# 20TH ANNUAL GCC THEORETICAL AND COMPUTATIONAL NEUROSCIENCE CONFERENCE

**FEB. 10, 2023**

**Houston, TX**

Gulf Coast Consortia
QUANTITATIVE BIOMEDICAL SCIENCES

The Gulf Coast Consortia (GCC), located in Houston, Texas, is a dynamic, multi-institution collaboration of basic and translational scientists, researchers, clinicians, and students in the quantitative biomedical sciences, who benefit from joint training programs, topic-focused research consortia, shared facilities and equipment, and exchange of scientific knowledge. Working together, GCC member institutions provide a cutting-edge collaborative training environment and research infrastructure beyond the capability of any single institution. GCC research consortia gather interested faculty around research foci within the quantitative biomedical sciences, and currently include: Theoretical and Computational Neuroscience, Antimicrobial Resistance, Cellular and Molecular Biophysics, Innovative Drug Discovery and Development, Immunology, Mental Health Research, Regenerative Medicine, Single Cell Omics,, and Translational Pain Research. GCC training programs currently focus on Biomedical Informatics, Cancer Therapeutics, Computational Cancer Biology, Molecular Biophysics, Pharmacological Sciences, Precision Environmental Health Sciences, and Antimicrobial Resistance. Current members include Baylor College of Medicine, Rice University, University of Houston, The University of Texas Health Science Center at Houston, The University of Texas Medical Branch at Galveston, The University of Texas M. D. Anderson Cancer Center, The Institute of Biosciences and Technology of Texas A&M Health Science Center and Houston Methodist Research Institute.

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# Agenda

February 10, 2023

9:00 Welcome and opening remarks
**Harel Shouval,** Univ. of Texas Health Science Center Houston
**Suzanne Tomlinson**, Gulf Coast Consortia

**Session I**

Moderator: **Xaq Pitkow**, Rice Univ./Baylor College of Medicine

9:10-10:00 *Flexible Identification of Cognitive Computations from Spikes*
**Tatiana Engel**, Cold Spring Harbor Laboratories

10:00-10:40 *Under Pressure: The Role of PIEZO ion Channels in Interoception*
**Kara Marshall**, Texas Children's Hospital

10:40-10:55 Break

**Session II**

Moderator: **Kreso Josic**, Univ. of Houston

10:55-11:45 *Neural Mechanism of Optimal Performance*
**Luca Mazzucato**, Univ. of Oregon

11:45-12:25 *Electrophysiologically-Anchored Analysis of Synaptic Dysfunction and Cognitive Deficits in the Continuum of Alzheimer's Disease Clinical Syndrome*
**Agenor Limon**, Univ. of Texas Medical Branch at Galveston

12:25-1:25 Lunch Break & Networking (event hall)

**Session III**

Moderator: **Itzel Olivos Castillo**, Rice Univ.

1:25-2:15 *The Origins of Irregular Spiking in the Neocortex*
**Nicholas Priebe**, Univ. of Texas

2:15-2:55 *A Lens into Cognition: The Geometry and Topology of Neural Systems*
**Evelyn Tang,** Rice Univ.

**Session IV**

Moderator: **Fabrizio Gabbiani**, Baylor College of Medicine

2:55-3:15 Poster highlights
*Phase Transitions in When Feedback is Useful*
**Lokesh Boominathan**, Rice University **Poster 2**

*Pattern Dynamics of Brain Waves affected by Alzheimer's Disease*
**Clarissa Hoffman**, University of Texas Health Science Center Houston **Poster 5**

## Agenda

*Bounded Rational Control When Confidence is Costly*
**Itzel Olivos Castillo**, Rice University **Poster 8**

*Role of Inactivating Potassium Current in Contrast-Dependent Collision Detection*
**Gil Shaulsky**, Baylor College of Medicine **Poster 9**

*A Deep Learning Approach to Naturalistic Tunings*
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*Cholinergic Modulation Of Firing Rate In CA1 Pyramidal Neurons Via TRPM4 Channel Activation: A Combined Modeling And Electrophysiology Approach*
**Carol Upchurch**, Louisiana State University Health Sciences Center **Poster 12**

*Conditioning with Mixed Representations in Heteroassociative Neural Networks Trained with Three-Factor Predictive Plasticity*
**Pantelis Vafidis**, Caltech **Poster 13**

*Intentional Activation of Arbitrary Hippocampal Place Cells in Rats*
**Yan Yu**, Baylor College of Medicine **Poster 14**

*Long-term Large-scale Tracking of Same Neuron Populations with Ultraflexible Oversampling Electrode Array in Mice Visual Cortex*
**Hanlin Zhu**, Rice University **Poster 15**

3:15-4:05 Poster presentation (event hall)

Moderator: **Harel Shouval,** Univ. of Texas Health Science Center Houston

4:10-5:00 *The Pervasiveness of Abstract Disentangled Representational Geometry in the Brain*
**Stefano Fusi,** Columbia Univ.

5:00 Closing Remarks-reception

Presenters in alphabetical order

**Tatiana Engel**, PhD
Assistant Professor
Princeton Neuroscience Institute
*Flexible Identification of Cognitive Computations from Spikes*

Tatiana Engel is an Assistant Professor at the Princeton Neuroscience Institute. She uses computational and theoretical approaches to investigate how coordinated activity arises from distributed neural circuitry to drive behavioral and cognitive functions. Her lab develops theory, models, and data analysis methods that leverage newly available large-scale activity recordings from behaving animals to uncover mechanisms of brain function. She also participates in a large-scale collaboration of experimental and theoretical neuroscientists, the International Brain Laboratory.

**Stefano Fusi**, PhD
Professor
Neuroscience
Columbia Univ.

*The Pervasiveness of Abstract Disentangled Representational Geometry in the Brain*

Stefano Fusi was born in Florence, Italy, and graduated in 1992 from the Sapienza University of Rome with a degree in physics. After his degree, he obtained a researcher position at the Italian National Institute for Nuclear Physics in Rome and started to work in the field of theoretical neuroscience. In 1999, he received a Ph.D. in physics from the Hebrew University of Jerusalem, Israel, and moved to the University of Bern, Switzerland, as a postdoctoral fellow. After visiting Brandeis University as a postdoctoral fellow in 2003, in 2005 he was awarded a professorial fellowship by the Swiss National Science Foundation and became an assistant professor at the Swiss Federal Institute of Technology in Zurich (ETHZ), Switzerland. In 2009, he joined the Department of Neuroscience at Columbia University as an associate professor. He is an associate editor of Frontiers in Computational Neuroscience and the Journal of Computational Neuroscience. Fusi's research involves the computational modeling and theoretical analysis of complex neural circuits with the goal of understanding the role of biological complexity and diversity in the nervous system. His laboratory collaborates with experimental neuroscientists at Columbia University, the Massachusetts Institute of Technology and Stanford University.

