Emery Brown, Leonard Guarente, H. Robert Horvitz** and **Susan Lindquist.**

They envision a new model for research at an institution uniquely positioned to implement it: a multidisciplinary, Boston-wide effort dedicated to collaborative research and interactive forums that integrate core sciences and technologies across a wide range of fields, from neuroscience and bioengineering to computer science and health economics.

Synergies built from these and other domains will create a new and comprehensive approach to understanding the aging brain and an investment platform for long-term commitments to address this health imperative. By encouraging collaboration and varied perspectives, results are likely to be more robust and come more quickly.

A new Picower project exemplifies this approach. It's long been known that the brains of people with Down syndrome exhibit many of the plaques and tangles that characterize the brains of people with AD. Understanding the connections between the conditions — and how treatments for one might benefit the other — is the focus of a new collaboration led by Li-Huei Tsai and collaborator Alberto Costa of Case Western Reserve University (read more on page 10).

MIT and its collaborators are particularly well equipped to begin the process of foundational research. The effort will leverage MIT's leadership in hard sciences, engineering and technology, its exceptional laboratory and educational infrastructure, its intellectual capital, can-do philosophy, well-known entrepreneurial culture, and its partners in the medical community and the Kendall Square commercialization corridor.

Research approaches and results won't be siloed but will be shared openly and collaboratively. Questions asked and answers proposed will be debated and expanded through regular seminars and workshops like the new **Aging Brain Seminar Series** (read more on page 11).

The first years of the effort will be devoted to a four-pronged, people — and project-based approach that helps us understand healthy and unhealthy brain aging and develop real solutions to reduce cognitive decline. The focus will be on identifying aging biomarkers, developing circuit-specific therapeutics, exploring personalized molecular-based medicine and identifying strategies to promote healthy brain aging.

The ultimate goal is to deliver the tools, technologies and pharmaceuticals needed to address the challenges of the aging brain — always with the aim of moving knowledge as quickly as possible from bench to bedside.

From left to right: **DEAN MICHAEL SIPSER**, School of Science; **EDWARD BOYDEN**, Associate Professor of Biological Engineering and Brain and Cognitive Sciences in the MIT Media Lab and the McGovern Institute for Brain Research, **EMERY BROWN**, Edward Hood Taplin Professor of Medical Engineering and Professor of Computational Neuroscience in the Department of Brain and Cognitive Sciences at MIT and Massachusetts General Hospital, **LEONARD GUARENTE**, David H. Koch Professor of Biology at MIT, **H. ROBERT HORVITZ**, David H. Koch Professor of Biology at MIT, and **SUSAN LINDQUIST,** Professor in the Department of Biology at MIT and The Whitehead Institute; **LI-HUEI TSAI**, Lead Investigator, Director of the Picower Institute and Picower Professor of Neuroscience in the Department of Brain and Cognitive Sciences at MIT.

We need your **help**

We have the talent and the tools. And we know what must be done to win the war against Alzheimer's disease and the dementias of aging.

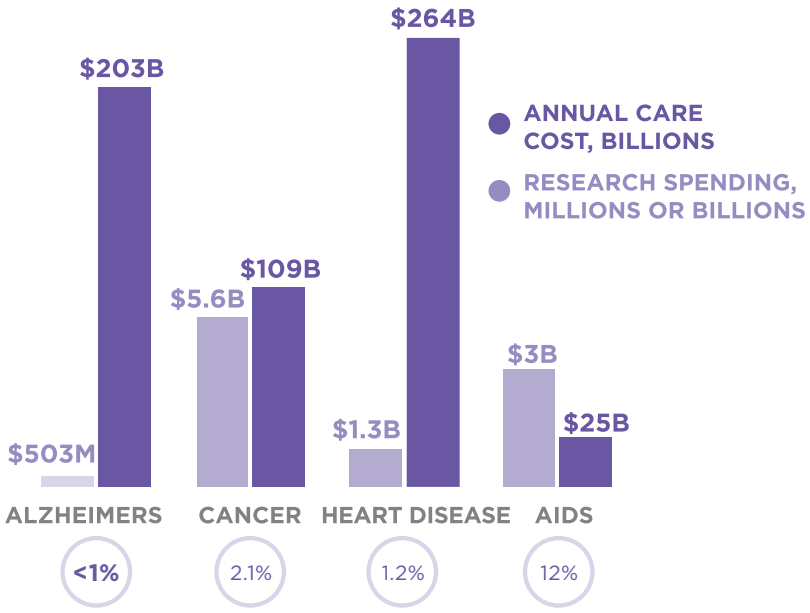
Our greatest challenge is to fund this future. We must find the resources to execute groundbreaking basic research despite the paucity of government funding. Private philanthropy can make a significant difference in this critical problem of our lifetimes. Please consider a gift in support of this effort.

