Welcome
Welcome to the twenty-fifth Annual Rush and Helen Record Forum and Lectures in Neuroscience, made possible through a generous gift from friends of Rush and Helen Record. Because of their involvement, we have the opportunity to meet at this Forum and focus on the research accomplishments, opportunities and challenges of the past year. This Forum is an important component of our Graduate Program that provides Neuroscience graduate students and postdoctoral fellows a setting in which to present their research findings to the Baylor College of Medicine Neuroscience community. The Forum also provides an opportunity to invite colleagues from other institutions to share their perspective on our research and training activities. We hope everyone will enjoy the forum and come away with new ideas and enthusiasm for our common interest in neuroscience.

Rush and Helen Record
Rush and Helen Record made their home in Houston for over 50 years and together made significant contributions to addressing the causes of mental illnesses. Rush and Helen Record were pioneering advocates of the biological origins of mental illness and spearheaded efforts to support research in the biochemical origins of schizophrenia. Through their leadership the Division of Neuroscience at Baylor College of Medicine was founded under the direction of our first Chair, Dr. James Patrick. Together, the Records have given to all of us, and we are proud to hold this Forum in their honor.

Acknowledgments
Graduate studies in Neuroscience at Baylor College of Medicine are supported by a training grant from the National Institute of General Medical Sciences of the National Institutes of Health, BCM Institutional support, and donations to Baylor College of Medicine by the Sabra Stratton Steed Fellowship, the Tenneco Graduate Student Scholarship Fund and the Record Family. The Forum is made possible by a gift to Baylor College of Medicine by friends of Rush and Helen Record.

The front cover for this year was designed by Gonzalo Viana DiPrisco, a Research Track faculty member in Mauro Costa-Mattioli’s laboratory. The images were taken from Fig 5 and Fig 2 of a review by Keynote Speaker Nicholas Spitzer (Spitzer, N.C. (2012) “Activity-dependent neurotransmitter respecification”. Nature Reviews Neuroscience 13, 94-106). The idea is that activity dependent transmitter expression in neurons can regulate swim behavior in Xenopus laevis larvae. In his own words: “Swim duration is regulated by a circuit that includes the serotonergic neurons in the raphe. Control animals swim once around a small well in response to sensory stimulation. Animals make several circles around the well following reduction in the number of serotonergic neurons by misexpression of voltage-gated sodium channels, introduction of a LIM homeobox transcription factor 1β (Imx1b) morpholino or blockade of 5-hydroxytryptamine 1A (5-HT_{1A}) receptors. Animals manage only half a circumference following an increase in the number of serotonergic neurons by misexpression of inward rectifier potassium channels or addition of 5-HT (serotonin)”.

The faculty, students, and fellows of the Department of Neuroscience wish to express their sincere appreciation to the administrative and technical staff for their outstanding support and assistance for our research and training activities. A special thank you goes to Wanda Waguespack, David Lee, Jay Villarreal, and Meg Ferris whose efforts behind the scenes made this Forum possible.

Paul J. Pfaffinger, Ph.D.
Professor
Director of Graduate Studies
Department of Neuroscience
Baylor College of Medicine

Matthew Rasband, Ph.D.
Professor
Co-Director of Graduate Studies
Department of Neuroscience
Baylor College of Medicine
### Schedule of Events

**Friday, February 20, 2015**

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<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tr>
<td>9:00 – 9:40</td>
<td><strong>Welcome and Breakfast</strong></td>
<td>Expo A, Section 1</td>
</tr>
<tr>
<td>9:40 – 11:30</td>
<td><strong>Session 1 (Dora Angelaki, Moderator)</strong></td>
<td>Expo A, Section 2,3</td>
</tr>
<tr>
<td>9:40 – 10:00</td>
<td>Lucy Liu. <em>Glial Lipid Droplets and ROS Induced By Mitochondrial Defects Promote Neurodegeneration</em></td>
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<tr>
<td>10:00 – 10:20</td>
<td>Alexandra Acevedo. <em>Cocaine Inhibition of Nicotinic Acetylcholine Receptors Influences Dopamine Release</em></td>
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<tr>
<td>10:20 – 11:25</td>
<td>Kwanha Yu, PhD. <em>Decoding the Nature of Astro-Glial Heterogeneity in Malignant Glioma Using a Simple System to Explore a Complex Question: The Role of Pattern Detection and Growth Factor Signaling in Memory Formation in Aplysia</em></td>
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<tr>
<td>11:30 – 1:00</td>
<td><strong>Lunch</strong></td>
<td>Expo A, Section 1</td>
</tr>
<tr>
<td>1:00 – 2:30</td>
<td><strong>Session 2 (Paul Pfaffinger, Moderator)</strong></td>
<td>Expo A, Section 2,3</td>
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<tr>
<td>1:00 – 1:20</td>
<td>Jennifer Johnson. <em>TORC2 Regulation of Aging and Age-Related Memory Impairment</em></td>
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<tr>
<td>1:20 – 1:40</td>
<td>Shan Shen. <em>LM to V1 Feedback Projections Temporally Sharpen the Firing Pattern of V1 Neurons</em></td>
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<tr>
<td>1:40 – 2:00</td>
<td>Joshua White. <em>Cerebellar Function in Dystonia</em></td>
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<tr>
<td>2:00 – 2:30</td>
<td>Javier Medina, PhD. <em>Deciphering the Neural Code for Motor Control in the Cerebellum</em></td>
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<tr>
<td>2:30 – 2:45</td>
<td><strong>Break</strong></td>
<td>Expo A, Section 1</td>
</tr>
<tr>
<td>2:45 – 4:00</td>
<td><strong>Session 3 (Matthew Rasband, Moderator)</strong></td>
<td>Expo A, Section 2,3</td>
</tr>
<tr>
<td>2:45 – 3:05</td>
<td>Sara Kee. <em>Hippocampal Functional Alterations in a Mouse Model of Rett Syndrome</em></td>
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<tr>
<td>3:05 – 3:25</td>
<td>Yu-Mei Huang. <em>αII-Spectrin is Essential for PNS Node of Ranvier Subdomain Formation</em></td>
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<tr>
<td>3:25 – 3:55</td>
<td>Jeannie Chin, PhD. <em>Epigenetic Regulation of Cognition in Neurological Disease</em></td>
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<tr>
<td>4:00 – 5:00</td>
<td><strong>Student Meeting with Advisors</strong></td>
<td>Expo A, Section 2,3</td>
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<tr>
<td>5:00 – 7:00</td>
<td><strong>Poster Session</strong></td>
<td>Expo A, Section 1</td>
</tr>
<tr>
<td>7:00 – 9:00</td>
<td><strong>Dinner/Keynote Address/Awards</strong></td>
<td>Expo A, Section 1</td>
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<tr>
<td>9:00:</td>
<td><strong>Neuroscience Social</strong></td>
<td>Garden Cay</td>
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**Introductions – Dora Angelaki, PhD**
- Introduction of special guests
- Presentation of the Rush and Helen Record Fellow in Neuroscience Award
- Neuroscience Educator Awards

**Rush and Helen Record Neuroscience Forum Keynote Lecture**
- Nicholas Spitzer, PhD. *Neurotransmitter Switching in the Adult Brain*
### Schedule of Events
Saturday, February 21, 2015

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<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tr>
<td>7:30 – 9:00</td>
<td><strong>Buffet breakfast</strong></td>
<td>Expo A, Section 1</td>
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<tr>
<td>9:00 – 10:10</td>
<td><strong>Session 4 (Andrew Groves, Moderator)</strong></td>
<td>Expo A, Section 2,3</td>
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<tr>
<td>9:00 – 9:30</td>
<td>Jeff Yau, PhD. <em>Human Auditory Cortex Represents Tactile Temporal Frequency</em></td>
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<td>9:30 – 9:50</td>
<td>George Denfield. <em>The Role of Internal Signals in Structuring V1 Population Activity</em></td>
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<tr>
<td>9:50 – 10:10</td>
<td>Angie Chiang. <em>Combination Therapy Maximizes Cognitive Recovery in a Mouse Model of Alzheimer’s Disease</em></td>
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<tr>
<td>10:10 – 10:25</td>
<td><strong>Break</strong></td>
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<tr>
<td>10:25 – 11:30</td>
<td><strong>Session 5 (Fabrizio Gabbiani, Moderator)</strong></td>
<td>Expo A, Section 2,3</td>
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<tr>
<td>10:25 – 10:40</td>
<td>Xiangling Meng. <em>Deficiency of MeCP2 in Glutamatergic Neurons Leads to Distinct Features Compared to Gabaergic Conditional Deletion</em></td>
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<tr>
<td>10:40 – 11:00</td>
<td>Claudia Huichalaf, PhD. <em>Cross-Species Genomic Screen for Therapeutic APP Reduction in Alzheimer’s Disease</em></td>
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<tr>
<td>11:00 – 11:30</td>
<td>Mingshan Xue, PhD. <em>Cortical Excitation and Inhibition in Health and Disease</em></td>
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<tr>
<td>11:30 – 1:30</td>
<td><strong>Lunch/Talk Awards</strong></td>
<td>Expo A, Section 1</td>
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*Adjourn*
Nick Spitzer received his Ph.D. from Harvard University and was a postdoctoral fellow at Harvard and University College, London. He joined the faculty of the University of California, San Diego, in 1972 and has been the recipient of a Sloan Fellowship, a Javits Neuroscience Investigator Award and a Guggenheim Fellowship. He is editor-in-chief of BrainFacts.org, a fellow of the American Association for the Advancement of Science, a member of the American Academy of Arts and Sciences and the National Academy of Sciences and director of the UCSD Kavli Institute for Brain and Mind.

His lab has discovered that spontaneous transient elevations of intracellular calcium, generated by ion channels and receptors, control several aspects of differentiation during an early period in embryonic development. Continued work is aimed at understanding the roles of electrical activity in assembly of the nervous system, by analyzing the effects of calcium transients on neuronal differentiation and determining the molecular mechanisms by which they exert these effects.

Specification of neurotransmitters and selection of transmitter receptors are processes that depend on patterned spontaneous embryonic calcium-dependent electrical activity. The Spitzer lab is investigating the triggers of this spontaneous activity to understand its origins. They study activity-dependent regulation of expression of serotonin and dopamine in the embryonic brain, because these transmitters have broad impact on cognitive states and on behavior. The Spitzer lab also has begun to analyze the signaling mechanisms mediating activity-dependent transmitter specification, generating transgenic lines expressing fluorescent reporters of neurotransmitter synthesis to enable mutant screens. The aim is to determine the extent to which there is environmental regulation of activity-dependent differentiation at early stages of development, revealing a partnership of electrical activity and genetic programs in the assembly of the nervous system.

Past Rush and Helen Record Lectures

1990  Michael Merzenich, Ph.D.
Professor of Otolaryngology and a Neuroscientist
University of California, San Francisco
Member, National Academy of Sciences
Origins of Learning and Non–Declarative Memory in the Cerebral Cortex

1992  Stephen F. Heinemann, Ph.D.
Professor of Molecular Neurobiology Laboratory
Salk Institute for Biological Studies
Member, National Academy of Sciences and the Institute of Medicine
The Glutamate Receptors Family: Structure, Function and Expression in the Brain

1993  James W. Patrick, Ph.D.
Professor and Head for the Division of Neuroscience
Baylor College of Medicine
The Diversity of Neuronal Nicotinic Acetylcholine Receptors

1994  Timothy Bliss, Ph.D.
Head of the Division of Neurophysiology and Neuropharmacology
National Institute of Medical Research
LTP Comes of Age (1973 – 1994): Where Now?
### 25th Rush and Helen Record Neuroscience Forum