**Agenor Limon**, PhD
Assistant Professor
Neurodegenerative Diseases
Univ. of Texas Medical Branch at Galveston

*Electrophysiologically-anchored Analysis of Synaptic Dysfunction and Cognitive Deficits in the Continuum of Alzheimer's Disease Clinical Syndrome*

Dr. Agenor Limon is Associate Professor at the George P. and Cynthia Woods Mitchel Center for Neurodegenerative Diseases at the University of Texas Medical Branch (UTMB). He obtained his Doctorate in Physiological Sciences (Cum laude) from the Institute of Physiology at the Meritorious University of Puebla, and then, moved to UCIrvine to train with Distinguished Professor Ricardo Miledi as a postdoctoral fellow. His current major focus is to elucidate the physiological and pathophysiological processes that underlie synaptic and extrasynaptic remodeling of inhibitory and excitatory signaling in neurological disorders, and how they determine the global excitatory to inhibitory synaptic balance (E/I ratio) that determines the electrical brain activity in the brain. The approach uses electrophysiological data from reactivated, native synaptic receptors extracted from healthy and diseased human brains by Microtransplantation of Synaptic Membranes (MSM), and by integrating this functional information with transcriptomic and clinical data from the same cohort (electrophysiologically-anchored data analysis; EDA). This integrative approach has allowed the analysis of previously overlooked deficits, for example, inhibitory dysfunction in Alzheimer's disease associated to uncoupling of postsynaptic densities, proexcitatory synaptic imbalance in Alzheimer's disease, multimodal convergence on reduced excitatory efficacy of AMPA receptors in schizophrenia.

**Kara Marshall**, PhD
Assistant Professor
Neuroscience
Baylor College of Medicine

*Under Pressure: The Role of PIEZO ion Channels in Interoception*

Dr. Kara Marshall in an Assistant Professor in the Department of Neuroscience at Baylor College of Medicine. She received her Ph.D. from Columbia University, where she studied touch receptor neurons in the Lumpkin Lab. She then worked to understand how mechanical forces are detected inside the body in the Patapoutian lab at Scripps Research. Sensing internal forces is critical for a wide variety of processes, such as blood-pressure control, feeding, digestion, and urination. Despite its importance, we know little about the sensors, cells and physiology of internal force sensing. Her lab focuses on understanding how the nervous system detects internal forces in the urinary tract and other systems, and how this impacts physiology and behavior in health and disease.

**Luca Mazzucato**, PhD
Assistant Professor
Biology, Mathematics, Neuroscience, Physics
Univ. of Oregon

*Neural Mechanism of Optimal Performance*

Dr. Mazzucato is a theoretical physicist by training. He obtained his PhD in Theoretical Particle Physics at SISSA/ISAS in Trieste, Italy, in 2005 and worked on string theory and beyond the Standard Model physics at the Department of Particle Physics at Tel Aviv University (2005-2008) and as a Visiting Researcher at the Racah Institute of Physics at Hebrew University (2006). He was a Member at the Simons Center for Geometry and Physics (2008-2011), and a Visiting Scientist at the Kavli Institute for Theoretical Physics, Santa Barbara (2009). He began his neuroscience research in 2012 at the Department of Neurobiology and Behavior, Stony Brook University. He was a Swartz Fellow in Theoretical Neurobiology (2013-2014) and, since 2014, a NIH-funded Principal Investigator. He was an Associate Research Scientist at the Center for Theoretical Neuroscience at the Zuckerman Mind Brain Behavior Institute at Columbia University (2017/2018).

Abstract: When we attend a demanding task, our performance is poor at low arousal (when drowsy) or high arousal (when anxious), but we achieve optimal performance at intermediate arousal, yielding the celebrated Yerkes-Dodson inverted-U law. Despite decades of investigations, the neural mechanisms underlying this inverted-U law are unknown. In this talk, I will elucidate the behavioral and neural mechanisms linking arousal and performance under the Yerkes-Dodson law in a mouse model. I will show that mice during auditory and visual decision-making express an array of discrete strategies, including optimal, suboptimal and disengaged. The optimal strategy occurs at intermediate arousal, measured by pupil size, consistent with the inverted-U law. Using neuropixels recordings from large neural populations in auditory cortex, I will show that sound encoding is optimal at intermediate arousal, suggesting that performance modulations occur as early as primary sensory areas. To explain the computational principle underlying this inverted-U law, I will show that in a recurrent network with E/I populations arousal induces a phase transition from a multi-attractor to a single attractor phase, and performance is optimized near the critical region. The model further predicts a monotonic decrease in neural variability induced by arousal, which was confirmed in the empirical data. Our results establish a biologically plausible theory of optimal performance near phase transitions in recurrent networks, whose implications for brain-inspired AI models will be briefly outlined.

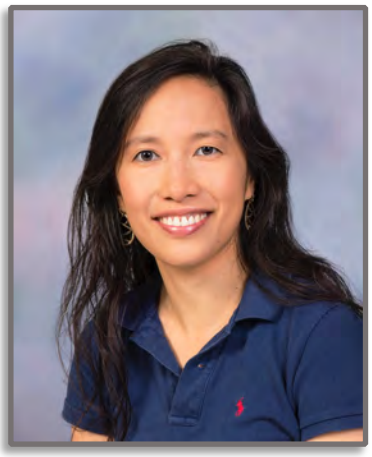
**Nicholas Priebe**, PhD
Professor
Neuroscience
University of Texas Austin

*The Origins of Irregular Spiking in the Neocortex*

I am interested in mechanisms underlying response properties of neurons in primary sensory cortex using both electrophysiology and imaging. I became interested in neuroscience as an undergraduate studying computer science and cognitive science at the University of California, San Diego. Upon graduation I worked in Dr. Terry Sejnowski's Computational Neuroscience Lab, where I enjoyed lively conversations and daily afternoon tea. I then entered graduate school at the University of California, San Francisco, where I worked initially with Dr Kenneth Miller using computational techniques and then completed my thesis with Dr Stephen Lisberger, in whose lab I studied the mechanisms of cortical adaptation and the neural representation of speed in the dorsal visual pathway. I then moved to Northwestern University to examine the mechanisms for selectivity in visual cortex using intracellular recordings with Dr David Ferster. We embarked on a series of studies testing whether a feedforward model could account for cortical motion and orientation selectivity. Following my work with Dr Ferster I established my lab at the University of Texas, Austin, where I study visual processing using a combination of 2 photon microscopy and electrophysiology in rodents and primates.