"I can think of no other condition that places such a heavy burden on society, families, communities, and economies. I can think of no other condition where innovation, including breakthrough discoveries, is so badly needed."

- Dr Margaret Chan

Director-General of the World Health Organization
Address to the 2013 G8 Dementia Summit

RESEARCH SPENDING ANNUAL CARE COST



EVERY YEAR, ALZHEIMER'S AFFECTS MORE THAN

5.4 MILLION AMERICANS

WHILE

CANCER AFFECTS

1.6 MILLION A YEAR

AIDS AFFECTS

1.1 MILLION PEOPLE A YEAR

HEART DISEASE AFFECTS

716 THOUSAND PEOPLE A YEAR

Source Alzheimers.net

Brain's Immune Response Tied to Risk for *Alzheimer's disease*

▲ Current and past Tsai lab members who attended the Tsai Lab 20th reunion in November 2014. Li-Huei Tsai is in the front center row wearing pearls. First author Elizabeta Gjoneska is standing directly above her.

New research from **Li-Huei Tsai's** lab ties changes in the brain's immune response to Alzheimer's disease. The research identified noncoding regions of DNA that mediate the progression of AD. These noncoding areas fall outside the genes themselves and instead regulate how and when genes are expressed.

The team analyzed the mouse brain epigenome, a collection of chemical modifications that package DNA within a cell, to define the noncoding regulatory regions. Then they demonstrated that these regions are present and function similarly in human brains, underscoring the importance of using animal models to study human disease.