**Baylor College of Medicine**

<table>
<thead>
<tr>
<th>Year</th>
<th>Name and Affiliation</th>
<th>Topic</th>
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</table>
| 1995 | Bert Sakmann, Ph.D.  
Director, Department of Cell Physiology  
Max-Planck-Institute for Medical  
Member, National Academy of Sciences  
1991 Nobel Prize in Physiology or Medicine | Electrical Properties of Dendrites Modulate Integrative Functions of Neurons |
| 1996 | Randall R. Reed, Ph.D.  
Professor, Departments of Molecular Biology and Genetics and of Neuroscience, Johns Hopkins University School of Medicine | The Genetics of Olfaction |
| 1997 | Zach W. Hall, Ph.D.  
President and CEO of Envivo Pharmaceuticals Inc.  
Member, Institute of Medicine | Assembly of the Synapse |
| 1998 | William T. Newsome, III, Ph.D.  
Professor of Neurobiology, Stanford University School of Medicine  
Member, National Academy of Sciences | Converting Sensory Signals into Perceptual Decision: Perspectives |
| 1999 | Erwin Neher, Ph.D.  
Director of the Department Membranbiophysik theMax-Planck-Institut für biophysikalische Chemie | Trying to Understand Short–Term Plasticity in Quantitative Terms |
| 2000 | Eric Nestler, M.D., Ph.D.  
Elizabeth Mears and House Jameson Professor of Psychiatry and Neurobiology.  
Yale University.  
Member, Institute of Medicine | Molecular Mechanisms of Drug Addiction |
| 2001 | Gail Mandel, Ph.D.  
Senior Scientist, The Vollum Institute  
Oregon Health & Science University | Regulation of Neuronal Phenotype by Transcriptional Repression: How to Quiet Your Nerves |
| 2002 | Lorna Role, Ph.D.  
Professor, Department of Anatomy and Cell Biology Center for Neurobiology and Behavior, Columbia | Cholinergic Modulation of Central Synapses Involved in Pain and Gain |
| 2003 | Simon Laughlin, Ph.D.  
Rank Research Professor in Opto–Electronics  
Department of Zoology, University of Cambridge | Energy, Information and the Design of Brains |
| 2004 | Alcino Silva, Ph.D.  
Professor, Departments of Neurobiology, Psychiatry, and Psychology, Brain Research Institute, UCLA | Unraveling the Molecular and Cellular Biology of Permanent Memory |
| 2005 | Eric Knudsen, Ph.D., The Edward C. and Amy H. Sewall Professor in the School of Medicine and Chair, Department of Neurobiology, Stanford School of Medicine.  
Member, National Academy of Sciences | Mechanisms of Learning in the Auditory System of the Barn Owl |
| 2006 | Richard Tsien, Ph.D.  
George D. Smith Professor of Molecular and Genetic Medicine, Department of Molecular and Cellular Physiology, Stanford University.  
Member, National Academy of Science and the Institute of Medicine | Unexpected Signaling at the Synapse |
2007  **David S. Weiss, Ph.D.**  
Professor and Chair, Department of Physiology  
University of Texas Health Science Center at San Antonio  
Emerging Views on GABA Receptor Structure and Function

2008  **Jonathan Cohen, M.D., Ph.D.**  
Eugene Higgins Professor of Psychology  
Co-Director, Princeton Neuroscience Institute, Princeton University  
The Vulcanization of the Human Brain: Neuroimaging Studies of Cognition-Emotion Interactions in Decision Making

2008  **Craig Jahr, Ph.D.**  
Senior Scientist, The Vollum Institute  
Professor, Department of Cell and Developmental Biology, School of Medicine, Oregon Health and Science University  
Neural-glial Interactions in the CNS

2009  **Craig Garner, Ph.D.**  
Professor of Psychiatry and Co-Director of the Down Syndrome Center, Stanford Univ School of Medicine  
Cellular and Molecular Mechanisms of Presynaptic Active Zone Assembly and Function

2009  **Elizabeth Gould, Ph.D.**  
Professor of Psychology, NARSAD Distinguished Investigator, Princeton University  
Structural Plasticity in the Adult Brain

2010  **Harold W. Sontheimer, Ph.D.**  
Professor, Department of Neurobiology. Director, Center for Glial Biology in Medicine, Director, Civitan International Research Center  
University of Alabama at Birmingham  
New Treatments for Primary Brain Tumors: Ion Channels and Amino-acid Transporters as Drug Targets

2011  **Joshua R. Sanes, Ph.D.**  
Professor of Molecular & Cellular Biology  
Director, Center for Brain Science, Harvard University  
Visualizing Circuits in the Visual System

2012  **Karl Deisseroth, M.D., Ph.D.**  
Associate Professor of Bioengineering and Psychiatry  
Stanford University  
Optogenetics: Application and Development

2013  **Cornelia Bargmann, Ph.D.**  
Investigator, Howard Hughes Medical Institute  
Torsten N. Wiesel Professor  
Lulu and Anthony Wang Laboratory of Neural Circuits and Behavior  
The Rockefeller University, New York  
Using Fixed Neural Circuits to Build Flexible Behaviors

2014  **Gilles Laurent, Ph.D.**  
Director, Max Planck Institute of Brain Research  
Frankfurt, Germany  
Explorations of a simple visual cortex

Please note that the speakers’ professional titles listed are from the time of their keynote lecture.
Thomas J. Carew, Ph.D.

Thomas J. Carew received his Ph.D. from the University of California, Riverside. For several years he was a member of the faculty of Columbia Medical School before moving to Yale, where he was the John M. Musser Professor of Psychology and a Professor of Molecular, Cellular and Developmental Biology. He is a former Chair of the Department of Psychology at Yale, a Fellow of the American Psychological Association and the American Psychological Society, an elected Member of the Society for Experimental Psychology, an elected Fellow of the AAAS, an elected fellow of the American Academy of Arts and Sciences, and a past Councilor of the Society for Neuroscience. He served as President of the Society for Neuroscience in 2008. He is also the recipient of several awards, including a MERIT Award from the National Institute of Mental Health, the Yale College Dylan Hixon Prize for excellence in teaching in the natural sciences, and the Chancellor's Award for Excellence in Undergraduate Research at UCI. He is a member of numerous Editorial and Advisory Boards.

From 2000 to 2011 Dr. Carew held an endowed chair at the University of California, Irvine, where he was a Bren Professor and Chair of the Department of Neurobiology and Behavior. In July, 2011 Dr. Carew became the Anne and Joel Ehrenkranz Dean of the Faculty of Arts and Science at New York University, where he is also a Professor of Neural Science.

Jennifer Raymond, Ph.D.

Jennifer Raymond, Ph.D. is an Associate Professor of Neurobiology and Associate Dean in the Office of Diversity and Leadership at the Stanford University School of Medicine. She received her B.A. in Mathematics from Williams College and her Ph.D. in Neuroscience from the University of Texas Health Science Center at Houston. Her dissertation work examined the neural events controlling the induction of plasticity in *Aplysia californica*. As a postdoc with Steve Lisberger at UCSF, she extended her study of the neural learning rules controlling the induction of plasticity to the mammalian cerebellum, and has continued this work in her lab at Stanford. Her research employs a broad range of experimental approaches, including behavioral analysis, *in vivo* physiology, *in vitro* physiology, molecular-genetic approaches and computational approaches, with the goal of functionally linking events occurring at different levels of organization in the nervous system.

Dr. Raymond is an award-winning teacher, and has initiated a number of programs at Stanford to improve graduate education and training outcomes. She also has worked on best practices in scientific training at the national level, as a member of the Education Committee and the Committee on Neuroscience Departments and Programs of the Society for Neuroscience. As Associate Dean, her work focuses on redesigning the faculty career track to accommodate and attract the next generation of scientists.
# Neuroscience Primary Faculty

- Dora Angelaki, PhD, Chair
- Jeannie Chin, PhD
- Mauro Costa-Mattioli, PhD
- Benjamin Deneen, PhD
- J. David Dickman, PhD
- David M. Eagleman, PhD
- Fabrizio Gabbiani, PhD
- Andrew Groves, PhD
- Joanna L. Jankowsky, PhD
- Javier Medina, PhD
- Paul J. Pfaffinger, PhD
- Xaq Pitkow, PhD
- Matthew N. Rasband, PhD
- Russell Ray, PhD
- David Ress, PhD
- Melanie Samuels, PhD
- H. David Shine, PhD
- Stelios M. Smirnakis, MD, PhD
- Andreas S. Tolias, PhD
- Kimberly R. Tolias, PhD
- Mingshan Xue, PhD
- Jeffrey Yau, PhD

# Neuroscience Joint Faculty

- Anne E. Anderson, MD
- Benjamin R. Arenkiel, PhD
- Scott F. Basinger, PhD
- Michael Beauchamp, PhD
- Hugo J. Bellen, DVM, PhD
- William E. Brownell, PhD
- Ching-Kang Jason Chen, PhD
- Gary D. Clark, MD
- Edward C. Cooper, MD, PhD
- Richard De La Garza II, PhD
- Herman A. Dierick, MD
- Frank T. Horrigan, PhD
- Daoyun Ji, PhD
- Thomas R. Kosten, MD
- Hui-Chen Lu, PhD
- Mirjana Maletic-Savatic, PhD
- Shailaja K. Mani, PhD
- Graeme Mardon, PhD
- Jeffrey L. Noebels, MD, PhD
- Bert O’Malley, MD
- Paul A. Overbeek, PhD
- Robia G. Pautler, PhD
- Richard E. Paylor, PhD
- Claudia S. Robertson, MD
- Joshua Shulman, MD, PhD
- Roy V. Sillitoe, PhD
- Lane Strathearn, MBBS, PhD
- John W. Swann, PhD
- Bob Thalman, PhD
- Meng Wang, PhD
- Theodore G. Wensel, PhD
- Samuel M. Wu, PhD
- Daniel Yoshor, MD
- Hui Zheng, PhD
- Huda Y. Zoghbi, PhD

# Neuroscience Adjunct Faculty

- Caleb Kemere, PhD
  - Assistant Professor
  - Electrical & Computer Engineering
  - Rice University
- Jacob Robinson, PhD
  - Assistant Professor
  - Electrical & Computer Engineering
  - Rice University
- Adam Zaidel, PhD
  - Senior Lecturer & Researcher
  - Bar Ilan University
  - Israel

# Neuroscience Research Track Faculty

- Kelly Anne Barnes, PhD
- Martin L. Basch, PhD
- Kalpana Dokka, PhD
- Joseph G. Duman, PhD
- Henry Jerng, PhD
- Xiaolong Jiang, PhD
- Smita Jha, PhD
- Eliana M. Klier, PhD
- Baowang Li, PhD
- Sheng Liu, PhD
- Gonzalo Viana Di Prisco, PhD
- Saumil Patel, PhD
- Ari Rosenberg, PhD
- Michael Shinder, PhD
- Chuansheng Zhang, PhD
- Ping Jun Zhu, PhD
25th Rush and Helen Record Neuroscience Forum
Baylor College of Medicine

Neuroscience Postdoctoral Researchers

Marife Arancillo, PhD
Hannah Arnson, PhD
James Bridgewater, PhD
Shelly A. Buffington, PhD
Zhao Lin Cai, PhD
Wu Chen, PhD
James Cotton, PhD
An Dao, PhD
Richard B. Dewell, PhD
Alexander Ecker, PhD
Renee Edlund, PhD
Reuben Fan, PhD
Jose Fernandez Leon Fellenz, PhD
Emmanouil Froudarakis, PhD
Stacey Glasgow, PhD
Stacey Grunke, PhD
Hamdan Hamdan, PhD
Gabe Haarsma, PhD
Tammy Ho, PhD
Claudia Huichalaf-Navarette, PhD
Madhu Keralapurath, PhD
Byoungoon Kim, PhD
Christian Lasagna-Reeves, PhD
Hyun Kyoung Lee, PhD
Sang Kyun Lee, PhD
Nele Annika Lefeldt, PhD
Caitlyn Elmore Limonciello, PhD
Chia-Ching Lin, PhD
Hui Lu, PhD
Jochen Meyer, PhD
Zakir H. Mridha, PhD
Shalaka Mulherkar, PhD
Dona Murphy, PhD
Pablo Ormachea, JD
Ganna Palagina, PhD
Alexis Perez-Bellido, PhD
Angelique Regnier-Golanov, PhD
Jacob Reimer, PhD
Aram Saravani, PhD
Sunita Singh, PhD
Konstantinos Sousounis, PhD
Loredana Stoica-Ghita, PhD
Krishnamurthy Subramaniam, PhD
Adhira Sukara, PhD
Tomohiro Torii, PhD
Sergey P. Torsky, PhD
Wangchen Wang, PhD
Hongxia Wang, PhD
Le-Qing Wu, PhD
Dimitri Yatsenko, PhD
Rong Zhao, PhD
Zhiyuan Zhang, PhD

Neuroscience Graduate Students

Alexandra Acevedo-Rodriguez
Hunter Allen
Ryan Ash
Amanda Brown
Cathryn Cadwell
Mingbo Cai
Angela Carter
Henry Cham
Chien-Ju Chen
Chi An (Angie) Chiang
Sarah Clupek
Lexi Crommett
George Denfield
Joel Eisenhofer
Paul Fahey
Olivia Fitch
Courtney Garcia
Savannah Gosnell
Caiwei Guo
Elizabeth Halfen
Asante Hatcher
Longwen Huang
Yu-Mei (Claire) Huang
Chih-Chun Hsu
Jennifer Johnson
Sara Kee
Elizabeth Lackey
Kaushik Lakshminarasimhan
Chih-hong Lee
Amber Levine
Steven Lien
Brian Lim
Lu (Lucy) Liu
Miguel (Alec) Marin
Xiangling (Cathy) Meng
Jessica Messier
Fatima Saldana Morales
Lena Nguyen
Mario Oyola
Jay Patel
Jaclyn Patterson
Amy Pohodich
Jasmine Rah
Shoaibur Rahman
Jasdeep Sabharwal
Shan Shen
Hongsup Shin
Trace Stay
Sharon Stevens
Jenny Sun
Baouyen Tran
Meike van der Heijden
Edgar Walker
Joshua White
Chun-Ting Wu
Michael Yetman
Daniel Zollinger
### Students from other PhD Programs in Neuroscience Labs