Abstract: The spiking responses of neocortical neurons are remarkably variable. Distinct patterns are observed when the same stimulus is presented in the sensory areas or when the same action is executed in motor areas. This is quantified across trials by measuring the Fano factor of the neuronal spike counts, which is generally near 1, consistent with spiking times following a noisy Poisson process. The two candidate sources for noise are the synaptic drive that converges on individual neurons or intrinsic transducing processes within neurons. To parse the relative contributions of these noise sources, we made whole-cell intracellular recordings from cortical slices and used in the whole cell dynamic clamp configuration while using dynamic clamp to injecting excitatory and inhibitory conductances previously recorded in vivo from visual cortical neurons (Tan et al. 2011). By controlling the conductance directly, we can test whether intrinsic processes contribute to poisson firing. We found that repeated injections of the same excitatory and inhibitory conductance evoked stereotypical spike trains, resulting in fano factors near 0.2. Varying the amplitude of both excitatory and inhibitory conductances changed the firing rate of recorded neurons but not the Fano factor. These records indicate that intrinsic processes do not contribute substantially to the Poisson spiking of cortical cells. Next, to test whether

differences in network input are responsible for Poisson spike patterns, we examined spike trains evoked by injecting excitatory and inhibitory conductances recorded from different presentations of the same visual stimulus. These records exhibited different behaviors depending on whether the injected conductances were from visually-driven or spontaneous epochs: during visually-driven epochs, spiking responses were Poisson (Fano factor near 1); during spontaneous epochs spiking responses were super-Poisson (fano factors above 1). Both of these observations are consistent with the quenching of variability by sensory stimulation or motor behavior (Churchland et al. 2010). We also found that excitatory conductances, in the absence of inhibition, are sufficient to generate spike trains with Poisson statistics. In summary, our results indicate that the Poisson spiking emerges not from intrinsic sources but from differences in the synaptic drive across trials, the nature of this synaptic drive can alter the nature of variability, and that that excitatory input alone is sufficient to generate Poisson spiking.



**Evelyn Tang**, PhD
Assistant Professor
Physics Department and the Center for Theoretical Biological Physics
Rice University

*A Lens into Cognition: The Geometry and Topology of Neural Systems*

Dr. Evelyn Tang is an assistant professor in the physics department and the Center for Theoretical Biological Physics, at Rice University. She did her PhD on quantum systems and topological phases at MIT with Xiao-Gang Wen. Thereafter, she switched into computational neuroscience as an Africk Family Postdoctoral Fellow at the University of Pennsylvania, focusing on cognition and brain networks. As a group leader subsequently at the Max Planck Institute of Dynamics and Self-Organization, her research has broadened to other questions in theoretical biological physics. Evelyn holds an MPhil from the University of Cambridge and a BS from Yale University. She is a recipient of the NSF CAREER award, a Scialog award from the RCSA and Kavli Foundation, a Simons-Berkeley Research Fellowship and the Gates Cambridge scholarship.

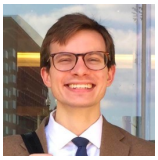
Rapid Fire Presenters

*Phase Transitions in When Feedback is Useful*
**Lokesh Boominathan**, Rice University **Poster 2**

*Pattern Dynamics of Brain Waves affected by Alzheimer's Disease*
**Clarissa Hoffman**, University of Texas Health Science Center Houston
**Poster 5**

*Bounded Rational Control When Confidence is Costly*
**Itzel Olivos Castillo**, Rice University **Poster 8**

*Role of Inactivating Potassium Current in Contrast-Dependent Collision Detection*
**Gil Shaulsky**, Baylor College of Medicine **Poster 9**



*A Deep Learning Approach to Naturalistic Tunings*
**Mitchell Slapik**, McGovern Medical School **Poster 11**

*Cholinergic Modulation Of Firing Rate In CA1 Pyramidal Neurons Via TRPM4 Channel Activation: A Combined Modeling And Electrophysiology Approach*
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**Hanlin Zhu**, Rice University **Poster 15**

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| Lokesh | Boominathan | Rice Univ. | Phase transitions in when feedback is useful | 2 |
| Melissa | Franch | UTHealth | Visual-frontal network interactions during learning social cooperation in freely moving macaques | 3 |
| Aravind Chenrayan | Govindaraju | Rice Univ. | Effect of K+ Relief of Block in gK,L on Non-Quantal Transmission at the Vestibular Hair Cell-Calyx Synapse | 4 |
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Poster 1

## *A Graph Signal Processing Model of the Cochlea*

Bonomo ME[1], Segarra S[2], Raphael RM[1]

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2. Department of Electrical and Computer Engineering, Rice University

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**Background:** Sound encoding in the cochlea has traditionally been understood to be the product of individual cells being excited at characteristic frequencies. However, this representation does not appreciate the relationships in the functional activity among all of these cells, which may be especially important while they are encoding music with many simultaneous pitches and timbre mixtures.

**Hypothesis/Goals:** Here, we take a novel graph approach to study sound encoding in the cochlea. Graph signal processing is used to look at how the response of individual cells is coordinated at the level of the whole-cochlea to pass complex information to the auditory nerve fibers.

**Methods:** We use a simulation (UR EAR 2020b) developed by the Carney Lab at the University of Rochester that incorporates the nonlinear response properties of the inner ear to compute hair cell voltages. Audiograms from patients with normal hearing, conductive hearing loss, sensorineural hearing loss, and both conductive and sensorineural hearing loss are extracted from the AudGenDB database (Children's Hospital of Philadelphia) and input as hearing health parameters in the simulation. We use the simulation to generate inner hair cell responses for various musical stimuli. The GSPbox Graph Signal Processing package (Version v0.7.5, 2018) is used to learn the graph links between hair cells from smoothness in their voltage signals and determine a cochlea graph for each patient. Graph theoretic metrics are then calculated to quantify differences among patients.

**Results:** There are significant differences in graph density, node weighted degree, and modularity between patients with normal hearing and those with hearing loss. Patients with normal hearing have lower graph density and higher weighted degree, meaning that these graphs generally have less links between hair cells, but these links are strong. These normal hearing graphs also have more modules, where a module is a group of tightly interacting hair cells. The graphs of patients with both conductive and sensorineural hearing loss were generally the noisiest (i.e., many weak links) and had fewer, less-specialized modules.