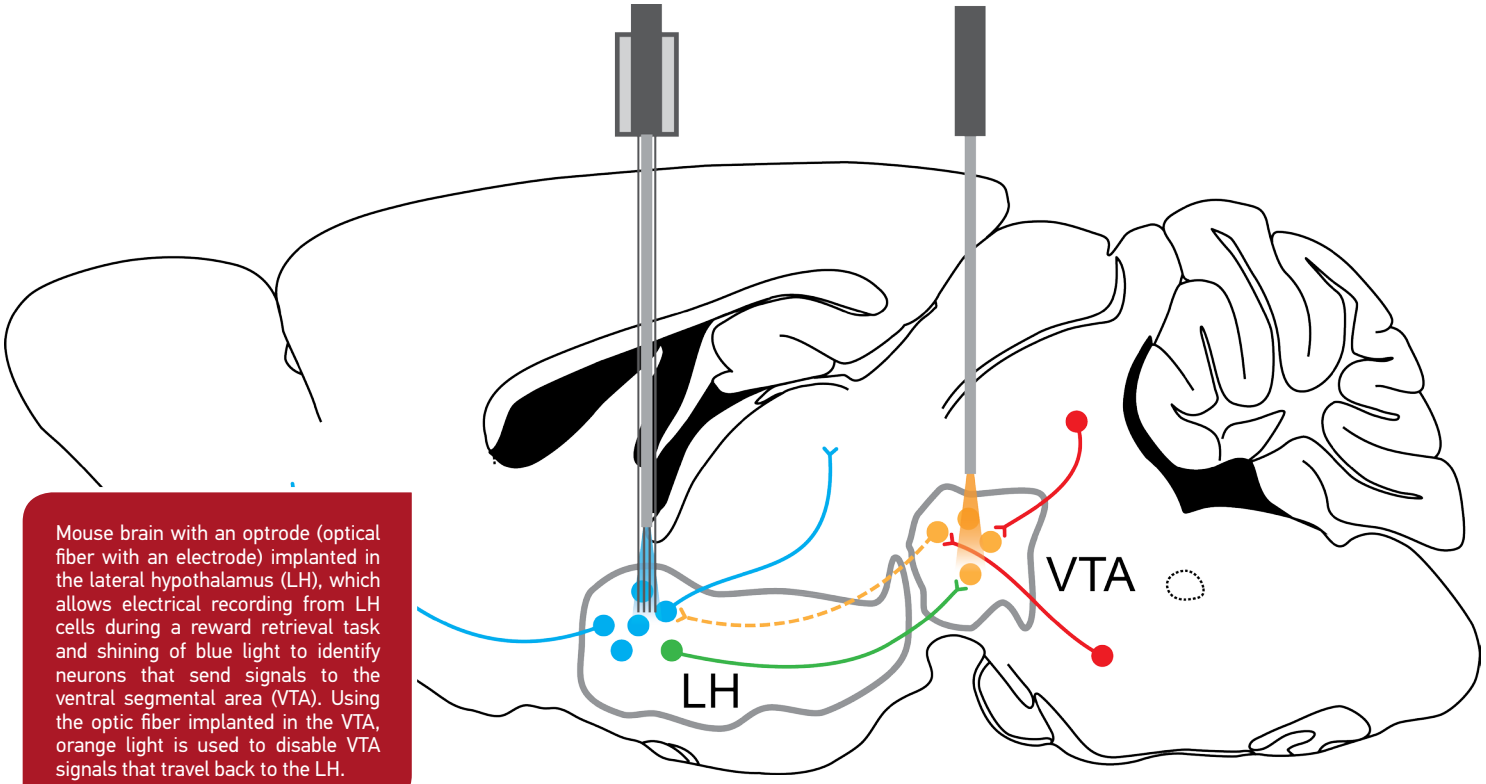
First author Elizabeta Gjoneska and her colleagues also used a mouse model of AD developed in the Tsai lab to identify the noncoding regions that regulate gene expression changes during disease progression. This "map" of conserved epigenomic regions allowed the researchers to examine the contribution of genetic variants commonly found in AD patients to the progression of the disease.

They found that the majority of the common genetic variants localized to these regulatory regions to increase genes that modulate immune function during AD. This finding indicates that our genetic susceptibility to developing AD is rooted in the immune system.

"These results also suggest that the decreased neuron function, that is a hallmark of AD is not due to genetic predisposition. Instead it's likely a consequence of exposure to environmental factors and aging as well as interactions with the altered immune function," says Tsai.

"We also identified a number of protein 'master regulators' that bind to these regulatory regions of DNA and control the gene expression changes underlying the disease," says Gjoneska. "This suggests that we've found novel targets for drug development and additional avenues for therapeutic interventions."

Published February 19, 2015 in ***Nature***.



Mouse brain with an optrode (optical fiber with an electrode) implanted in the lateral hypothalamus (LH), which allows electrical recording from LH cells during a reward retrieval task and shining of blue light to identify neurons that send signals to the ventral segmental area (VTA). Using the optic fiber implanted in the VTA, orange light is used to disable VTA signals that travel back to the LH.

Decoding Sugar *Addiction*

Everybody loves sugar — including lab mice. The problem, of course, is that this love slides easily into addiction, as the rising tide of obesity and type 2 diabetes in the US shows.

Kay Tye and her lab made this near-universal craving the basis of research that holds promise for dealing with a host of addictions, including **obesity**, **diabetes** and **drug dependency**.

The Tye team was able to identify and control a previously unknown brain circuit that regulates compulsive sugar consumption without interfering with normal, healthy eating.

"For the first time we've identified how the brain encodes compulsive sugar seeking," says Tye, "and we've shown it seems to be distinct from normal eating." This raises the possibility of curbing our drive to eat unhealthy food without interfering with eating food that's good for us.

The study first focused on the connections between the ventral tegmental area — the brain's reward-processing center — and the lateral hypothalamus, which controls eating. But because the LH controls other behaviors and connects to other brain regions, nobody had yet isolated this eating and reward-processing circuit.