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<thead>
<tr>
<th>Name</th>
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<th>City, State</th>
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<tr>
<td>Mussie Araya</td>
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<td>Philipp Berens</td>
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<td>Rogers Brown II</td>
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<td>Onur Birol</td>
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<td>Jinxuan Cheng</td>
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<td>Hsin I Jen</td>
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<td>Tongchao Li</td>
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<td>Scott Novich</td>
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<td>Jiyoungh Park</td>
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<td>Yen-Kuei (Peter) Tu</td>
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<td>Ricky Savjani</td>
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<td>Chih-Chuan Wang</td>
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<td>Wenyi Zhu</td>
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<tr>
<td>Ying Zhu</td>
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### Neuroscience Visiting Student Candidates

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<thead>
<tr>
<th>Name</th>
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<tbody>
<tr>
<td>Erin Boldt</td>
<td>Trinity University</td>
<td>San Antonio, Texas</td>
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<tr>
<td>Jiakun Fu</td>
<td>Skidmore College</td>
<td>Saratoga Springs, New York</td>
</tr>
<tr>
<td>Robert Brockman</td>
<td>Rice University</td>
<td>Houston, Texas</td>
</tr>
<tr>
<td>Margaret Hayne</td>
<td>University of Wisconsin</td>
<td>Madison, Wisconsin</td>
</tr>
<tr>
<td>Youtong Huang</td>
<td>University of California</td>
<td>Los Angeles, California</td>
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An increase in lipid droplets (LD) has been implicated in some metabolic disorders but their role in neurodegeneration (ND) is ill defined. Through an unbiased forward genetic screen to uncover genes that lead to ND in photoreceptors, we identified various nuclear encoded genes that affect mitochondrial function, including fusion, translation, and complex I function. Mutations in these genes lead to a transient but severe accumulation of LD in glia prior to the onset of ND. These mutants exhibit increased levels of reactive oxygen species (ROS), which promote c-Jun-N-terminal Kinase (JNK) and Sterol Regulatory Element Binding Protein (SREBP) activity in neurons, leading to LD accumulation in glia. These LDs are peroxided and cause to the demise of neurons. However, ND can be significantly delayed with the cell specific reduction of ROS, JNK or SREBP levels, or by overexpressing lipases. Importantly, a similar pathway leads to glial LD accumulation in Ndufs4 mutant mice, suggesting that LD accumulation following mitochondrial dysfunction is an evolutionarily conserved phenomenon. Importantly, brief administration of antioxidants to mutant flies and Ndufs4 mutant mice significantly delays the onset of ND. We show a novel model for ND based on increased ROS in neurons that leads to LD accumulation in glia. Preventing LD accumulation or reducing ROS delays the demise of neurons. This evolutionarily conserved synergism between ROS and LD may be a biomarker and accelerator of neurodegenerative disease.

Cocaine inhibition of nicotinic acetylcholine receptors influences dopamine release.


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Cocaine is a highly abused drug resulting in a large economic burden, and its main mode of action is the blockade of dopamine (DA) transporters. This allows for DA to remain in the synapse thereby prolonging DA signaling. In the brains of abusers, cocaine can reach concentrations of 5-10µM, and at this concentration, cocaine inhibits nicotinic acetylcholine receptors (nAChRs). This nAChR inhibition may play a prominent role in DA release as DA neurons highly express nAChRs with β2 subunits. We hypothesized that inhibition of these nAChRs may decrease DA release in the striatum. Using fast-scan cyclic voltammetry, electrically evoked DA release was measured in brain slice preparations following blockade of DA transporters and then subsequent bath administration of 10µM cocaine. Cocaine blocked nAChRs and decreased DA release which was mediated by β2 nAChRs. DA has two modes of release with a slower frequency release corresponding to basal DA levels termed tonic DA release and a higher frequency release in response to behaviorally relevant stimuli termed phasic DA release. Nicotinic AchR mediated inhibition of DA release has a stronger influence on tonic release compared to phasic release in the dorsolateral striatum. This selective inhibition of tonic release increases the contrast between phasic and tonic release possibly contributing to the enhanced motivational value of cocaine associated memories and behaviors.

Decoding the nature of astro-glial heterogeneity in malignant glioma.

Kwanha Yu1, Jeff Carlson1, John C Lin1, Wendy Zhu1, Nabil Ahmed2, Xiao-nan Li2, Carrie Mohila2, Michelle Monje3, Akash J Patel4, Benjamin Deneen1

1Department of Cell and Gene Therapy, Baylor College of Medicine, Houston, TX; 2Texas Children’s Hospital, Houston, TX; 3Department of Neurology and Neurological Sciences, Stanford University, Stanford, CA; and 4Department of Neurosurgery, Baylor College of Medicine

Glioma is the most common malignancy of the central nervous system with glioblastoma multiforme (GBM) being the most frequent (60%) and deadliest form. Despite significant clinical advances, a median survival of ~15 months for GBM patients has remained relatively unchanged for six decades. GBM consists of a heterogeneous mixture cells, primarily comprised of malignant astro-glia. In their non-disease, astro-glia cells also demonstrate extraordinary diversity. Therefore, it would stand to reason that this astro-glial diversity contributes to GBM heterogeneity. To tackle the question of cellular heterogeneity in GBM, we have employed a novel, mouse glioma model capable of generating pathologically verified GBM in less than 3 weeks. Combining this high throughput, in vivo model with flow cytometry and FACS, we identified 5 novel subpopulations that exhibit functional differences. Analysis of primary human tissue indicates that these subpopulations are present in the human disease, and analysis of primary cells lines suggests the functional properties are conserved in humans. Together, this multi-species, multi-modal approach demonstrates that subpopulations outside the previously identified cancer stem cell and vasculature exhibit differential tumorigenic properties and also suggests these properties are conserved across species.

TorC2 regulation of aging and age-related memory impairment.

Jennifer Johnson1, Wei Huang1, Gregg Roman2, Herman Dierick1, Mauro Costa-Mattiol1

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As our population ages, cognitive decline and dementia are becoming more prevalent. In the United States, there are currently 4.7 million people with dementia. By 2050, the number is expected to double. Memory declines with age indicating crosstalk between these two processes. Yet, mechanisms are not fully understood nor have shared components been identified. This information is critical as different mechanisms of lifespan extension may benefit certain functions and impair others such as memory. One important example is the Target of Rapamycin Complex 2 (TorC2) signaling pathway. Given the evolutionary conservation of TorC2 between flies and humans, we measured lifespan in flies lacking torc and sin1, essential components of TorC2 formation. We found that TorC2 mutants are significantly long lived, suggesting that TorC2 regulates lifespan in the fly. In addition, given that TorC2 is required for long-term memory (LTM) and mTorC2 activity declines with age in mice, we asked whether TorC2 dysregulation contributes to age-related memory impairment. Intriguingly, we found that a specific small molecule that activates TorC2, A-443654, restored the impaired LTM in aged wild-type flies and mice. These results suggest that TorC2 plays a role in age-related memory impairment via an evolutionarily conserved mechanism. Ongoing work will determine if TorC2-regulated memory and aging processes can be uncoupled. The knowledge gained in this study will lead to a better understanding of the molecular, cellular and neuronal circuit mechanisms underlying aging and age-related cognitive impairment.
**O-05**

**LM TO V1 FEEDBACK PROJECTIONS TEMPORALLY SHARPEN THE FIRING PATTERN OF V1 NEURONS**

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The mammalian visual system is composed of multiple hierarchically organized cortical areas that extract progressively more complex features of the visual scene as information from the retina is fed forward from one cortical area to the next. These multiple visual areas are also extensively connected via feedback pathways, which allow the information extracted by higher areas in the visual pathway to influence more primitive visual responses in earlier areas such as primary visual cortex (V1). It has been shown that lateral medial area (LM) send a large amount of feedback projections to V1, but the functions of these projections are largely unknown. To dissect the feedback circuit from LM to V1, we injected adeno-associated virus (AAV) expressing channelrhodopsin 2 (ChR2) in LM. We found that LM to V1 feedback projections mainly target the retinotopically related area in V1. We recorded from different types of neurons in V1 across different layers with multi-patch whole-cell slice recording, while photostimulating the axon terminals of LM to V1 feedback neurons. We found that among all the cell types we recorded from, L2/3 parvalbumin (PV) positive cells and somatostatin (SST) positive cells receive strongest monosynaptic excitatory input from feedback activation. Pyramidal cells also receive monosynaptic excitatory input, but the amplitude is much smaller. The net effect of feedback excitation to V1 pyramidal cells is a depolarization followed by a hyperpolarization. Consistently, when we made the pyramidal cell fire by injecting current in slices, we found that the photostimulation of feedback axon terminals temporally sharpened the firing of pyramidal cells. Similar effects were found in vivo. Our results indicate that feedback projections from LM to V1 can help precisely control the firing time of V1 pyramidal cells.

**O-06**

**CEREBELLAR FUNCTION IN DYSTONIA**

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Dystonia is a severe neurological disorder that can cause painful muscle contractions. Although recent work has identified several brain regions that are altered in dystonia, a major obstacle to understanding why and how the brain sends erroneous signals to the muscles has been the lack of an accessible animal model. While genetically engineered mouse models have been useful for defining the molecular mechanisms of dystonia, none of these mice show dystonic motor behaviors. And although there are spontaneous mutant models that do show dystonia, they also have unrelated pathological defects. Additionally, despite the severe dystonia that can be induced by certain chemicals, the methods of delivery often damage brain circuits and the dystonia that's induced is transient and variable. To overcome these limitations, we developed an inducible dystonia model by using conditional genetics approaches to target the olivo-cerebellar pathway, a connection that may be central to the defects in humans and animal models with dystonia. Here, we show using a combination of high-resolution anatomy, behavioral paradigms and in vivo awake electrophysiology that loss of olivo-cerebellar synaptic transmission induces abnormal burst firing within the cerebellar circuit, and causes severe muscle co-contractions that produce dystonic movements. We also used pharmacologic and deep brain stimulation approaches to overcome the dystonic phenotype by blocking the abnormal cerebellar output. Our new model offers a unique opportunity to determine the circuit defects that trigger dystonia and an ideal system for testing therapeutic strategies in a model that displays obvious dystonia.

**O-07**

**HIPPOCAMPAL FUNCTIONAL ALTERATIONS IN A MOUSE MODEL OF RETT SYNDROME**

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Rett Syndrome (RTT) is a neurological disorder affecting primarily girls, characterized by pervasive learning disability, along with motor and autonomic dysfunction. RTT is caused by loss of function mutations to the X-linked gene MECP2, which encodes a transcription regulator that binds to methylated CpGs. The Mecp2⁻/⁻ mouse model recapitulates most of the phenotypes seen in the human disease, including learning and memory deficits. A candidate for the neural circuit mediating some of these behaviors is the hippocampus (HP), which is a brain area essential for learning and memory. In particular, spatial memories are thought to be represented by the activities of hippocampal neurons. The synchronization, or finely tuned timing, of hippocampal neuronal activity is required for intact memory encoding and consolidation. Therefore, we are interested in studying the hippocampus in naturally behaving animals of the Mecp2 mouse model, specifically to discern the circuit properties that lead to the spatial memory deficits. Our hypothesis is that, improper synchronization of hippocampal neuronal firing activity leads to failure in memory consolidation and poor spatial memory representation. Our preliminary data suggests that there is a deficit in spatial memory consolidation in this model of Rett Syndrome.

**O-08**

**ALL-SPECTRIN IS ESSENTIAL FOR PNS NODE OF RANVIER SUBDOMAIN FORMATION**

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All-spectrin is a submembranous cytoskeletal protein highly expressed in both neurons and myelinating glia. It is implicated in the neurological disorder, West syndrome, which is the infantile spasm with symptoms including cerebral hypomyelination, epilepsy and brain atrophy. Moreover, the all-spectrin constitutive knockout mice die in utero with nervous system malformations. Therefore, I generated all-spectrin conditional knockout (cko) mice to bypass the embryonic lethality and to identify the roles of neuronal and glial all-spectrin in myelination, node of Ranvier formation and axon integrity.