**Conclusion:** This project is the first to explore the representation of cochlear sound encoding as a graph using graph signal processing. Using this approach with patient data, we observe various graph-theoretic features that distinguish hearing loss diagnoses. Our method has the potential to improve cochlear implant signal processing of music. In particular, patient cochlea graphs can be used to develop a graph deep learning model for learning individualized signal processing settings, to ensure that melody information is not lost between channels.

**Acknowledgements:** M.E.B. is supported by a training fellowship from the Gulf Coast Consortia, on the NIH National Library of Medicine Training Program in Biomedical Informatics and Data Science (T15LM007093). This work additionally supported by a Rice University IDEA Grant.

Poster 2

***Phase Transitions in when Feedback is Useful***

Boominathan L[1], Pitkow X[1, 2]

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2. Department of Neuroscience, Baylor College of Medicine
Corresponding author: Lokesh Boominathan, Department of Electrical and Computer Engineering, Rice University, 6100 Main St, Houston, TX, E-mail: lb36@rice.edu

**Background:** A critical computation for the brain is to infer the world's latent variables from ambiguous observations. Computational constraints, including metabolic costs and noisy signals, limit the performance of these inferences. Efficient coding is a prominent theory that describes how limited resources can be used best. In one incarnation, this leads to the theory of predictive coding, which posits that predictions are sent along feedback channels to be subtracted from signals at lower cortical areas; only the difference returns to the higher areas along feedforward channels, reducing the metabolic or informational cost of sending redundant signals already known to the higher areas. This theory does not, however, account for the additional costs or noise associated with the feedback.

**Hypothesis/Goals:** Depending on the costs for sending predictions and the reliability of signals encoding those predictions, we expect different optimal strategies to perform computationally constrained inferences. For example, if the feedback channel is too unreliable and expensive, we hypothesize that it is not worth sending any predictions at all. Here we offer a more general theory of inference that accounts for the costs and reliabilities of the feedback and feedforward channels, and the relative importance of good inferences about the latent world state.

**Methods:** We formulate the inference problem as control via message-passing on a graph, maximizing how well an inference tracks a target state while minimizing the message costs. Messages become control actions with their own costs to reduce while improving how well an inference tracks a target state. We call this method inference as control, as it flips the interesting perspective of viewing optimal control as an inference problem. We solve this problem under Linear-Quadratic-Gaussian (LQG) assumptions: Linear dynamics, Quadratic state and control costs, and Gaussian noise for the process, observations, and messages.

**Results:** Our theory enables us to determine the optimal predictions and how they are integrated into computationally constrained inference. This analysis reveals phase transitions in when feedback is helpful, as we change the computation parameters or the world dynamics. Finally, we connect our theory's constraints to biology by providing a simplified example of how feedback/feedforward pathways with different anatomical structures can yield different optimal strategies.

**Conclusions:** We defined a new class of dynamic optimization tasks that reflect essential biological constraints on inference in the brain, by including cost and noise for each recurrently connected computational element. We solve this optimization problem by modeling inference as a control problem with prediction as self-control. The resultant optimization provides nontrivial predictions for when we expect suppressive feedback as a function of biological constraints, computational costs, and world dynamics.

**Acknowledgments:** The authors thank Itzel Olivos Castillo, Ann Hermundstad, Krešimir Josi´c, and Paul Schrater for useful discussions. This work was supported in part by NSF CAREER grant 1552868, the McNair Foundation, and AFOSR grant FA9550-21-1-0422 in the Cognitive and Computational Neuroscience program.

Poster 3

***Visual-frontal Network Interactions During Learning Social Cooperation in Freely Moving Macaques***

Franch M[1], Yellapantula S[2], Wright A[1], Aazhang B[1,2], Dragoi V[1,2]

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2. Department of Electrical and Computer Engineering, Rice University, Houston, TX

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**Background:** Social interactions provide meaning and fulfillment to our everyday life. Learning social behavior relies on interpreting and responding to visual cues from others, especially in humans and primates. Yet the neural mechanisms of viewing social information to support social goals has remained unknown.

**Hypothesis/Goals**: We predicted that neural populations within cortical brain regions that process visual information and decision-making would improve computation of social variables to support social learning, and that enhanced communication between regions would also correlate with learning.

**Methods:** Here, we wirelessly recorded the spiking activity of populations of neurons in visual and prefrontal cortex in conjunction with wireless recordings of oculomotor events while freely moving macaques learned to cooperate. This allowed us to examine how visual representations relevant for social interactions are used by executive areas that encode reward value and decision making.

**Results:** Animals learned to cooperate by increasing viewing of social cues (reward and conspecific) to influence or predict cooperation, thus improving coordination of their actions. We discovered that as animals learn to cooperate, there is an improvement in the representation of social variables, such as the conspecific or reward, across visual and executive brain regions. Decoding population activity in prefrontal cortex reveals that viewing social cues influences self and other's decision to cooperate. Specifically, during social events, there is an increase in coordinated spiking between visual and prefrontal cortical neurons that occurs during learning.

**Conclusions:** These results demonstrate how the visual-frontal cortical network prioritizes relevant sensory information to facilitate learning social interactions while freely moving macaques interact in a naturalistic environment.

**Acknowledgements:** All data was recorded and analyzed by Melissa Franch in the Dragoi lab. Eye tracking data was also analyzed by Sudha Yellapantula with assistance from the Aazhang lab**.** Supported by NINDS NIH BRAIN Initiative U01NS108680 and by NIMH NIH 1F31MH125451-01A1

Poster 4

## *Effect of $K^+$ Relief of Block in $g_{K,L}$ on Non-Quantal Transmission at the Vestibular Hair Cell-Calyx Synapse*

Govindaraju AC[1,2] Lysakowski A[3], Eatock RA[4], Raphael R[1]

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2. Applied Physics Graduate Program, Rice University.
3. Department of Anatomy and Cell Biology, University of Illinois at Chicago.
4. Department of Neurobiology, University of Chicago.

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**Background.** Type I hair cells, which detect head motion, sit within and transmit to cup-shaped terminals (calyces) of afferent neurons. These neurons guide motor reflexes that maintain gaze, balance, and our sense of orientation. In addition to glutamate release from vesicles (quantal transmission), ions flow through the basolateral hair cell (pre-synaptic) membrane into the synaptic cleft and through the inner calyx (post-synaptic) membrane (nonquantal transmission-NQT). The synaptic cleft cannot be accessed by reference electrodes without disrupting its structure and function. As a result, gradients in ion concentrations and electric potential within the cleft cannot be measured and pre-/post-synaptic membrane voltages cannot be directly obtained. We have developed a computational, biophysical, model of the synapse to overcome this limitation. Using this model, we previously presented the sequence of events underlying NQT and the role of changes in cleft $[K^+]$ and cleft electrical potential in driving post-synaptic currents during hair cell stimulation (Govindaraju et al. 2023). We now present simulations that explore the effects of $K^+$ relief of block (KRB) in $g_{K,L}$, a depolarization activated $K^+$ conductance on the hair cell basolateral membrane, (Contini et al. 2020) on NQT.