First Tye and graduate student Edward Nieh identified the LH neurons that connect to the VTA and used electrodes in mice to record the activity of these LH neurons as the mice ate.

They found that one type of LH neuron became active only after the mice learned to look for a sugar reward. Another set of neurons encoded the response to a cue predicting the reward and the reward itself.

Next, using a technique called optogenetics, which uses light to control neurons that have been genetically sensitized to light, they used pulses of light to turn these neurons on or off in mice. Turning the neurons on led to compulsive overeating of a sugar reward even when the mice were full: behavior that looks very much like addiction. Turning the neurons off reduced sugar overeating but didn't stop the mice from eating normal food when they were hungry.

"That's exciting, because our recording data show how this compulsive sugar-seeking happens," says Nieh, "We can drive or suppress just the compulsive behavior by making very precise changes in neural circuitry."

Addiction researchers have hypothesized that the transition from actions to habits to compulsion is the path to addiction formation, but exactly where and how this happens in the brain has been a mystery. Now there's evidence that it happens in the LH-VTA circuit.

The lab is now investigating how eating and sugar-seeking behaviors differ based on neuron types. Says Dr. Tye: "One goal is to develop safe, noninvasive approaches to avert maladaptive eating, first in mice and eventually in people."

This work appeared January 29, 2015 in ***Cell***.

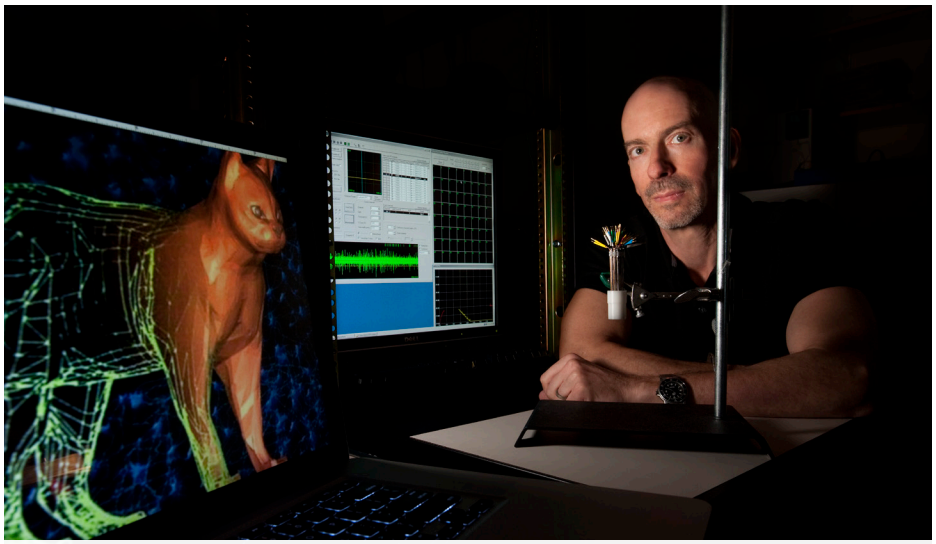


Photo by Lex Rabenstein

Professor Earl Miller

Brain Waves Make ***Memories***

Our brains are humming. Literally. As the neurons in the brain fire, they produce waves that oscillate at different frequencies. Now **Earl Miller** and his lab have developed data that suggest that these different frequencies tell the brain which memories to store and which to discard, which could have clinical relevance for enhancing **learning and memory** and treating neurological disorders.

"It's like you're playing a computer game and you get a ding when you get it right, and a buzz when you get it wrong," says Miller. "These two areas of the brain are playing two different notes for correct and incorrect guesses."

The research team showed pairs of images to animals, rewarding them when they learned which pairs went together. Correct guesses made the waves oscillate at a higher beta frequency. Incorrect guesses triggered a lower theta frequency.

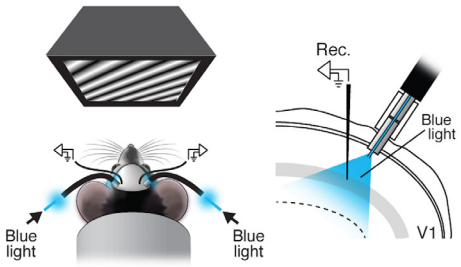
The results are a major step in determining how memories are formed. "Brain waves have been ignored for decades," Miller says. "But we're discovering that they may be the infrastructure of neural communication."