When all-spectrin is deleted in the CNS (Nestin-cre), mutant animals die perinatally. Nestin-cre cko mice are smaller and they have motor coordination deficits. Besides, immunohistochemistry (IHC) shows that neurodegeneration occurs throughout the brain and cko mice have defects in neuronal migration in the cerebral cortex while Purkinje cells are dramatically reduced in the cerebellum. Furthermore, axon initial segments are fragmented and remarkably reduced.

all-spectrin is eliminated exclusively in myelinating glia by crossing CNP-cre mice with floxed mice. Surprisingly, myelin and the nodes of Ranvier subdomains can still form. On the other hand, when neuronal all-spectrin is knocked out only in the periphery sensory nervous system (adillin-cre), animals have hind leg clasping and much worse motor coordination. IHC shows that mutant mice have less node number and intensity. Moreover, paranodes are extensively disrupted and there is less potassium channel clustering and the localization is aberrant. In addition, the conduction velocity of dorsal root is dramatically reduced, examined by compound action potential recording. This study will reveal the function of all-spectrin and provide insights into the different roles of spectrin networks in neurons and in myelinating glia.
O-09 THE ROLE OF INTERNAL SIGNALS IN STRUCTURING V1 POPULATION ACTIVITY

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Neuronal responses to repeated presentations of identical visual stimuli are variable. The cause of this variability is unknown, but it is commonly treated as noise and seen as an obstacle to understanding neuronal activity. We offer an alternative explanation: this variability is not noise but reflects, and is due to, computations internal to the brain. Internal signals such as cortical state or attention interact with sensory information processing in early sensory areas. However, little research has examined the effect of fluctuations in these signals on neuronal responses, leaving a number of uncontrolled parameters that may contribute to neuronal variability. One such variable is attention. We hypothesize that fluctuations in attentional signals contribute to neuronal response variability and that controlling for such fluctuations will reduce this variability. To study this interaction, we use multi-electrode recordings with laminar probes in primary visual cortex of macaques while subjects perform a cued-spatial attention, change-detection task. We induce varying degrees of fluctuation in the subject’s attentional signal by changing whether the subject must attend to one stimulus location while ignoring another, or attempt to attend to both locations simultaneously. We demonstrate that attention increases stimulus-evoked firing rates and gain-modulates the tuning curves of V1 neurons in a manner that is consistent with results from higher order areas. Future experiments will examine the effect of attentional fluctuations on neuronal response variability and interneuronal correlations as well as the laminar profile of these effects. Under this hypothesis, this variability can aid, rather than hinder, our understanding of brain function.

O-10 COMBINATION THERAPY MAXIMIZES COGNITIVE RECOVERY IN A MOUSE MODEL OF ALZHEIMER’S DISEASE

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Antibodies against various Aβ epitopes have been shown to reduce amyloid levels in animal models of AD and many are being actively pursued in clinical trials. Despite this progress, we know little about which species of the peptide - soluble, insoluble, or both - should be targeted for maximal benefit. More specifically, is slowing the aggregation of newly synthesized Aβ sufficient for cognitive recovery, or must we also remove existing deposits to achieve full functional rescue? Using the controllable tet-off APP transgenic model of AD, we have shown that combining two complementary approaches for Aβ reduction – using passive immunization to sequester existing peptide, while simultaneously suppressing transgenic APP to reduce further Aβ production – improves neuropathological outcome over either treatment alone. In this model, we found that both monotherapies forestalled further plaque deposition, while combination treatment not only halted accumulation but also cleared existing deposits. In the current study, we tested whether combination treatment and concomitant plaque clearance provide any cognitive benefit over plaque stasis through passive immunization or Aβ suppression alone. Following 9 weeks of treatment, animals underwent behavioral testing to examine spatial learning, working memory, and associative memory. Preliminary findings suggest that combination treatment produces greater improvement in cognitive performance than either passive immunization or Aβ suppression alone. These results may guide development of future therapeutics by demonstrating that greatest cognitive benefit is attained when all forms of Aβ are reduced.

O-11 DEFICIENCY OF MeCP2 IN GLUTAMATERGIC NEURONS LEADS TO DISTINCT FEATURES COMPARED TO GABAergic CONDITIONAL DELETION

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Rett Syndrome (RTT) is a postnatal neurological disorder caused by loss of function mutations in the gene encoding methyl-CpG-binding protein 2 (MeCP2). Deleting Mecp2 only from brain tissue at embryonic day 12 leads to phenotypes identical to those of the null mutation, indicating that loss of MeCP2 from the CNS is responsible for the RTT phenotypes. Deletion of Mecp2 only from inhibitory GABAergic neurons recapitulates many RTT phenotypes including the stereotypes and altered social interaction, but does not replicate anxiety-like behaviors and tremor. The role that excitatory glutamatergic neurons play in the pathogenesis of RTT has not been explored in detail. We conditionally deleted Mecp2 from glutamatergic neurons in the mouse brain using a vGlut2-Cre line, and characterized the mice by a comprehensive battery of behavioral tests as well as neurophysiological methods. The glutamatergic conditional knockout mice (CKO) became obese, and developed impaired acoustic startle and motor deficits. Interestingly, unlike the GABAergic CKO, the glutamatergic CKO showed anxiety-like behaviors as early as 5 weeks of age, and developed severe tremor. Furthermore, they died early with half of them dead by 10 weeks. These phenotypes are identical to the disease progression pattern of the Mecp2 null mutation. These data demonstrate that dysfunction of MeCP2 in excitatory glutamatergic neurons contributes to numerous neuropsychiatric phenotypes. Especially, it drives the onset of anxiety-like behaviors, tremor, and obesity in RTT, indicating an excitatory neuron-dependent mechanism underlying these phenotypes of Rett syndrome.

O-12 CROSS-SPECIES GENOMIC SCREEN FOR THERAPEUTIC APP REDUCTION IN ALZHEIMER’S DISEASE

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Amyloid aggregates are a common feature of both inherited and sporadic Alzheimer’s disease, suggesting that amyloid formation, or APP processing more broadly, may be a common mechanism of pathogenesis. Given the central role of APP in this process, and the fact that genetic alterations increasing the production of APP (i.e., gene duplication, Down syndrome) correlate with development of AD, therapeutic strategies targeting the precursor protein may be more effective than targeting its derivatives. However, we must better understand the cellular machinery controlling steady-state APP levels before we can identify promising entry points for pharmaceutical intervention. To meet this need, our consortium is conducting parallel siRNA screens in human cell lines and Drosophila expression models against the major classes of ‘druggable’ proteins to identify targets whose own reduction diminishes the level of APP. Results from these screens were cross validated between the two systems, and then used to construct in silico models of cellular pathways regulating APP. Screening of our first target category, the kinase, identified three candidates for further study. Mouse shRNAs for each candidate were engineered into AAV to test the impact of target reduction on APP levels in the mouse brain. Virus was introduced by intraventricular injection into wild-type and APP transgenic neonates, and achieved approximately 50% reduction of each candidate over 4 weeks of expression in vivo. Preliminary results corroborate the role of these three kinases in the regulation of steady-state APP levels. Our long-term goal is to identify new molecular pathways, and more precisely, new ‘druggable’ proteins within those pathways, that might serve as tractable pharmacological targets for treatment or prevention of Alzheimer’s disease.
SEIZURE ACTIVITY AFFECTS THE MACHINERY NECESSARY FOR THE FORMATION OF LONG-TERM MEMORY

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Epilepsy is a disorder characterized by recurrent seizures and deficits in learning and memory. While current antiepileptic drugs target seizures, they do not treat and may exacerbate the comorbidities. Studies in epilepsy models suggest that seizures induce learning and memory deficits. Seizures may also result in hyperactive signaling of the mechanistic target of rapamycin (mTOR), a pathway that promotes protein synthesis and learning and memory. Yet whether hyperactive mTOR signaling underlies the comorbidities remains unclear. In these studies, we evaluated how a seizure affects learning, memory, mTOR signaling, and translational efficacy.

To test our hypotheses, rats were subjected to a seizure and tested in Fear Conditioning (FC). We performed western blotting (WB) to assess mTOR activation and ribosomal efficacy. Finally, we tested whether the mTOR inhibitor rapamycin (Rap) would restore aberrant signaling and behavioral deficits. Our data indicate that neither control nor seizure animals displayed deficits in learning or short-term memory. However, only seized rats exhibited long-term memory deficits. WB revealed that seizures resulted in hyperactive mTOR signaling and impaired ribosomal activity. Rap blocked aberrant mTOR signaling but did not restore long-term memory or ribosomal function.

These data indicate that while seizures result in hyperactive mTOR signaling, the cascade may not underlie memory deficits. Studies are underway to test additional therapies that may ameliorate the seizure-related deficits. Results from this work may reveal novel strategies for patients with epilepsy-associated comorbidities.

MTOR INHIBITION SUPPRESSES ESTABLISHED EPILEPSY IN A MOUSE MODEL OF CORTICAL DYSPLASIA

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Hyperactivation of the mechanistic target of rapamycin (mTOR) pathway has been demonstrated in human cortical dysplasia (CD) and animal models of epilepsy. While inhibition of mTOR early in epileptogenesis suppressed epileptiform activity in the neuron subset-specific Pten knockout (NS-Pten KO) mouse model of CD, the effects after epilepsy is fully established are unknown. Here, we investigated whether mTOR inhibition suppresses epileptiform activity and associated pathology in NS-Pten KO mice with severe and well-established epilepsy. NS-Pten KO mice were treated with the mTOR inhibitor rapamycin (10 mg/kg, 5 days/week) starting at postnatal week 9. We monitored epileptiform activity using video-electroencephalography (EEG) and evaluated mTOR pathway and glial markers using western blotting and immunohistochemistry. Epilepsy worsened with age in NS-Pten KO mice, with parallel increases in hippocampal mTOR dysregulation and astrocyte and microglia. Rapamycin treatment significantly suppressed epileptiform activity, improved baseline EEG activity, and increased survival in severely epileptic NS-Pten KO mice. At the molecular level, rapamycin treatment was associated with decreased mTOR signaling and gliosis. These findings reveal a wide temporal window for successful therapeutic intervention with rapamycin in NS-Pten KO mice and support mTOR inhibition as a candidate therapy for established epilepsy associated with CD and genetic mTOR pathway dysregulation.

A KCNQ2/3 MUTATION CAUSING SEVERE EPILEPSY DISRUPTS CHANNEL TARGETING TO THE AXON INITIAL SEGMENT

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KCNQ2/3 are voltage-gated potassium channels underlying the M-current (Ih) regulating neuronal excitability. Localization and concentration of KCNQ2/3 channels at the axon initial segment (AIS) tightly regulates normal firing patterns. Genetic mutations in KCNQ2/3 may lead to mild disorders such as Benign Familial Neonatal Seizures (BFNS) or severe disorders such as epileptic encephalopathy, which may be caused by mechanisms that fail, including gating, trafficking, or rapid degradation. In order to test for this, I performed immunofluorescence labeling in rodent tissue, cultured rat hippocampal neurons, and perform biotinylation assay in CHO cells transfected with mutant KCNQ2 in order to determine whether or not trafficking of the KCNQ2/3 channels are altered or abnormally degraded. I immunolabeled tissue sections from a transgenic hKCNQ2-G279S mouse model. Immunofluorescence microscopy performed on tissue sections from transgenic mice overexpressing the dominant negative mutant G279S revealed an aberrant labeling pattern: KCNQ2 was completely absent at the AIS and was retained at intracellular puncta in the soma and dendrites. KCNQ3 was partially redistributed to these puncta. However, surface biotinylation assays in CHO cells show no changes in mutant KCNQ2’s ability to be detected at the surface. Some mutations may act by preventing surface trafficking and AIS concentration. Since such effects that may not be easily revealed through heterologous expression, further development of in vivo models is warranted.

TRANSGENE EXPRESSION IN THE NEUROPSIN TTA DRIVER LINE IS NOT RESTRICTED TO THE ENTORHINAL CORTEX

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The entorhinal cortex (EC) plays a central role in episodic learning and memory and is among the earliest sites of neuronal loss and neurofibrillary tangle formation in Alzheimer’s disease. The EC has therefore been an attractive target for genetic manipulation to selectively modify gene expression in various models of neurological disease. Many models utilize the neuropin (Nop) promoter to limit spatial distribution of the tetracycline transactivator (TfA). When crossed with a second tet-responsive transgenic line, the resulting bimice will express the transgene of interest in neurons where TTA is active. The Nop-TTA mouse line was reported to restrict tet-responsive transgenes to the medial EC and the pre- and parasubiculum. Nop-TTA mice have been used in several experimental studies examining functional properties of the entorhinal-hippocampal circuitry. The utility of this transgenic driver line is contingent on the specificity of the spatially restricted gene expression, yet detailed neuroanatomical mapping of its expression has not yet been done. We therefore crossed the Nop-TTA driver line with a reporter strain expressing β-galactosidase, and established an online histological atlas of Nop-TTA regulated gene expression. This atlas resource was used to perform a detailed brainwide analysis of TTA expression in bimice. Our findings highlight strong expression in regions beyond the EC and suggest caution in interpreting experiments that depend on precise localization of gene products controlled by the Nop-TTA driver.
Despite substantial evidence supporting the role of ERβ in the regulation of stress and anxiety, the role of ERβ neurons in the stress circuitry has not been delineated. The focus of my studies will be to determine which populations of ERβ neurons are involved in controlling stress and anxiety, and how these neurons are integrated into the stress circuitry. To address these questions, I will use several novel transgenic mouse models that facilitate the exploration of ERβ neurons in the brain. An ERβ-EGFP mouse that expresses a green fluorescent reporter gene in ERβ-expressing cells enables identification of these neurons in the brain. A second novel transgenic mouse has been developed that expresses the enzyme Cre-recombinase in ERβ neurons. This Cre-driver mouse targets ERβ neurons for selective gene deletion or expression, which allows functional circuitry mapping of ERβ neurons using state of the art approaches. The results of my proposed studies of the molecular and neurobiological mechanisms of ERβ neurons and their integration into the regulatory pathways controlling neuroendocrine and behavioral responses to stress hold considerable promise for elucidating new insights into brain pathways regulating stress responses, sex differences in these pathways, and the genesis of novel treatments for stress and anxiety disorders.