**Hypothesis/Goal.** To investigate the effects of KRB on nonquantal transmission.

**Methods.** To simulate transmission between hair cell and afferent neuron, our model uses Hodgkin-Huxley-style ion currents based on whole-cell recordings, continuity equations to describe changes in electric potential within hair cell, cleft, afferent calyx and fiber, and electro-diffusion equations for cleft $K^+$ and $Na^+$. Step or sinusoidal hair bundle deflection or voltage step protocols are used as input. This was accomplished by extrapolating the concentration dependence of voltage block parameters from two (4 and 20 mM) datapoints provided in recent literature (Contini et al. 2020).

**Results and Conclusion.** Upon replacing the steady-state activation-voltage curve for $g_{K,L}$ in our previous model with the KRB (cleft $[K^+]$ and voltage-dependent) curve, the current through $g_{K,L}$ is slightly reduced and resting hair cell potential shifts positively. This may be of relevance to quantal transmission. The KRB activation curve is broader, with a higher open probability negative to rest and lower open probability positive to rest. For sinusoidal hair bundle displacements, use of the KRB curve had modest effects on the amplitude and phase of the simulated calyx postsynaptic potential. More experimental data at different concentrations are needed to refine the KRB model.

**Acknowledgements.** Supported by NIH-NIDCD R01 DC01234, Emerging Research Grant from the Hearing Health Foundation, and a seed grant from the Rice University ENRICH program.

Poster 5

***Pattern Dynamics of Brain Waves affected by Alzheimer's Disease***

Hoffman C[1], Cheng J[2], Ji D[2], Dabaghian Y[1]

1. Department of Neurology, The University of Texas McGovern Medical School
2. Department of Neurosciences, Baylor College of Medicine

Corresponding author: Yuri Dabaghian, Department of Neurology, The University of Texas McGovern Medical School, 6431 Fannin St, Houston, TX, Email: Yuri.A.Dabaghian@uth.tmc.edu

**Background:** We focus on understanding the impact of Alzheimer's Disease (AD) pathology at the neurocircuit level. The main physiological manifestation of circuit activity is the synchronized extracellular field, which gives rise to the recorded local field potential (LFP). These fields are widely studied using a variety of methods which primarily address time-localized (instantaneous) or the time-averaged characteristics of LFPs.

**Hypothesis/Goals:** We hypothesize that AD-damaged hippocampal circuits produce abnormal LFP dynamics that clearly manifest at the level of wave patterns and waveforms.

**Methods:** We propose an alternative approach that focuses on the morphologies of waveforms—the patterns of the brain waves over finite timescales. Specifically, we use two independent methods for quantifying the structural regularity and irregularity of brain waves and correlate the resulting "stochasticity scores" with behavior. The first, quantifies the pattern's consistency with the underlying mean. The second, measures how "structured" or "orderly" (e.g., periodic-like or time-clustered) the pattern is.

**Results:** Our previous work in wild-type mice revealed a curious interrelationship between morphologies of θ-waves, γ-waves, and sharp wave events (SWE) and the animal's speed and acceleration. We also noticed spatial clustering of waves with different morphology along the animal's trajectory, reminiscent of hippocampal place fields. Based on these observations, we studied circuit activity of hippocampal networks in AD brains and found a number of alterations in LFP rhythmicity. For example, there is a loss of distinct quiescence and movement states in the AD model, indicating an ambiguity of physiological context. Furthermore, the coupling of waveform patterns with speed is weaker in the $\theta$ and SWE in AD, yet, the strength of coupling between $\gamma$-patterns and speed is strengthened revealing potential $\theta$-$\gamma$ dysfunction. Additionally, the spatial selectivity of patterns is lost in AD, suggesting that damaged synaptic circuits compromise wave patterning and information exchange between brain regions. This failure to discriminate environmental position may be a potential cause of pattern dysfunction, and thus memory loss in AD.

**Conclusions:** These differences in brain wave patterns can be used to better understand and potentially to detect circuit-level pathologies in AD brains. Overall, these results offer a novel perspective on studying the structure, the dynamics, and the functionality of the brain waves and will provide a deeper understanding of AD at a neurocircuit level.

Poster 5

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Poster 7

***An Extension of Poisson Gaussian-Process Latent Variable Model for Unsupervised Neural Decoding***

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**Background:** Hippocampal neural activity exhibits both a precise temporal code reflecting an animal's position in an environment as well as a large-scale contextual code reflecting distinct maps for different environments. While contextual place codes can be extracted from correlated neural firing rates, to elucidate the more complex memory codes which the hippocampus supports, unsupervised methods for extracting context-dependent ensemble firing patterns are required.

**Methods:** The Poisson Gaussian-process latent variable model (P-GPLVM) is a probabilistic, nonlinear, and dynamic dimensionality reduction approach that infers temporally smooth low-dimensional latent neural trajectories and smooth, non-parametric tuning curves from spike trains without referring to external variables [1]. However, the original model lacks an approach for projecting new input into the learned latent space, limiting its utility for decoding new neural data, especially during the time-compressed sequential reactivation of hippocampal neuronal ensembles during population burst events (PBEs) within sharp-wave ripples, which don't have behavioral variables as reference. Here, we extend the P-GPLVM framework to enable the projection of new neural data constrained by the learned smoothness parameters and tuning curves. We also describe a principled approach for projecting PBE data and providing metrics for assessing the projection.

**Results:** We apply our methods to hippocampal neural activity recorded from a rat running back and forth on a linear track. We simulate remapping in another context by randomly permuting the cell identities during the second half of the session. Trained from neural data during running, the model clusters data points from the two contexts into two separate manifolds in the latent space, each with a bifurcating shape representing the two running directions. The animal's position is encoded smoothly along each manifold. When projecting new neural data during behavior and PBEs into the learned latent space, neural trajectories lay accordingly and smoothly on the manifold, thereby allowing the external variables to be derived.

**Conclusions:** Our results indicate that this extension of P-GPLVM is capable of revealing neural trajectory evolution, disentangling neural patterns between different contexts, and decoding external variables (i.e. animal position, running direction, context) from neural activity both during behavior and during PBEs in an unsupervised manner.