Now the Miller lab is investigating whether we can speed up learning by delivering noninvasive electrical stimulation at beta frequencies for right answers and theta frequencies for wrong answers. "The idea is that correct guesses feel more correct to the brain, and incorrect guesses feel more incorrect," Miller says.

"This technique has been approved for use in humans," says Scott Brincat, the paper's first author. "So if it works here it could have clinical relevance for enhancing memory or treating neurological disorders."

Published February 23, 2014 in ***Nature Neuroscience***.

How We ***Remember*** What We See



Optogenetic strategies to inactivate the primary visual area of the brain (called V1): An opto-genetic or "light-sensitive" protein called Channelrhodopsin-2, has been placed in V1 to turn a specific type of neuron off using blue light while an animal views a visual stimulus.

A research team led by **Mark Bear** has made a surprising discovery about how our brains form memories of what we've seen, a process that helps us detect novelty.

The Bear lab showed that dramatic changes occur in the primary visual cortex of mice when the mice familiarize themselves with simple visual stimuli. But if these changes are prevented, the mice can't form memories of what they've seen.

Before this discovery, scientists thought of the primary visual cortex as simply the "first responder" to visual stimuli that passes information along to higher-order brain regions for memory storage.

"The study points to the visual cortex as a tool of learning and memory in its own right," says Samuel Cooke, the study's lead author.

Bear anticipates the results will surprise neuroscientists. "Contrary to the dogma that the primary visual cortex is relatively immutable in adults, we've found that a form of visual experience induces synaptic modifications in this area, and these modifications are necessary for a type of visual recognition memory."

The work may eventually have an impact on research into psychiatric disorders: difficulties in recognizing familiar stimuli are characteristic of conditions like **autism** and **schizophrenia**.

Published January 19, 2015 in ***Nature Neuroscience***.

Photo by Len Rubenstein

Different Causes, Similar *Treatment*

Professor Mark Bear

Can a treatment that works on one genetic risk for **autism** and **intellectual disability** work on others too? A research team led by **Mark Bear** suggests this is possible for two very different disorders.

The Bear team looked at fragile X syndrome and 16p11.2 microdeletion, two unrelated genetic disorders that can result in intellectual deficits and autism. They found that both disorders disrupt the way protein is synthesized at the brain's synapses, the sites that facilitate communication between neurons.

The team established that mouse models of fragile X and 16p11.2 both had altered protein synthesis in the hippocampus, an area that's important for memory formation: altered protein levels gives rise to many of the psychiatric and neurological symptoms of autism and intellectual disability.

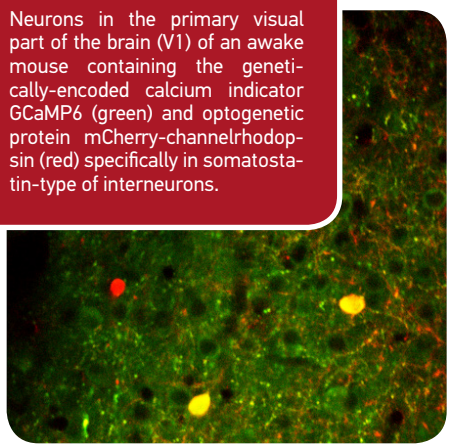
Together with scientists from Cold Spring Harbor Laboratory and Roche Laboratories, they found that blocking this synthesis improved memory and cognition and reversed many of the disorders' cognitive deficits, even when treatment started well after birth.

The implication, says Bear, is that some cognitive aspects of the disorders may not be an intractable consequence of altered early brain development but could be caused by ongoing changes in synaptic signaling. These changes might be correctable by drugs. And treatment for one genetic deficit could well apply to others.

The possibility is heartening since well over 100 distinct gene mutations can manifest as intellectual disability and autism.

Published January 12, 2015 in ***Nature Neuroscience***.