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Spike-wave seizures, which involve abnormal synchronization of cortex and underlying thalamic nuclei and are associated with behavioral arrest, represent a major category of human epilepsy, namely absence epilepsy. Mutations in ion channels have been implicated in absence epilepsy and, in particular, Cacna1a, the P/Q voltage-gated calcium channel, has been widely studied. We sought to determine the minimal cellular lesion required to produce this network disturbance and, therefore, used the Neurotensin Receptor (Ntsr) 1 Cre-driver to selectively ablate P/Q channel expression in layer VI pyramidal neurons that supply the descending cortical synaptic input to thalamic relay neurons and interneurons in the nRT. This selective ablation of Cacna1a resulted in mice which displayed the spontaneous appearance of typical spike-wave absence seizures. The spike-wave seizures could be inhibited by using ethosuximide to inhibit T-type calcium currents. To verify the selectivity of the Ntsr-Cre driver, we utilized a tdTomato reporter mouse. Using immunohistochemistry, we have verified that the P/Q subunit protein was reduced in thalamic terminal zones. Additionally, evoked P/Q mediated glutamate release was absent at corticothalamic terminals, although baseline exocytosis was preserved by N-type subunit rescue, demonstrating that neurotransmitter release at this synapse relies on both P/Q and N-type. We have found that an early synaptic imbalance due to a release defect limited to a single cell type within the thalamocortical circuit is sufficient to produce a stable generalized epileptic phenotype in adult brain.

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Methyl-CpG-Binding Protein 2 (MeCP2) was first described as a transcriptional repressor that binds to methylated DNA and interacts with transcription-regulating protein complexes, and it garnered much research interest after the discovery that mutations in or duplications of MECP2 cause neurodevelopmental disorders known as Rett syndrome and MeCP2 duplication syndrome, respectively. However, despite much research into its cellular functions, it is still unclear what MeCP2 does in neurons and how either its loss or its excess produces such profound neurologic dysfunction and inverse gene expression changes in the loss and gain models. I propose that MeCP2 regulates transcription in response to neuronal activity and that this regulation is dependent on its modifications and subsequent interactions with chromatin remodeling proteins and DNA. To test this hypothesis and determine whether MeCP2 regulates activity-dependent gene expression in the mature brain, I adapted a deep brain stimulation (DBS) protocol to elicit robust, in vivo activation of dentate gyrus neurons in awake, freely moving mice. I have optimized this protocol and showed it can induce expression of activity-dependent genes. Using wild-type, MeCP2 null, and MeCP2 duplication mice, I will determine the gene expression changes in these mutants and controls before and after DBS. I will also evaluate the modifications, interactions, and DNA-binding pattern of MeCP2 before and after DBS in wild-type mice to pinpoint the mechanisms by which MeCP2 contributes to gene expression changes.

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Methyl-CpG-Binding Protein 2 (MeCP2) was first described as a transcriptional repressor that binds to methylated DNA and interacts with transcription-regulating protein complexes, and it garnered much research interest after the discovery that mutations in or duplications of MECP2 cause neurodevelopmental disorders known as Rett syndrome and MeCP2 duplication syndrome, respectively. However, despite much research into its cellular functions, it is still unclear what MeCP2 does in neurons and how either its loss or its excess produces such profound neurologic dysfunction and inverse gene expression changes in the loss and gain models. I propose that MeCP2 regulates transcription in response to neuronal activity and that this regulation is dependent on its modifications and subsequent interactions with chromatin remodeling proteins and DNA. To test this hypothesis and determine whether MeCP2 regulates activity-dependent gene expression in the mature brain, I adapted a deep brain stimulation (DBS) protocol to elicit robust, in vivo activation of dentate gyrus neurons in awake, freely moving mice. I have optimized this protocol and showed it can induce expression of activity-dependent genes. Using wild-type, MeCP2 null, and MeCP2 duplication mice, I will determine the gene expression changes in these mutants and controls before and after DBS. I will also evaluate the modifications, interactions, and DNA-binding pattern of MeCP2 before and after DBS in wild-type mice to pinpoint the mechanisms by which MeCP2 contributes to gene expression changes.
**P-09**

FORNICEAL DEEP BRAIN STIMULATION RESCUES THE IMPAIRMENT OF CONTEXTUAL FEAR MEMORY IN A MOUSE MODEL OF RETT SYNDROME

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Deep brain stimulation (DBS) is an established therapy for several neurological disorders. By stimulating disease-specific target regions of the brain, DBS in both human patients and animal models has been shown to improve symptoms in Parkinson’s disease, obsessive-compulsive disorder, depression, schizophrenia as well as improve cognitive deficits in Alzheimer’s disease (AD) and epilepsy. However, the mechanistic dissection of DBS is rare, especially in awake, freely moving DBS-induced memory enhancement was abolished. These results suggest that fornixal DBS might improve hippocampus-dependent memory retrieval via modulation of the cholinergic system. Fornixal DBS may serve as a therapeutic intervention to rescue the cognitive deficits of developmental neurological diseases.

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**P-10**

TRANSLATIONAL CONTROL OF MGLUR-DEPENDENT LONG-TERM DEPRESSION AND OBJECT-PLACE LEARNING BY EIF2α

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At hippocampal synapses, activation of group-I metabotropic glutamate receptors (mGluRs) induces long-term depression (LTD), which requires new protein synthesis. However, the underlying mechanism remains elusive. Here we describe the translational program that underlies mGluR-LTD and identify the translation factor eIF2α as its master effector. Genetically reducing eIF2α phosphorylation, or specifically blocking the translation controlled by eIF2α phosphorylation, prevents mGluR-LTD and the reduction of surface AMPA receptors (sAMPARs). Conversely, direct phosphorylation of eIF2α, bypassing mGluR activation, triggers a sustained LTD and removal of sAMPARs. Combining polysome-profiling and RNA-sequencing, we identify the mRNAs translationally up-regulated during mGluR-LTD. Translation of one of these mRNAs mediates the LTD induced by eIF2α phosphorylation. Remarkably, mice with deficient p-eIF2α-mediated translation are impaired in object-place learning, a behavioral task that induces hippocampal mGluR-LTD in vivo. Our findings identify a novel model of mGluR-LTD, which promises to be of value in the treatment of mGluR-LTD-linked cognitive disorders.

**P-11**

CELL TYPE SPECIFIC ABLATION OF RAPTOR IN REGULATING MEMORY STORAGE

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The mechanistic target of rapamycin (mTOR) is at the center of an evolutionarily conserved signaling pathway that integrates information from various synaptic inputs and controls long-term memory storage. mTOR forms two different complex. mTOR complex 1 (mTORC1), which is defined by Raptor (Regulatory-Associated Protein of mTOR), is sensitive to the immunosuppressant rapamycin and regulate translation rates. We have previously shown that the formation of long-term memory requires mTORC1 activity (Stoica et al., PNAS, 2011). Full understanding of mTORC1's function in cognitive processing requires its dissection at the cellular level. We used molecular genetic approaches to silence mTORC1 activity in various cell types (e.g., excitatory and inhibitory neurons, as well as glia cells). Here we show that in the hippocampus of raptor fb-KO mice – where raptor was conditionally deleted in glutamatergic neurons in the post-natal forebrain - mTORC1 activity is significantly reduced but phosphorylation of Akt at Ser-473, a substrate of mTORC2, was increased. Interestingly, long-term fear and spatial memory storage is blocked in raptor fb-KO mice but social memory is spared in these mice. Because mTORC1 regulates translation rates, we are performing genome-wide analyses of in vivo translation using ribosome profiling to determine the nature of the specific proteins that are synthesized during learning and memory. Future experiments will involve electrophysiological recording and study two major forms of synaptic plasticity, long-term potentiation (LTP) as well as long-term depression (LTD).

**P-12**

MUTUAL ANTAGONISM BETWEEN SOX10 AND NFIA REGULATES DIVERSIFICATION OF GLIAL LINEAGES AND GLIOMA SUB-TYPES

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Lineage progression and diversification is regulated by the coordinated action of unique sets of transcription factors. Oligodendrocytes (OL) and astrocytes (AS) comprise the glial sub-lineages in the central nervous system (CNS) and how their associated regulatory factors orchestrate lineage diversification during development and disease remains an open question. Sox10 and NFIA are key transcriptional regulators of gliogenesis associated with OL and AS. We found that NFIA inhibits Sox10 induction of OL differentiation through direct association and antagonism of its function. Conversely, we found that Sox10 antagonizes NFIA function and suppresses AS differentiation. Using this developmental paradigm as a model for glioma, we found that this relationship similarly regulates the generation of glioma sub-types. These studies describe the antagonistic relationship between Sox10/NFIA that regulates the balance of OL and AS fate during development and demonstrate for the first time that the transcriptional processes governing glial sub-lineage diversification oversee the generation of glioma sub-types.
**P-13**

**DAAM2-PIP5K IS A NOVEL REGULATORY PATHWAY FOR WNT SIGNALING AND THERAPEUTIC TARGET FOR REMYELINATION IN THE CNS**

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Wnt signaling plays an essential role in developmental and regenerative myelination of the CNS, however contributions of proximal regulators of the Wnt receptor complex to these processes remain undefined. To identify components of the Wnt pathway that regulate these processes, we applied a multifaceted discovery platform and found that Daam2-PIP5K comprise a novel pathway regulating Wnt signaling and myelination. Using dorsal patterning of the chick spinal cord we found that Daam2 promotes Wnt signaling and receptor complex formation through PIP5K-PIP2. Analysis of Daam2 function in oligodendrocytes (OLs) revealed that it suppresses OL differentiation during development, after white matter injury (WMI), and is expressed in human white matter lesions. These findings suggest a pharmacological strategy to inhibit Daam2-PIP5K function, application of which stimulates remyelination after WMI. Put together, our studies integrate information from multiple systems to identify a novel regulatory pathway for Wnt signaling and new therapeutic target for WMI.

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**P-14**

**ROLE OF RAC-GEFS TIAM1 AND TIAM2 IN SYNAPSE DEVELOPMENT AND FUNCTION**

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Neurons communicate with one another through synapses. Synapses are plastic and can strengthen or weaken in response to neuronal activity, which is important for learning and memory. It is not surprising that synapse abnormalities are found in many brain disorders including Intellectual disabilities, autism, bipolar disorder, and depression. Elucidating the mechanism that regulate synapse development and plasticity will enhance our understanding of brain function and disease, and may also help in the development of new therapeutic targets to treat neurological diseases. How is synapse development and plasticity regulated and what’s the connection between synapse defects and neurological diseases?

The Rho family small GTPase Rac1 is a key regulator of nervous system development. Previously, our lab identified the Rac-GEF Tiam1, a Rac activator, as a critical regulator of synapse development in cultured neuron. Tiam2, a highly related homolog of Tiam1, has also been shown to be required for cultured neuron development. However, nothing is known about their role in vivo. Therefore, we generated conditional knockdown (Kd) of the adhesion-GPCR brain-specific neuronal surface marker.

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**P-15**

**THE ADHESION-GPCR BAI1 INSTIGATES RHOA-MEDIATED RESTRICTION OF HIPPOCAMPAL DENDRITIC ARBORIZATION**

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The geometry of dendritic arbors determines the availability of neurons to different circuits and the computational properties of neurons. Study of arbor formation has lagged behind study of synaptogenesis, yet arbor defects are apparent in numerous neurological pathologies. We observed that knockdown (Kd) of the adhesion-GPCR brain-specific angiogenesis inhibitor 1 (BAI1) in hippocampal neurons leads to excessive arbor growth late in development when control neurons cease net arbor growth. BAI1 Kd neurons exhibit elevated rates of dendrite branch formation and elimination, and quantitative analysis reveals a defect in arbor polarity. Using live cell imaging, we saw an increase in the activation of RhoA, a small GTPase involved in restricting arborization, at the time of growth arrest in control neurons. This increase was muted and delayed in BAI1 Kd neurons. Further, we observed a strong correlation between RhoA activation state at the tips of dendritic branches and branch behavior (growth, retraction, stability). BAI1 Kd led to a global decrease in RhoA activation, an uncoupling of RhoA activation from dendrite behavior, and an increase in branch growth late in development. Using GST fusions, we screened a battery of neuronal RhoA GEFs (activators) and identified breakpoint cluster region Bcr and active Bcr-related (Abr) as potential mediators of BAI1’s effect on RhoA. Moreover, we obtained pharmacological evidence that BAI1 Kd effects are mediated by RhoA dysregulation and that BAI1 signals to kinases via RhoA. Because BAI1 Kd alters the cell density dependence of arbor growth, we hypothesize that BAI1 recognizes a neuronal surface marker.