**Acknowledgements:**This work is supported by CRCNS R01- R23340.

**Reference**

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Poster 8

***Bounded rational control when confidence is costly***

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**Background:** Optimal feedback control appeals to the conceptual framework of accumulating evidence and selecting actions based on the synthesized information to maximize expected utility; however, it neglects the cost of representing information. Conversely, efforts to study metabolically efficient ways of coding sensory information do not consider the consequences of closed-loop control or focus only on optimizing the sensory apparatus. Here we combine concepts of efficient coding with control theory to develop a version of stochastic control that accounts for computational costs in the brain.

**Goal:** To analyze how bounded-rational agents balance task performance against the metabolic cost of representing uncertainty and the moving effort required to compensate for suboptimal inferences.

**Methods:** Brains evolved to efficiently transform sensory information into motor commands that satisfy an optimality criterion. We mathematize this phenomenon as a Partially Observable Markov Decision Process (POMDP). An agent solving a POMDP perceives the world through noisy sensors, uses recursive Bayesian inference to synthesize an evolving posterior distribution (belief) on the hidden world state, and uses this belief to guide behavior. For tractability, we restrict our study to Linear Quadratic Gaussian (LQG) settings, where combining a Kalman filter with a linear regulator suffices to minimize expected costs optimally. In our study, an agent aims to minimize the deviation of a controllable state from a target while accounting for moving effort and the neural cost involved in encoding the sources of uncertainty. We implement the Kalman filter neurally using a dynamic Probabilistic Population Code, in which linear projections of spiking neural activity approximate the natural parameters of Gaussian posteriors. To the classic LQG cost, we add the total integrated number of spikes used by the neural circuit to encode observations and inferences; this results in a bounded framework where confidence is costly. To solve this problem, we first create an augmented system that describes the interactions between state, observations, estimates, and actions; next, we rewrite the expected total cost in terms of the system's steady-state covariance matrix; finally, we use a numerical optimization method to minimize the loss subject to stability constraints.

**Results:** Different world properties trigger a phase transition that dictates when to switch from reacting immediately to evidence to drawing conclusions based on past observations, actions, and assumed world model. In contrast to classic LQG, resource constraints link control to estimates (this is because saving spikes requires stronger control gains to compensate for estimation errors). Unpredictable worlds are fragile to model mismatch, so a bounded-rational agent benefits from modeling as much information about the world as possible. In both near-stable and volatile worlds, optimization mainly reduces neural costs, with a small impact on task costs.

**Conclusions:** For PPCs encoding probability distributions, the precision of observations and inferences is directly proportional to the number of spikes. In this framework, high-quality observations and world models that yield optimally updated beliefs may not be affordable. To compensate for this constraint, the bounded-rational agent follows two strategies (reactive and integration) that balance error-correction efforts and neural cost. Simulations confirm our analytical predictions.

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Poster 9

***Role of Inactivating Potassium Current in Contrast-Dependent Collision Detection***

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**Background:** In grasshoppers, visual detection of approaching predators is accomplished by the Lobula Giant Movement Detector (LGMD). The LGMD is an identified neuron in the optic lobe that integrates OFF retinotopic inputs originating from every facet of the eye within a large dendritic field A. An inactivating $K^+$ conductance ($K_D$) in field A is critical to discriminate the spatial coherence of black looming stimuli. ON excitation for white looming stimuli impinges non-retinotopically on a separate dendritic field C, and the LGMD does not discriminate their spatial coherence.

**Hypothesis:** $K_D$ conductance is likely restricted to field A, where retinotopic inputs impinge.The lack of retinotopic ON inputs suggests that block of $K_D$ should increase responses to white looms, independent of coherence.

**Methods:** To determine how $K_D$ impacts ON/OFF spatial coherence computations, we applied blockers to dendritic fields A and C and recorded neural responses while presenting ON and OFF looming stimuli. In addition, we performed simulated visual experiments on a biophysical model of the LGMD with and without $K_D$ conductance.

**Results:** Application of a $K_D$ blocker to field A caused an increase in responses to all visual stimuli. Responses to spatially incoherent black stimuli increased more than did responses to solid black ones, but no similar spatial selectivity was observed in responses to white stimuli. Application of the same $K_D$ blocker to field C caused no increase in responses at all. Simulations with $K_D$ conductance only in field A best recapitulate experimental results.

**Conclusions:** $K_D$ conductance in field A contributes to spatial discrimination in the LGMD's response to black visual stimuli but not to white, and there is likely no functional $K_D$ conductance in field C. These experiments provide insights into dendritic computations required for contrast-polarity specific approaching object segmentation.

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Poster 10

***Synaptic Motility and Functional Stability in the Whisker Cortex***

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**Background:** Synaptic weights are highly volatile, raising the question: how can the brain retain its functionality in the face of constant synaptic remodeling? Here we use the whisker system of rats and mice to study the interplay between synaptic plasticity (motility) and the transmission of sensory signals downstream .By rhythmically moving their whiskers back and forth, rats and mice probe their surroundings. The azimuthal position of a whisker can be estimated from the activity of whisking neurons, which are neurons that respond selectively to a preferred phase along the whisking cycle. However, simple models for the transmission of the whisking signal downstream predict a distribution of preferred phases that is an order of magnitude narrower than empirically observed.

**Goals:** Here we examine the hypothesis that spike timing-dependent plasticity (STDP) may underlie the unexplained distribution of preferred phases in layer-2/3 (L2/3). We study under what conditions inhibitory STDP of layer-4 (L4I) to L2/3 synapses can generate a non-trivial distribution of preferred phases and investigate how the STDP rule shapes this distribution.

**Methods:** This hypothesis is addressed in the framework of a modelling study, that investigates the STDP dynamics of a population of synapses that propagate the whisking signal downstream in a feed-forward manner. In the framework of our model, we ignore the contribution of recurrent connections to the STDP dynamics.

**Results**: We find that for a wide range of parameters, STDP dynamics do not relax to a fixed point, but rather, converge to a limit cycle in which the synaptic weights remain dynamic. As a result, the preferred phases of downstream neurons drift in time with a non-uniform velocity governed by the STDP rule, which, in turn, induces a non-uniform distribution for the preferred phases of the downstream population, Fig. 1.