Technology Sheds Light on Interneuron Function



Neurons in the primary visual part of the brain (V1) of an awake mouse containing the genetically-encoded calcium indicator GCaMP6 (green) and optogenetic protein mCherry-channelrhodopsin (red) specifically in somatostatin-type of interneurons.

In a study that could shed new light on normal and abnormal brain function, **Mriganka Sur's** lab used an innovative combination of visual stimuli, short pulses of light and large-scale recording of responses in mice to measure the response times and inhibitory effect of interneurons in the brain.

Sur and Sami El-Boustani focused on inhibitory neurons in the visual cortex — also called interneurons — that use the equivalent of subtraction and division of information to control the cells they target.

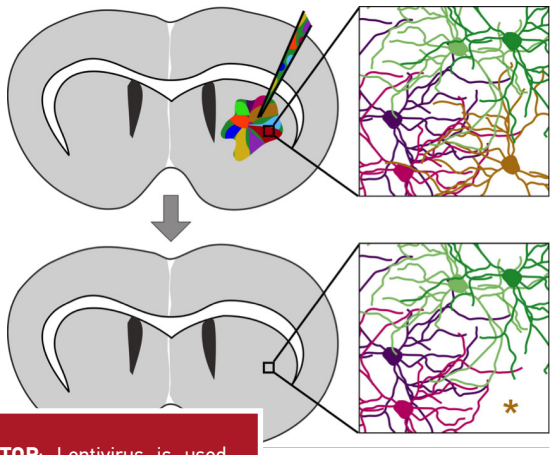
While earlier studies suggested the way these interneurons worked was fixed and unalterable by outside circumstances, the team identified one type of interneuron that can use either subtraction or division to affect the way its target cells respond.

This demonstration of dramatic and distinct response-dependent switching in the live brain suggests that interneuron function is a result of more complex dynamics within the brain's cortical networks.

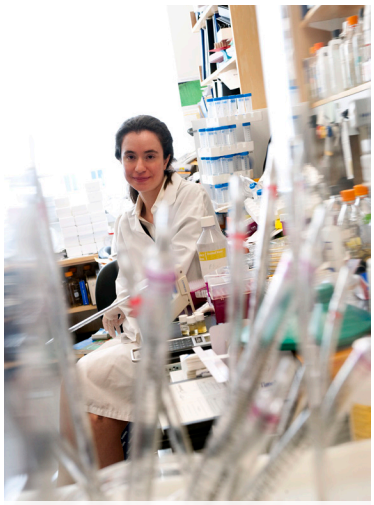
"Neuronal functionality is dynamic: it's governed by firing coordination and the overlap of response timing in circuits and target cells," says El-Boustani, who conducted the research.

And since interneurons play a critical role in disorders like **autism**, **schizophrenia** and **epilepsy**, the results are an important step toward understanding the mechanisms that underlie these conditions.

Published December 11, 2014 in ***Nature Communications***.



TOP: Lentivirus is used to infect brain cells with a "library" of different genetic material into specific parts of a mouse brain. **BOTTOM**: Cells that have received a synthetic lethal hit will die (*) and this can be detected by sequencing the genetic material from remaining lentivirus. If done in parallel on a mouse model of disease, genes that cause this lethality only in combination with a disease-causing mutation can be identified.



Len Rubenstein

Professor Myriam Heiman

New Method Explores Links between *Aging* & *Huntington's Disease*

Neurodegenerative disorders like Alzheimer's, Parkinson's and Huntington's diseases are significantly more likely to develop as we age. Understanding the links between aging and neurodegeneration is an important step toward developing therapeutics for these devastating conditions.

Now a research team led by **Myriam Heiman** has developed a methodology for studying these diseases. Called SLIC – for Synthetic Lethal in the Central Nervous System – the method makes it possible to screen thousands of genes in the central nervous system of mice to identify those that make affected cells vulnerable to neurodegenerative disease.

In vitro screens – performed in a culture dish – can't always pinpoint these genes. But the new method uses in vivo screens – performed in a living animal – that let researchers look at aging factors in the brain as the mice grow older. The result is a much clearer idea of the role aging plays in disease development.