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**P-16**

**THE SMALL GTPASES RHOA AND RAC1 MEDICATE CEREBELLAR DEVELOPMENT BY CONTROLLING CELL MORPHOGENESIS, MIGRATION AND FOLIATION**

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The small GTPases RhoA and Rac1 are key cytoskeletal regulators that function in a mutually antagonistic manner to control the migration and morphogenesis of a broad range of cell types. However, their role in shaping the cerebellum, a unique brain structure composed of an elaborate set of folia separated by fissures of different lengths, remains largely unexplored. Here we show that dysregulation of both RhoA and Rac1 signaling results in abnormal cerebellar ontogenesis. Ablation of RhoA from neuroprogenitor cells drastically alters the timing and placement of fissure formation, the migration and positioning of granule and Purkinje cells, the alignment of Bergmann glia, and the integrity of the basement membrane, primarily in the anterior lobules. Furthermore, in the absence of RhoA, granule cell precursors located at the base of fissures fail to undergo cell shape changes required for fissure initiation. Many of these abnormalities can be recapitulated by deleting RhoA specifically from granule cell precursors but not postnatal glia, indicating that RhoA functions in granule cell precursors to control cerebellar morphogenesis. Notably, mice with elevated Rac1 activity due to loss of the Rac1 inhibitors Bcr and Abr show similar anterior cerebellar deficits, including ectopic neurons and defects in fissure formation, Bergmann glia organization and basement membrane integrity. Together, our results suggest that RhoA and Rac1 play indispensable roles in patterning cerebellar morphology.
P-17  THE CELL-ADHESION GPCR BAI1 REGULATES EXCITATORY SYNAPSE DEVELOPMENT

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The formation, regulation and maintenance of excitatory synapses are crucial for normal cognitive function. Recently, we have identified the adhesion G protein-coupled receptor (GPCR) brain-specific angiogenesis inhibitor 1 (BAI1) as a key regulator of synapse development. BAI1 is highly localized to spines, and knockdown of BAI1 results in decreased spine and synapse density in both cultured hippocampal neurons and cortical neurons from intact mouse brains. Synaptic loss caused by BAI1 knockdown can be rescued by full-length BAI1, but not by a BAI1 truncation mutant, which fails to interact with the Par3/Tiam1 polarity complex. Tiam1 is a Rac1-guanine nucleotide exchange factor that promotes spine and synapse development by inducing Rac1-dependent actin remodeling. Tiam1 is restricted to spines by the polarity protein Par3. We show that BAI1 regulates spine and synapse development by recruiting the Par3/Tiam1 complex to spines, resulting in localized Rac1 activation. Although these findings elucidate how BAI1 signals inward to promote pre-synaptic differentiation, it is unclear whether BAI1 also signals across the synapse to induce pre-synaptic differentiation. In a COS7 cell-neuron co-culture system, we show that BAI1 induces pre-synaptic termini formation on the axons of cultured hippocampal neurons that contact BAI1-expressing COS7 cells. These results indicate that BAI1 can induce pre-synaptic as well as post-synaptic development. Our future directions are to identify synaptic ligands for BAI1. Our findings should help to elucidate mechanisms that regulate excitatory synapse development, and provide potential therapeutic targets for the treatment of neurological disease.

P-18  DEGENERATION AND REGENERATION OF AXONAL SUB DOMAINS AFTER OPTIC NERVE CRUSH

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Myelinated axons are divided into several distinct domains, which includes axon initial segment (AIS), nodes of Ranvier, paranodes, and juxtaparanodes. The AIS serves as both a physical barrier between the axonal and somato-dendritic compartments of the neuron and as the site of action potential (AP) initiation. Nodes of Ranvier are responsible for the rapid and efficient propagation of APs along the axon. Disruption of the AIS or nodes of Ranvier by genetic and/or pharmacological manipulation has a dramatic impact on the central nervous system. With this in mind, we have designed a series of experiments, which will allow us to assess the efficacy of neuroprotective drugs upon axons of the central nervous system after insult. Using the optic nerve crush paradigm, we have established a timeline for degenerative events of the nodes of Ranvier of the optic nerve and AIS of retinal ganglion cells. We will use this data as a baseline to compare the efficacy of two neuroprotective paradigms. For this study, we will assess the efficacy of the PTEN-Socs3 regeneration model as well as intravitreal administration of MDL-28170 - a calpain inhibitor that has been shown to protect from AIS degeneration after ischemic injury.

P-19  LOSS OF PALS1 IN SCHWANN CELLS LEADS TO RADIAL SORTING DEFECTS

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Schwann cells in the PNS and oligodendrocytes in the CNS surround axons with myelin to enable rapid and reliable action potential propagation by sequestering Na+ channels at nodes of Ranvier. The developmental mechanisms regulating myelin formation and the radial sorting of axons in the PNS are only partially understood. Recently, highly conserved polarity proteins have been implicated in myelination and radial sorting. One polarity protein, Protein Associated with Lin7 (Pals1), which localizes to paranodes, Schmidt-Lanterman incisures, and the adaxonal domain in Schwann cells, has been proposed to regulate myelin thickness, length, and ultrastructure. To determine whether the role of Pals1 in myelination, we generated conditional knockout (cKO) mice lacking Pals1 in myelinating glia using the Cre-lox recombinase system under the control of the 2.3’-cyclic nucleotide phosphodiesterase (CNP) promoter. As adults, CNP-Cre;Pals1 cKO mice demonstrate hind limb clamping. Axons in the CNS and PNS of adult cKO mice are myelinated. Mature Schwann cells exhibit normal length and thickness and subcellular domains are present. However, some axon bundles in the sciatic nerve are aberrantly myelinated. Developmental analysis reveals a significant delay in PNS myelin formation up to postnatal day 21. Furthermore, transmission electron microscopy reveals that radial sorting of axons by non-myelinating Schwann cells is perturbed up to postnatal day 21. In contrast, CNS myelination is normal throughout development, suggesting divergent polarity mechanisms between Schwann cells and oligodendrocytes.

P-20  GENETIC CHARACTERIZATION OF CENTRAL NORADRENERGIC FUNCTION IN RESPIRATORY CONTROL

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The central noradrenergic (NA) system is a critical constituent in a complex array of neuronal networks that maintain respiratory homeostasis, constantly and consistently responding to physiological and environmental changes to sustain breathing. While past studies point to a high molecular and functional complexity within the system, this diversity has been largely unexplored and the underlying functional organization remains unknown. Because early gene expression is critical to the development of neural circuits and cell identity, we hypothesize that subpopulations of NA neurons originating from different rhombomeres, differential gene expression domains in the developing brainstem, regulate distinct and specific aspects of respiration. Our preliminary data show that NA neurons are required for the respiratory response to hypercapnic (high CO2) and hypoxic (low O2) conditions, as pharmaco-genetic perturbation of NA neurons results in reduced ventilatory responses. Additional pilot studies also suggest that perturbation of populations of neurons derived from different rhombomeres results in a variety of respiratory phenotypes. These data present a working model to query the function of a genetically defined neuron population, and linking these developmentally distinct subtypes of neurons to their role in respiratory homeostasis will give us greater insight into the functional organization of neuromodulatory networks and improve potential therapeutic strategies for life-threatening respiratory disorders.
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Purkinje cell activity is essential for controlling motor behavior. During motor behavior Purkinje cells fire two types of action potentials: intrinsically-generated simple spikes and climbing fiber input-induced complex spikes. Although the functions of these spikes are becoming clear, how they are established is still poorly understood. Here, we used in vivo electrophysiology approaches conducted in anesthetized and awake mice to record Purkinje cell activity starting from the second postnatal week of development to adulthood. We found that the rate of complex spike firing increases sharply at 3 weeks of age whereas the rate of simple spike firing gradually increases until 4 weeks of age. We also found that compared to adult, the pattern of simple spike firing during development is more irregular as the cells tend to fire in bursts that are interrupted by long pauses. The regularity in simple spike firing only reached maturity at 4 weeks of age. In contrast, the adult complex spike pattern was already evident and consistent by the second week of life. Analyses of Purkinje cells in alert behaving mice suggested that the adult patterns are attained more than a week after the completion of key morphogenetic processes such as migration, lamination, and foliation. Purkinje cell activity is therefore dynamically sculpted throughout postnatal development, traversing several critical events that are required for circuit formation. Overall we show that simple spike and complex spike firing develop with unique developmental trajectories.

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Creating accurate 3D representations of the world from 2D retinal images is a fundamental task for the visual system. However, the reliability of different 3D visual signals depends inherently on viewing geometry, such as how much an object is slanted in depth. Human perceptual studies have correspondingly shown that texture and binocular disparity cues for object orientation are combined according to their slant-dependent reliabilities. Where and how this cue combination occurs in the brain is currently unknown. Here, we search for neural correlates of this property in the macaque caudal intraparietal area (CIP) by measuring slant tuning curves using mixed-cue (texture + disparity) and cue-isolated (texture or disparity) planar stimuli. We find that texture cues contribute more to the mixed-cue responses of CIP neurons that prefer larger slants, consistent with theoretical and psychophysical results showing that the reliability of texture relative to disparity cues increases with slant angle. By analyzing responses to binocularly viewed texture stimuli with conflicting texture and disparity information, some cells that are sensitive to both cues when presented in isolation are found to disregard one of the cues during cue conflict. Additionally, the similarity between texture and mixed-cue responses is found to be greater when this cue conflict is eliminated by presenting the texture stimuli monocularly. The present findings demonstrate reliability-dependent contributions of visual orientation cues at the level of the CIP, thus revealing a neural correlate of this property of human visual perception.
Channels of escape: how HCN channels influence locusts' predator detection

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Successfully escaping predation requires not just reliable detection of approaching predators, but successfully discriminating non-threatening stimuli as well. In locusts, visual detection of approaching predators can be accomplished by a single neuron within the lobula of each optic lobe. This lobula giant movement detector neuron, or LGMD, integrates inputs from every photoreceptor of the ipsilateral eye. As the excitatory arbor of the LGMD is retinotopically organized, the spatial stimulation pattern determines the spatial pattern of excitatory inputs. We investigated the role of the hyperpolarization-activated inward rectifying current (Ih) within the LGMD of Schistocerca americana to better understand the neural computations implemented within this looming sensitive neuron and their role in escape behavior. LGMD responses to looming objects decreased dramatically after Ih blockade, while visual stimuli with decreased spatial coherence elicited less frequent escape behavior and LGMD responses unaffected by Ih blockade. Further, injection of a HCN specific blocker into the lobula inhibited escape behavior to looming stimuli. This suggests that not only are the H channels playing a key role in the processing of looming stimuli within the LGMD, but that their activity is also required for the animals' normal escape behavior. To further test the mechanism of Ih excitation of coherent objects, detailed neural modeling was combined with behavioral and electrophysiological experiments.

Joint representation of translational and rotational components of self-motion in the parietal cortex

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Navigating through the world involves processing complex visual inputs to extract information about self-motion relative to one's surroundings. When translations (T) and rotations (R) are present together, the velocity patterns projected onto the retina (optic flow) are a combination of the two. Since navigational tasks can be extremely varied, such as deciphering heading or tracking moving prey or estimating one's motion trajectory, it is imperative that the visual system represent both the translational and rotational components. Despite the importance of such joint representations, most previous studies have only focused on the representation of translations and emphasized the role of extra-retinal cues (efference copies of self-generated rotations) rather than visual cues for decomposing the optic flow.

We recorded single units in the macaque ventral intraparietal area (VIP) to understand the role of visual cues in decomposing optic flow and representing both the translational and rotational components. Through this study, we establish that the visual system can rely on purely visual cues to extract both components of self-motion. Interestingly, we find that individual VIP neurons can jointly represent both T and R. We hypothesize that such a separable and joint representation of translation and rotation may provide a flexible mechanism for estimating self-motion trajectories.