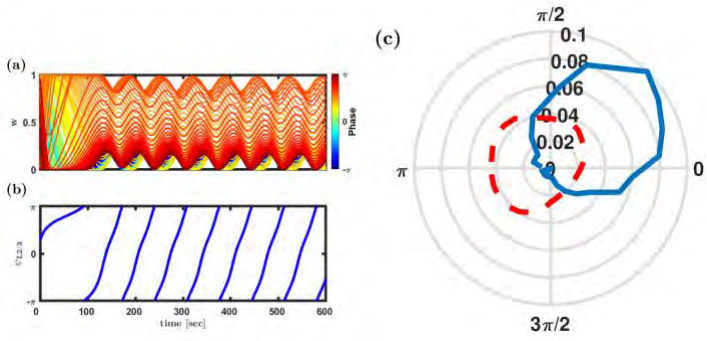


Fig. 1: **Simulation of the STDP dynamics**. (a) Synaptic weight dynamics. Each trace depicts the time evolution of a single synaptic weight, differentiated by color according to its preferred phase, see legend. (b) Dynamics of the preferred phase of the downstream neuron, ψ_(L2/3) is shown as a function of time. (c) Polar plots of the phase distributions of the L4I neurons (blue) with κ_L4I=1.5 and ψ_L4I=0.25π and the L2/3 neuron (dashed red) with κ_L4I≈0.4 and ψ_(L2/3)=2.6 rad. The width and mean phase of the distribution are approximated by the von-mises distribution Pr[fo](ϕ)=e^κCos(ϕ-ψ) /2πI_0 (κ), where I_0 (κ) is the modified Bessel function of order 0.

**Conclusions**: Our theory provides a natural prediction by linking the distribution of preferred phases and the STDP rule. In particular, we find that temporally symmetric rules yield phase distributions that are different when compared with asymmetric rules. Moreover, our theory demonstrates how functionality can be retained not only despite but because of the

constant synaptic motility. Thus, a continuous change in synaptic weights is not an artifact, but rather a feature that plays an important role in the transmission of spatiotemporal signals in the whisker system.

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Poster 11

***A Deep Learning Approach to Naturalistic Tunings***

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**Introduction:** Visual cortex builds a visual representation of our world, starting from basic features like lines and edges and leading up to more complex features like shapes and objects. However, these tunings are generally examined separately: it remains unclear how they come together when many visual features are combined in naturalistic settings. Using new machine learning techniques, we investigate tunings to these multi-faceted stimuli and demonstrate the encoding of naturalistic features in early visual cortex. In the process, we better capture the functioning of visual cortex in everyday life.

**Methods:** We present visual stimuli to a macaque monkey on a computer monitor, and record from early visual cortex (V1) using a Plexon laminar electrode. Our machine learning algorithm was pioneered by Ponce et al. (2019) and consists of two components: an image generator and an optimizer. The image generator, a generative adversarial network or "GAN," is trained on over a million natural images and can flexibly produce a wide range of stimuli that resemble textures, objects, and landscapes. Meanwhile, our optimizer, a co-variate matrix adaptation evolution strategy or "CMA-ES," uses neural feedback to iteratively evolve these stimuli and maximize the firing rate of a target neuron in early visual cortex.

**Results:** Our results validate a new method of creating optimal stimuli in early visual cortex. By combining an image generator and an optimizer, we can consistently develop "optimal stimuli" that vastly outperform traditional stimuli such as oriented gratings or uniform colors. These optimal stimuli tend to incorporate preferred orientations and colors as measured by single-modality tasks, showing how these features optimally combine in naturalistic images. However, they also include suboptimal orientations and colors, as well as more complex features like textures and shapes not traditionally associated with early visual cortex. These findings reveal the diverse tunings of early visual cortex in response to naturalistic stimuli.

**Discussion:** In this study, we demonstrate the power of using machine learning and neural feedback in stimulus design. Optimal stimuli vastly outperform traditional, single-modality stimuli and incorporate a diverse range of visual features such as color, shape and texture. This supports an overarching view of visual cortex as encoding multi-faceted, naturalistic features rather than simple, single-modality features like orientation or color alone. Furthermore, it shows how neural feedback can be used to guide stimulus design and discover new properties of neural circuits.

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Poster 12

***Cholinergic modulation of firing rate in CA1 pyramidal neurons via TRPM4 channel activation: a combined modeling and electrophysiology approach***

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**Background:** Many CA1 pyramidal cells function as place cells, firing at higher rates within a preferred place field. Upon repeated traversals of the place field, firing has been shown to shift earlier, becoming asymmetric, particularly as novel environments become more familiar. Higher levels of the neuromodulator acetylcholine (ACh) are associated with novelty, and thus may decrease with experience. Such a decrease in ACh may partially account for the shift in the center of mass of the place field.

**Goals:** Through a combination of *in vitro* electrophysiology and *in silico* modeling, we aim to show possible mechanisms of cholinergic modulation of CA1 place cells.

**Methods:** Using *in vitro* electrophysiology in slices from male rats and *in silico* simulations in a multicompartmental model of a CA1 pyramidal neuron, we investigated cholinergic modulation of the firing rate adaptation that occurs in response to symmetric ramps of depolarizing current input. This symmetric input approximates the spatially-tuned, temporally-diffuse depolarizing synaptic input received by these neurons while traversing a place field. Left or rightward shifts in firing along the current ramp were quantified as a normalized adaptation ratio, positive numbers indicate firing mostly on the up-ramp, while negative numbers indicate firing mostly on the down-ramp. Our model was a multicompartment conductance-based model based on our previous work modeling adaptation in these cells. To model cholinergic modulation, we incorporated a step change in IP3, and the resulting increased calcium induced calcium release (CICR) from the endoplasmic reticulum. As TRPM4 has a high calcium EC50, we assumed this CICR was tightly paired to TRPM4 channels via a nanodomain.

**Results:** The cholinergic agonist carbachol (CCh) modulates spike rate adaptation and shifts the normalized ratio in the negative direction. The non-specific TRP antagonist flufenamic acid (FFA) significantly reverses the effect of CCh, suggesting that this effect is due to activation of the Ca2+-activated nonspecific cation current, ICAN, carried by TRP channels. The TRPC-specific antagonist SKF 96365 does not significantly affect the CCh-associated decrease, but CBA, a blocker specific for TRPM4, does. Our model with a nanodomain paired to TRPM4 replicated the shift. Without the nanodomain, the model failed to replicate the shift. These results suggest the TRPM4 channel and IP3 mediated CICR are spatially colocalized.

**Conclusions:** In summary, our synergistic experimental/computational approach led to improved mechanistic understanding of the intrinsic mechanisms involved in the cholinergic modulation of firing rate, that have implications for the dependence of place cell firing on position within the place field.

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Poster 13

## *Conditioning with Mixed Representations in Heteroassociative Neural Networks Trained with Three-Factor Predictive Plasticity*

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**Background:** Animals learn from experience the value of environmental stimuli to guide their behavior. This requires the capacity to associate neural activity patterns induced by unconditioned stimuli (*US*) with patterns produced by conditioned stimuli (*CS*). Existing models of reward-modulated synaptic plasticity explain conditioning when the neural representations of behavioral stimuli are unmixed [1], but not when they are mixed. This is a problem because the relevant frontal cortical neurons typically display mixed selectivity [2].