When they used SLIC to study Huntington's disease, the Heiman team identified a gene called Gpx6 that's important in regulating the emergence of HD symptoms in mice. And when they stimulated the gene to overproduce protein in mice with HD, they dramatically alleviated the behavioral and molecular signs of the disease.

"In principle, SLIC can be used to study any neurodegenerative disease for which there's a mouse model," says Reut Shema, first author on the paper. The team plans to use SLIC to generate new hypotheses for neurodegenerative diseases of all types.

Published January 5, 2015 in the ***Proceedings of the National Academy of Sciences***.

Can We Make *Traumatic* Memories Fade?

Traumatic memories can have disabling effects even years after the event that caused them. These overly-persistent fear memories are at the root of anxiety disorders like post traumatic stress disorder. But the mechanisms that make these memories so long-lasting are far from clear.

Now research by **Weifeng Xu** and **A Holmes**, has found a causal connection between a well-known synaptic scaffold protein called PSD-95 and how durable these fear memories are. Synaptic scaffold proteins – found in the synapses of neurons – tether important components in the synapse together for communication and stability.

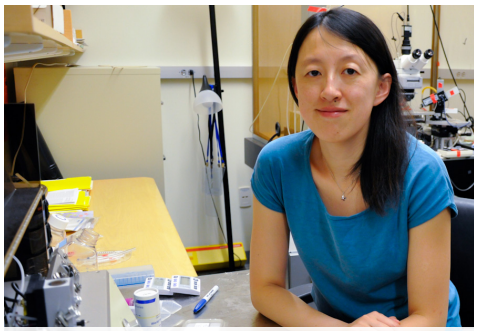
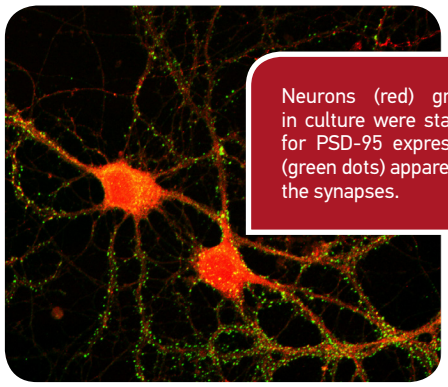


Photo by Azeddine Tahiri

Professor Weifeng Xu



Neurons (red) grown in culture were stained for PSD-95 expression (green dots) apparent in the synapses.

In work with genetically-modified mice, Xu found that PSD-95 was essential for forming precise fear memories and making them last so long. In contrast, mice with little or no PSD-95 in their brains forgot frightening experiences that happened just weeks before.

The research tells us PSD-95 is critical for maintaining traumatic memories over time. And it adds to our understanding of the neural and molecular bases of disorders in which frightening memories are overly persistent.

By showing how these too-persistent fear memories fade, the results have important implications for developing new treatments for trauma-based anxiety disorders.

Published December 16, 2014 in ***Molecular Psychiatry***.

Picower **News**

Photo by Azeddine Tahiri

▲ Picower Professors Elly Nedivi (left), J. Troy Littleton (middle right) and Weifeng Xu (far right) with JPB Foundation President Barbara Picower (center).

Thank you to the *JPB Foundation*

For its generous gift to the Picower Institute Innovation Fund in support of the **high-risk, high-reward research** for which the Picower Institute is known.

Begun six years ago with an initial award from the same foundation, PIIF gives the twelve talented scientists of the Picower Institute the freedom to conduct basic research into some of the most difficult challenges in neuroscience: research that by its daring and inherent risk is often not fundable by traditional sources but can lead to leaps in understanding as opposed to more modest incremental advances.

PIIF-supported research has produced breakthrough discoveries in neurodegenerative and neuropsychiatric disease, generated new insights into how memories are stored and recalled, and produced novel technologies, open-source software, startup companies and important therapeutic advances.

Perhaps most critically, PIIF has allowed Picower researchers to generate the data needed to attract traditional funding and support career transitions for the next generation of scientific leaders.

Photo by Asha Bhakar

▲ From left to right: Tsai Lab graduate student Rebecca Canter explaining her research to MIT's Director of Global Initiatives Marco Munoz, and Alana Foundation President Ana Lucia de Mattos Barretto Villela and Vice-President Marcos Nisti.