Neural motion detection circuits underlying looming-evoked escape behaviors in Drosophila

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In this study, we used a behavioral approach to investigate whether the ON and OFF motion detection pathways stemming from L1/L2 provide inputs to Foma-1. Our experiments were designed to start with Foma-1 and proceed upstream along the putative pathways towards L1 and L2. To trigger jump escape behaviors, we used light (ON) and dark (OFF) looming stimuli presented on a dark and light background, respectively. We used the GAL4/UAS system to silence specific cell classes belonging to the two pathways. In this system, the transcription factor GAL4 is expressed specifically in neurons such as L1, L2, T4, T5, or Foma-1 neurons. When the flies are shifted to 28-30 °C, UAS-Shp will disrupt synaptic vesicle recycling, effectively silencing the neurons at the restrictive temperature. Our results show that silencing T4 and T5 results in a decrease in escape probabilities very close to that observed when silencing Foma-1. This suggests that T4 and T5 provide the main input to Foma-1. When we presented ON stimuli at the restrictive temperature, the escape jump probabilities of T4 blocked flies and L1 blocked flies were strongly suppressed. The decrement tendencies were similar to those observed in T4&T5 blocked flies. However, in the OFF stimuli experiments, T4 blocked flies and L1 blocked flies responded normally. In contrast, the jump probabilities of T5 blocked flies and L2 blocked flies were decreased in response to OFF stimuli. These results show that T4 and L1 mediate responses to ON looming stimuli, while T5 and L2 mediate responses to OFF looming stimuli. We are further investigating this hypothesis using imaging electrophysiology.
CHARACTERIZATION OF MEDULLARY NEURON PROPERTIES IN RESPONSE TO ON AND OFF STIMULI IN A LOCUST LOOMING DETECTION CIRCUIT

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In the locust visual system, the lobula giant movement detector (LGMD) and its postsynaptic target, the descending contralateral movement detector (DCMD) in the protocerebrum, are a pair of looming sensitive interneurons responding strongly to objects approaching on a collision course (looming stimuli). To date, the neurons presynaptic to the LGMD in the medulla remain to be identified. We stimulated medullary neurons expressing channelrhodopsin with laser light pulses, recording their spiking activity with extracellular electrodes and the postsynaptic response of the LGMD with intracellular recordings. Spike sorting and correlation analysis helped to identify spontaneous medullary spike-triggered IPSPs in the LGMD. Moving bars and moving edges were used to stimulate the same medullary neurons. Both medullary neurons and the LGMD exhibited a burst of spikes when a bar just started its motion at the border of the screen, whereas their activity was weaker during the motion of the bar on the screen. In contrast to their response to bar movement, medullary neurons exhibited robust spiking during the movement of an OFF edge. The movement of an ON edge evoked relatively fewer spikes in the same neurons. Thus, our extracellular recordings indicate that these neurons prefer to respond to OFF edge movement. We infer that the neurons we identified belong to the locust OFF visual pathway, and that they likely synapse onto inhibitory branch C of the LGMD. We will next investigate the coding properties of OFF visual pathway medullary neurons in the context of looming detection.

MOUSE RETINAL GANGLION CELLS ALTER THEIR SPATIOTEMPORAL PROPERTIES TO ENCODE VISION IN DIM LIGHT SETTINGS

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The retina must encode visual activity in dim starlight as well as bright sunlight. In order to accomplish this feat the retinal circuitry is organized into two parallel pathways originating from rods or cones for dim or bright light conditions, respectively. Each of these pathways has unique properties that are passed onto the retinal ganglion cells (RGC), the output cells of the retina. By using a multielectrode array (MEA) to record from many RGCs and probing with stimuli at different light intensities we find that RGCs slow their temporal tuning and widen their spatial tuning in dim light. These changes allow the RGC to encode light over longer time and wider space which is critical in low light settings because fewer photons are present. Identifying how RGC modify their properties will shed light on normal visual encoding and also improve our understanding of diseases that damage RGCs and cause deficit in dim light settings, like glaucoma.

IN VIVO TWO-PHOTON CALCIUM IMAGING OF SUBCELLULAR INPUT RETINOTOPY IN AN IDENTIFIED VISUAL INTERNEURON

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The Lobula Giant Movement Detector (LGMD) is an identified visual interneuron in the locust that responds preferentially to objects approaching on a collision course. The LGMD receives excitatory input from the entire visual hemifield sampled by one eye that preserves retinotopy down to the level of a single facet on the compound eye. Because single photon imaging with CCD sensors has a relatively low penetration and stronger scattering, previous work could not investigate this retinotopic mapping at the level of individual thin dendritic branches. Our current work employs a custom-built two-photon microscope with sub-micron resolution in conjunction with an OLED (Organic Light-Emitting Diode) microdisplay that provides visual stimuli to the locust compound eye adequate to explore this retinotopy at the finest level. We find that the adjacent facets on the compound eye have overlapped mappings on the LGMD excitatory dendritic branch. When the size of the visual stimuli decreases while their center is kept unchanged, the overlap of the mapping and the number of activated branches also decrease.

ANATOMICAL AND FUNCTIONAL ORGANIZATION OF ONTOGENETIC COLUMNS

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The mammalian neocortex gives rise to complex cognitive processes such as perception and decision-making. Despite tremendous progress in understanding the physiology and cell biology of individual neurons in the cortex, the process by which networks of neurons become wired together during development and organize into functional circuits remains poorly understood. Recent studies suggest that cell lineage influences the connectivity and functional properties of excitatory pyramidal neurons in the neocortex, but it remains unclear to what extent cell lineage influences circuit assembly. We have developed a tamoxifen-inducible Cre/lox system for lineage tracing in the neocortex that allows us to sparsely label neural progenitors and trace their daughter cells. When progenitors are labeled at the onset of neurogenesis, they give rise to columns of clonally related pyramidal neurons spanning all six cortical layers. Interestingly, we find that sister cells in ontogenetic columns show enhanced connectivity across cortical layers but not within the same cortical layer. By utilizing 3D random-access multiphoton (3D-RAMP) imaging, we are also able to study the functional organization of ontogenetic columns in the visual cortex. These experiments will shed light on how functional networks are established during development and could provide a circuit level foundation to study neurodevelopmental disorders.
Neural responses are modulated by brain state, which varies with arousal, attention, and behavior. In mice, running and whisking desynchronize the cortex and enhance sensory responses, but the quiescent periods between bouts of exploratory behaviors have not been well-studied. We found that these periods of “quiet wakefulness” were characterized by state fluctuations on a timescale of 1-2 seconds. Small fluctuations in pupil diameter tracked these state transitions in multiple corticaIs. During dilatation, the intracellular membrane potential was desynchronized, sensory responses were enhanced, and population activity was less correlated. In contrast, constriction was characterized by increased low-frequency oscillations and higher ensemble correlations. Specific subtypes of cortical interneurons were differentially activated during dila- tion and constriction, consistent with their participation in the observed state changes. Pupilometry has been used to index attention and mental effort in humans, but the intracellular dynamics and differences in population activity underlying this phenomenon were previously unknown.

To deal with rich visual information, it is helpful to have a short-term buffer — visual short-term memory (VSTM). To understand how VSTM works in the real world, it is necessary to understand VSTM for objects that have multiple features. Indeed, this has been an area of enduring interest in cognitive psychology. One prominent question has been whether such multi-feature objects get stored in VSTM as entire objects or as bags of features. In the literature, this question has been turned into a testable hypothesis in several, not mutually exclusive ways. In recent years, the focus of the field has shifted to studying exactly this noise corrupting memories. Whereas vigorous debate is ongoing about the nature of memory noise, it is not disputed that memories are noisy and a complete account of VSTM should take noise into account. Here, we simultaneously address previous hypotheses using a paradigm that is closely related to the classic change detection experiments but that measures memory noise. Based on human psychophysics experiments, we rule out that, for our stimuli, memory resource is shared between orientation and color. We also reject that, for our stimuli, the irrelevant feature is both encoded and taken into account during decision-making although subjects could recall the irrelevant information of multi-feature stimuli. Finally, we found that some portion of a feature’s resource is leaked to irrelevant locations; for example, some orientation resource is leaked to color-only items and “wasted” there.

Methyl-CpG-binding-protein-2 (MECP2) duplication syndrome is a progressive X-linked disorder of autism, intellectual disability, and epilepsy. Interestingly, the mouse model of MECP2 duplication syndrome exhibits enhanced motor and contextual fear learning in addition to stereotyped behaviors and social avoidance. We hypothesized that a bias towards increased synaptic stability could lead to abnormally enhanced memory consolidation, reminiscent of savant-like behaviors occasionally associated with autism. Learning-associated structural synaptic plasticity was measured in motor cortex of MECP2 duplication mice by 2-photon imaging. An increased stabilization rate of learning-associated spines was observed in mutants. Analysis of the spatial distribution of stabilized spines increased stabilization rate of learning-associated spines was observed in mutants. Analysis of the spatial distribution of stabilized spines revealed that the mutant’s enhanced consolidation was due to a specific increase in the stability of spines jointly formed in 10-micron clusters. Clustered spine stabilization predicted enhanced motor performance in mutants. The Ras-MAPK signaling pathway, previously implicated in clustered synaptic plasticity, was found to be hyperactive specifically after training in MECP2 duplication mouse motor cortex. Pharmacologic inhibition of MAPK signaling normalized motor learning in mutants. Experiments in progress will assess if inhibiting MAPK signaling also normalizes the mutant’s increase in clustered spine stabilization, enabling us to determine if indeed aberrant clustered plasticity drives enhanced learning in this form of syndromic autism.
NEUROPIL RESPONSE TO VISUAL STIMULI AND ITS CORRELATION TO CELL ACTIVITY

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Large areas in the brain are allocated for forming synaptic connections between neurons for neuronal information transmission. The area containing axons, dendrites, and glia cells, but not neuronal cell bodies, is named neuropil. In the neocortex, it has been shown that axons including axonal buttons and dendrites constitute 70-80% of the neuropil volume. This suggests that neuropil activity mainly arises from the interaction between axonal presynaptic and dendritic postsynaptic activity. Assuming random entanglement of axons and dendrites of numerous neurons with diverse functional specificities, Kerr and colleagues showed that spontaneous activity of a large neuropil patch in layer 2/3 of mouse motor and barrel cortex correlated well with the simultaneously recorded electrocorticogram (ECoG). However, stronger layer 2/3 of mouse motor and barrel cortex correlated well with the visually responsive for days and weeks following the lesion. We found that a near-total removal of the apical dendritic arbor had no effect on the orientation tuning function of L2/3 pyramidal neurons. Now, we are ablating basal dendrites to verify their contribution to the orientation selectivity in the superficial layers of mouse V1.

IN VIVO 2-PHOTON MICROSCOPY OF CORTICAL ABSENCE EPILEPSY IN STARGAZER MICE

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Our understanding of neuronal network malfunction in epilepsy and its relation to seizure evolution and progression in intact cortical circuitry is limited. We examined the stargazer mouse, where a genetic mutation in Cacng2 leads to dendritic AMPA receptor trafficking defects and rhythmic 6-7/sec cortical spike-wave discharges. Using in vivo two-photon microscopy and simultaneous intracranial EEG in awake mice, we are able to record calcium activity in individual neurons in layer 2/3 and 4 of somatosensory and visual cortex during spontaneous spike-wave seizures over many weeks using the chronic calcium indicator GCamp6. Preliminary data (n=8 animals) show that typically <15% of layer 2/3 and layer 4 neurons display significant calcium activity modulation during seizures. Various patterns of activation and deactivation were observed in different cells. Aggregate population activity was not significantly changed between interictal and interictal periods. Full-field analysis of the calcium activity revealed structures other than cell bodies, presumably apical dendrites, whose activity was modulated significantly around seizure times. Additionally, we observed high-amplitude, subthreshold membrane voltage modulations that were highly synchronized with the spike-wave rhythms during seizures in the EEG, using single-cell in vivo current clamp recordings. Identification of the specific cell types using CLARITY will facilitate the evaluation of cell-specific network mechanisms in these mice with the two-photon microscopy. Combining these techniques offers a unique opportunity to bridge the gap between cellular and network behavior in epilepsy.

CONTRIBUTION OF APICAL AND BASAL DENDRITES TO ORIENTATION SELECTIVITY IN LAYER 2/3 OF MOUSE V1

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Neurons in the superficial layers (L2/3) of primary visual cortex (V1) fire action potentials in response to visual stimuli with specific orientations. A long-standing hypothesis in the field is that this orientation selectivity arises from the retinotopic alignment of inputs from the thalamus via layer 4. As these inputs primarily target the basal dendrites of L2/3 pyramidal neurons, it is possible that orientation tuning depends solely on the input to the basal dendritic domain. We therefore developed a targeted 2-photon-guided micro-ablation method to selectively remove individual dendrites from the neuronal soma in vivo. Neurons with ablated dendrites survive and remain visually responsive for days and weeks following the lesion. We found that a near-total removal of the apical dendritic arbor had no effect on the orientation tuning function of L2/3 pyramidal neurons. Now, we are ablating basal dendrites to verify their contribution to the orientation selectivity in the superficial layers of mouse V1.