**Hypothesis/Goals:** We propose a computational model that learns *US*-*CS* associations with mixed representations, which is inspired by experimental findings on the associative power of single cortical pyramidal neurons [3].

**Methods:** The model uses a local learning rule operating in compartmentalized neurons, which mirrors the capacity of cortical pyramidal neurons to implement predictive learning through coincidence detection [4]. This allows L5 feedforward (external) *US* inputs to be separated from L1 feedback (contextual) *CS* information and compared within the same neuron via backpropagating action potentials, which drives learning.

**Results:** We model a primary reinforcer area with a population of such neurons and find that, over time, the population response to the *CS* resembles that to the *US*, thus implementing the psychological process of stimulus substitution. Importantly, after learning, downstream decoding units can predict the *US* when only the *CS* has been presented and guide behavior. We show that adding global gating from reward prediction errors allows the model to account for a wide gamut of conditioning phenomena, including S-shaped acquisition, interstimulus interval effects, extinction, blocking, salience effects and overexpectation. Moreover, we find that the model offers a reductionist mechanism for causal inference by resolving the *post hoc* fallacy, which states that when event Y occurs after event X, then X is considered its cause.

**Conclusions:** The model makes testable predictions about the evolution of neural mixed representations during canonical conditioning experiments that we hope will be useful to understand existing datasets and guide future experiments. Other potential applications include decoding evolving beliefs in real time, validating conditioning models and investigating how it can go awry in brain disorder. We believe that pyramidal neurons are not by chance the most populous in the mammalian cortex; it might have been favored by evolution for its ability to accomplish one of the feats of organized life: predicting external contingencies.

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Poster 14

***Intentional Activation of Arbitrary Hippocampal Place Cells in Rats***

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**Background:** Hippocampal place cells fire spikes when an animal is at specific locations of an environment, called place fields. Place cells encode spatial memory and can be selectively activated during memory recall for spatial decisions. However, how these cells are selectively activated to meet intended task demands in the hippocampal neural circuits remains unknown.

**Hypothesis:** Aiming to establish a rodent model of intentional activation of memory cells, we tested whether rats could be trained to activate arbitrarily chosen place cells in the hippocampus.

**Methods:** We built a system for online detection of place cell firing activity (spikes), for closed-loop delivery of activity-driven feedback sensory cues (sounds), and for cue-triggered rewards. We trained five freely moving rats in a novel task to activate arbitrarily assigned single hippocampal CA1 place cells for rewards in three environments (Y-maze, rectangle maze, and a small confined space).

**Results:** In the Y-maze and the rectangle maze, rats were able to trigger the firing of a specific assigned place cell (target cells), by repeatedly running into its place field, which was originally hidden to the animals, like seeking a hidden platform in the Morris water maze. As a result, compared to control running sessions in the absence of sounds or rewards, the target cell's firing rate was increased and its inter-spike intervals were reduced. As the activity threshold for rewards was gradually increased, the target cell was activated with stronger and stronger intensity to meet the requirement. These changes of activity were specific to the target cell and were not observed in non-target cells that were active during running in the same session. Moreover, in the confined small space, certain target cells naturally silent or with very low firing rates were activated with much stronger place fields than control sessions in the same small space.

**Conclusions:** Our findings support our novel task as a rodent model for studying neural circuit mechanisms in intentional memory recall and reveal a remarkable flexibility and selectivity in the activation of hippocampal memory cells.

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Poster 15

***Long-term Large-scale Tracking Of Same Neuron Populations With Ultraflexible Oversampling Electrode Array In Mice Visual Cortex***

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**Background**, the brain is formed by massively interconnected and constantly evolving networks of neurons that communicate in millisecond timescale. Therefore, the ability to reliably track gradual changes of local neural circuits longitudinally with high temporal resolution is crucial for studying how changing brains maintain stable representations of external world. Conventional rigid electrodes provide simultaneous large scale neuronal recording. However, their capability to follow the same neurons over extended period remain elusive due to the gradual degradation of cell-electrode interface attributable to the chronic neuro-inflammatory responses induced by the mechanical mismatch. In addition, such mechanical mismatch under physiological micro-motions induces relative displacements at the biotic-abiotic interface, making same neurons difficult to track.

**Methods,** we demonstrate large scale chronic tracking of the same neuronal ensembles with ultraflexible electrode arrays featuring oversampling contact densities. A total of 25 ultraflexible probes, 32 channels each, were implanted in the visual cortices in 5 mice. After 50 days of surgery recovery and 10 days familiarization, repeated recording under visual stimulation was performed for 15 consecutive days during which a battery of diverse stimuli, including drifting gratings (DG), static gratings, spatially sparse gabors, and natural images were shown for 2.5hr total. Recordings days were spike-sorted individually before comparing units identified in each day against those detected on all other days by waveform similarity and firing autocorrelogram and linked same neurons according to a semi-automatic ensemble hierarchical clustering method.

**Results**, 1204 single units were identified across 15 days. They appeared for 10.96 ± 5.25 days, with 757 neurons appeared on both day 1 and day 15, whose weighted center location moved 3.0875±2.2331μm. Average unit firings rate did not change with time ($p=0.7383$) in this two-week period. For the 329 neurons significantly tunned to DG stimuli across all appearing days, they had a tuning curve similarity (correlation) of 0.7036 ± 0.0133 (mean+se) across 15 days. Their tunning is more ($p<0.0001$) like themselves across days than to that of other units within the same day 0.0064 ± 0.0003. Low dimensional UMAP representations of neuron population vector of 16 DG directions across 15 days (total: 240 patterns) from neuron population firing rate vectors formed a two-ring structure, represented DG directions are more like themselves across days than other directions within days ($p<0.0001$). A fixed weight independent decoder trained with 301 neurons using trials from first 7 days deciphered DG direction with mean absolute error 10.0013 ± 0.2372 degree on 15$^{th}$ day, far above chance (90 degrees). Similar results were obtained for other types of visual stimuli. Finally, with 1370 units trained on first 10 days, we were able to reconstruct 5100 natural images (played 1 or 60 trials/day) in the last 5 days with PSNR of 16.7 and image structural similarity of 0.7

**Conclusions**, stably tunned neurons and stable representations could be identified in mice visual cortex. We envision the capacity to reliably detect populations of the same neurons longitudinally using ultraflexible oversampling electrode arrays will advance our neuro-scientific understanding on how the brain balance between stability versus plasticity and catalyst robust neuroprosthetics design.

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