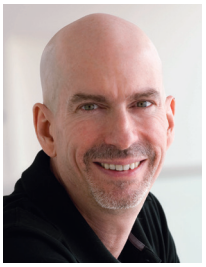
Thank you to Brazil's *Alana Foundation*

For a generous gift that enables collaborative study of the links between **Down syndrome** and **Alzheimer's disease**. The gift – support to the Picower Institute and to Case Western Reserve University – will fund a series of joint studies led by **Li-Huei Tsai**, Director of the Picower Institute and **Alberto Costa** of CWRU with a focus on developing effective new therapies.

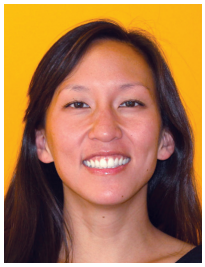
By age 40, the brains of almost all Down syndrome patients show significant levels of the plaques, tangles and abnormal protein deposits that are hallmarks of Alzheimer's disease. Understanding this biological overlap may provide insights into both conditions, and therapies for one may be applicable to the other.

Picower **Accomplishments**

Mark Bear has been honored with the prestigious 2015 **Neuronal Plasticity Prize** of the Fondation IPSEN, awarded to researchers in recognition of outstanding contributions to the field of neuronal plasticity. Created in 1983 under the auspices of the Fondation de France, Fondation IPSEN fosters progress in biomedical research through recognition and support of fundamental advances. Bear is being recognized for his seminal work in genes, synapses and psychiatric disorders.



Earl Miller has launched **SplitSage**, an innovative company that uses neuroscience to revolutionize the ways people view information. SplitSage employs patented analytics to create a "cognitive map" personalized for each user, which maximizes what they see and minimizes what they miss. The company's goal is to increase effectiveness and safety for military and law enforcement, enhance driver safety, and help online retailers and advertisers connect more effectively with customers.



Kay Tye has received the **Harold E. Edgerton Faculty Achievement Award** for 2015 from MIT. The award recognizes exceptional distinction in teaching, research and service to the MIT community by a junior faculty member.

Mette Rathje of Elly Nedivi's lab has won a **Sapere Aude Award for Young Research Talent** from the Danish Council of Independent Research, which recognizes elite researchers across every scientific field.

Rebecca Canter of Li-Huei Tsai's lab and **Laura Stoppel** of Mark Bear's lab have been named **MIT Graduate Women of Excellence** for outstanding contributions to the MIT community.

Upcoming **Events**

For a list of ongoing scientific lectures, colloquia, and workshops, please go to picower.institute

New! The Aging Brain Seminar Series, sponsored by the Picower Institute and the MIT School of Science, is an integral part of the Aging Brain Initiative detailed in this newsletter. The inaugural seminar, held in Cambridge, MA on **May 13**, will be given by **Lennart Mucke**, director and senior investigator at the Gladstone Institute of the University of California San Francisco.

Professor Mucke, a distinguished clinical neurologist and research neuroimmunologist, studies memory loss with an emphasis on Alzheimer's and Parkinson's diseases. His talk will focus on Neuronal and Glial Mechanisms Underlying Cognitive Dysfunction in Alzheimer's Disease.

Aging Brain Seminar Series

The Picower Institute and MIT's MISTI program will collaborate with Germany's European Neuroscience Institute in the MIT/ENI Symposium to be held in Gottingen, Germany on June 13 and 14. The symposium will explore the Synaptic Basis of Neuron Network Dysfunction in Brain Disorders.

On October 6, the Picower Institute Fall Symposium: Synapses in Health and Disease will bring the world's most distinguished neuroscientists to Cambridge to discuss their latest research into synaptic function, with an emphasis on learning, memory and brain disorders.

THE PICOWER INSTITUTE
FOR LEARNING AND MEMORY

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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); Kwanghun Chung, Assistant Professor, Departments of Chemical Engineering and Brain and Cognitive Sciences. 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.
MIDDLE ROW: Earl Miller, Picower Professor of Neuroscience, Department of Brain and Cognitive Sciences; 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.
BOTTOM ROW: 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.

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:

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