SPATIAL MEMORY ENCODING IN A MOUSE MODEL OF TAU-MEDIATED NEURODEGENERATION

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Improper aggregation of tau protein occurs in many neurodegenerative dementias. A major target of degeneration in these tauopathies is the hippocampus, a classical brain region for memory. There, spatial memory is encoded by place cells, pyramidal neurons that fire based on an animal’s location in space. Also crucial for proper memory encoding are oscillations in the local field potential (LFP) and entrainment of neuronal firing to those oscillations. In tauopathies, however, we do not know which aspects of hippocampal memory encoding are altered and when in disease they occur. Forebrain specific over-expression of human tau mutations associated with the human tauopathy FTDP-17 in rTg4510 (Tau) mice results in the formation of neurofibrillary tangles, learning and memory impairment, and neuronal death. Using this model, our lab has seen unstable place cells in old Tau mice with profound hippocampal degeneration. Here, we record in vivo from the hippocampus of Tau mice at 2 to 4 months of age as they run through a linear track and sleep. At this young age, which is prior to major degeneration, Tau mice have only minor deficits in place cell stability and size compared to WT mice. In contrast, young Tau mice do exhibit LFP and synchrony changes. Specifically, there is a decrease in the amplitude of sharp-wave ripples (high-frequency LFP oscillations associated with memory consolidation) during slow-wave sleep in Tau mice and a synchrony deficit during ripple events. Ripple amplitude is decreased even at the youngest age examined (2 mo), and gets worse with age in Tau mice while it is constant in WT mice. Results help illuminate the poorly understood network level alterations that lead to debilitating memory loss seen in tauopathy patients.
**P-41 REACTIVATION OF HIPPOCAMPAL ENSEMBLES DURING VICARIOUS FREEZING**

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Watching others experiencing an event that is similar to one’s own prior experience often induces memory retrieval. This phenomenon plays an important role in social behaviors such as empathy. To study if and how memory is retrieved during social events, we created a novel behaviorally paradigm where rats either with or without self-shock experience observe a conspecific being shocked. We found that rats with shock experience show robust freezing while observing a conspecific being shocked. This “vicarious freezing” doesn’t occur in rats without shock experience, suggesting that vicarious freezing involves the memory retrieval of self-shock experience. We hypothesize that during vicarious freezing, the hippocampal activity patterns associated with previous self-shock experience are reactivated. To test this hypothesis, we use tetrodes to simultaneously record a large number of hippocampal neurons during self-shock experience and during vicarious freezing. The preliminary data suggests that during vicarious freezing, hippocampal activity patterns occasionally flickers to those associated with previous self-shock experience. These activities occur during hippocampal ripple oscillations.

**P-42 THE EFFECTS OF BLAST WAVE EXPOSURE ON VESTIBULAR FUNCTION**

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The dramatic escalation of world conflict and the use of close range explosives has led to higher incidences of exposure to blast-related traumatic brain injury. Active service members who have experienced blast waves report high rates of vestibular dysfunction, such as vertigo, imbalance, and dizziness. Thus, accumulating evidence suggests that blast wave trauma causes damage to both the peripheral and central vestibular system. Previous work has established that blast wave exposure is capable of causing damage to the auditory hair cells of the inner ear; however, the mechanisms by which blast wave exposure induces vestibular dysfunction remains unclear. Here, we study how blast-wave exposure affects vestibular receptor morphology, the horizontal vestibulo-ocular reflex (hVOR), and vertical vestibulo-ocular reflex (vVOR) in mice. We hypothesize that blast wave exposure will produce damage to the vestibular peripheral receptors and their signals that drive the hVOR and vVOR gaze stabilization responses which underly the vestibular dysfunction observed in blast-exposed patients.

**P-43 EFFICACY OF ELECTRONIC CIGARETTES (E-CIGS) FOR SMOKING CESSATION IN VETERANS**

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E-cigarettes have become increasingly popular among US citizens in recent years. One reason for this boom in popularity may be the claim that they aid smoking cessation attempts in a similar manner to nicotine replacement therapies (NRTs). However, this claim has not been previously been assessed in a clinical setting. Veterans were randomized to either NRT (16mg patch; N=7) or E-cigs (16mg cartridge; N=4). Thrice-weekly visits occurred during the first two weeks (week 1 “baseline” with participants smoking ad libitum) and five visits occurred during the third week (week 3 “efficacy” with participants smoking as little as possible while using NRT or E-cigs). Participants were mostly African American (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores (64%) males (82%), 52.6±1.9 Mean±S.E.M years of age, and smoked cigarettes for 35.0±1.9 years, 26.5±3.0 cigarettes/day, and FTND scores.

**P-44 DURATION PERCEPTION WITH MULTIPLE STIMULI: SUBOPTIMAL CUE INTEGRATION**

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The perception of duration can be biased by the physical properties of a sensory stimulus. For example, visual stimuli with higher temporal frequency are perceived as lasting longer (Kanai et al., 2006). Objects of different temporal frequencies often appear simultaneously in the environment, providing conflicting information about duration. Does the brain keep separate duration representations for each object, or form a single representation? If a single duration representation is kept, how is it formed? One possibility is by integrating the cues of duration from each stimulus (Ahrens & Sahani, 2011); another is by reading out the total neural energy for encoding all the stimuli (Eagleman & Pariyadath 2009, 2012), alternatively, the duration representation may be based on one single stimulus depending on attention. Human participants compared the duration of Gabors patterns drifting at 1Hz and 6Hz (denoted by L for low and H for high frequency, and LH when the two were simultaneously presented). We compare different models based on Akaike Information Criterion and found that the data are consistent with hypotheses that the brain either suboptimally integrate duration cues from multiple stimuli, or perform optimal integration while the sensory noises from different cues are correlated. The data rule out the possibility of summing total neural energy or selectively attending to one stimulus. We also found that a robust discrepancy in psychometric curve steepness depending on presentation order of stimuli can be explained by the joint effect of memory decay and incorporation of prior distribution.
P-45  SOUND-TO-TOUCH SENSORY SUBSTITUTION FOR THE DEAF AND SEVERELY HEARING IMPAIRED

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There are at least 2 million functionally deaf individuals in the United States alone and an estimated 53 million worldwide. The cochlear implant is an effective solution for regaining hearing; however, such implants are expensive, require invasive surgery, and have low efficacy in early-onset deaf adults. To this end, we have developed a vibratory wearable—The Versatile Extra-Sensory Transducer (VEST)—by which auditory information is captured, digitally processed, and delivered to the skin of the torso via an array of small vibratory motors. We here present the current development status of our device and results of a speech perception experiment involving both hearing and deaf participants. Participants trained on the Vest by engaging in an identification task: on each trial, the participant was presented with a vibration-mapped stimulus of a spoken word from a training set of 50 phonetically balanced words. The participant was then presented with a set of options displayed on a screen from which they selected the word thought to have been felt, and they were given feedback on their choice. After 12 days of training, participants then ran the same procedure on a novel set of 50 words. Our results demonstrate evidence of learning and transfer of knowledge: participants perform better on their first day with the novel test set than their first day on the training set. Further, participants perform at or near chance the first time identifying a word from the training set, and above chance the first time identifying a word from the test set. Funding for this research is supported by the Renz foundation and a training fellowship from the Keck Center for Interdisciplinary Bioscience Training of the Gulf Coast Consortia (NIBIB Grant No. 5T32EB006350-05).

P-46  MODULATING CRAVING STATES IN COCAINE ADDICTION VIA REAL-TIME fMRI FEEDBACK

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Behavioral and neuroimaging studies suggest that cocaine addicts have executive dysfunction (Hester & Garavan, 2004), impairments in self-control (Goldstein et al., 2007), and elevations in aberrant activation of limbic and striatal networks (Kilts et al., 2001). Collectively, these findings suggest a major cognitive role in cocaine addiction. Here, we propose a potential approach for controlling cocaine addiction by directly modulating cognitive craving states using real-time fMRI feedback. Chronic cocaine users were recruited to participate in a 9-session fMRI study over the course of 3 weeks. In each session, subjects were shown cue-induced craving images of cocaine and paraphernalia, and their unique craving and suppression networks were identified. In another run of the same session, the trained model was used to show subjects, in real-time, feedback of their level of craving via a meter on the screen. The visual feedback allowed subjects to adjust their neural states to crave or suppress as instructed. Drug usage during and after the experimental course was assessed by urine analysis and self-report. By leveraging real time visual feedback about neural states, we aim to provide a therapy for treatment-seeking cocaine addicts that will allow them to directly manipulate the cognitive processes giving rise to their drug use.

P-47  INFERRING READOUT OF DISTRIBUTED POPULATION CODES WITHOUT MASSIVELY PARALLEL RECORDINGS

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Information about task-relevant variables is often distributed among neurons across multiple cortical areas. Neuronal responses are rarely independent of each other, but are correlated to some degree. Consequently, determining how neurons drive behaviour requires not only examining how individual neurons are correlated with behaviour, but also estimating the correlated variability among neurons. Precisely estimating the structure of correlated variability requires massively parallel recordings, which remains very difficult with current technology. Fortunately, it has recently been shown that the expansion in neural representation from sensory periphery will lead to a predictable pattern of correlations that ultimately limits the information content in brain areas downstream. We examined the implications of these so-called information-limiting correlations for the readout of distributed population codes in a simple discrimination task. Surprisingly we found that both the behavioural precision, as well as the correlation of individual neurons with behavioural choice (choice correlation) were determined largely by the relative magnitudes of neuronal weights in the different brain areas and not on their specific pattern. We also found that, in the presence of information-limiting correlations, the choice correlations of neurons within an area should all scale by the same factor following inactivation of other potentially task-relevant brain areas. Together, our results lead to a novel framework for inferring how different brain areas contribute to behavioural response. Specifically, we show that the contribution of a brain area can be inferred simply by observing how the magnitude of choice correlations of individual neurons within the area and the behavioural precision are affected by inactivating other areas, thus obviating the need for large-scale recordings.

P-48  TEMPORAL EVOLUTION OF INFORMATION IN NEURAL NETWORKS WITH INFORMATION

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Feedback constitutes an important attribute of information processing in the brain. Here we use a simple model system to identify how neural feedback transforms the internal representation of dynamic sensory variables. For analytical tractability, we investigate a linear dynamical system with additive gaussian noise, and relate the synaptic weight matrix to an effective Kalman filter that is embedded within it. The architecture of a recurrent network influences the information content of the network, as well as the dynamics of two experimental measurements often used to describe the computations of neural networks: the psychophysical kernel and choice probability. For this model, we compute the Fisher information of a general readout and compare its efficiency to that of an optimal leaky integrator. The optimal structure of the network is determined by both the persistence time of the stimulus and the Fisher information of sensory measurements. The psychophysical kernel correlates brief fluctuations of a dynamic stimulus to subsequent actions or perceptual estimate; the choice probability is the same measure applied to neural responses. Despite the similarity of these measures, we show they have quite different behaviors. The psychophysical kernel may be dominated by early or late stimuli, depending on the recurrence strength. Yet because the neural network accumulates signals, the choice correlations always increase, albeit with different shapes.
MOTHER’S TRAUMA MODULATES AMYGDALA RESPONSE TO INFANT DISTRESS

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While the neurobiology of post-traumatic stress disorder has been extensively researched, much less attention has been paid to the neural mechanisms underlying more covert but pervasive types of trauma (e.g., those involving disrupted relationships and insecure attachment). Here, we report on a neurobiological study documenting that mothers’ attachment-related trauma, when unresolved, undermines her optimal brain response to her infant’s distress. We examined the amygdala blood oxygenation level-dependent response in 42 first-time mothers as they underwent functional magnetic resonance imaging scanning, viewing happy and sad face images of their own infant, along with those of a matched unknown infant. Whereas mothers with no trauma demonstrated greater amygdala responses to the sad faces of their own infant as compared to their happy faces, mothers who were classified as having unresolved trauma in the Adult Attachment Interview displayed blunted amygdala responses when cued by their own infants’ sadness as compared to happiness. Unknown infant faces did not elicit differential amygdala responses between the mother groups. The blunting of the amygdala response in traumatized mothers is discussed as a neural indication of mothers’ possible disengagement from infant distress, which may be part of a process linking maternal unresolved trauma and disrupted maternal caregiving.

AUDITORY AND TACTILE FREQUENCY INTERACTIONS

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Temporal frequency information can be combined across modalities to form unified percepts. In separate experiments we explored frequency interactions by addressing two fundamental questions: (1) Is frequency information from audition and touch optimally integrated? (2) Do auditory and tactile frequency processing rely on shared neural mechanisms?

To determine whether auditory-tactile integration is statistically optimal, we tested subjects on a frequency discrimination task comprising unimodal (A or T) and bimodal (AT) trials and assessed whether a maximum-likelihood estimation (MLE) model accounts for AT frequency perception. According to MLE, 1) variance in the combined AT condition should be lower compared to the A and T conditions and 2) perceived frequency on AT trials should be a linear function of the A and T cues weighted according to their relative unimodal reliabilities. Preliminary results indicate that temporal frequency information may not be combined in a statistically optimal manner across audition and touch.

To test whether common neural mechanisms support auditory and tactile frequency processing, we tested subjects using a perceptual adaptation paradigm. Subjects performed tactile frequency judgments following prolonged auditory frequency adaptation. We predicted that tactile frequency sensitivity would be altered in a frequency-specific manner if audition and touch rely on the same frequency processing mechanisms. Preliminary data show greater sensitivity to vibrations when the auditory adapting frequency is similar to the tactile test frequencies.