## Second Annual Rutgers Brain Health Institute Symposium

Friday, December 16th, 2016

The Edward Nash Theater
Raritan Valley Community College
Branchburg, NJ 08876

### Schedule

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.00 AM – 8.30 AM</td>
<td>Registration and Breakfast</td>
</tr>
<tr>
<td>8.30 AM – 9.00 AM</td>
<td><em>Dr. Gary Aston-Jones: BHI Overview</em></td>
</tr>
<tr>
<td>9.00 AM - 9.10 AM</td>
<td>&quot;Regulation of gene expression and mRNA isoform abundance in Down Syndrome patient-derived neurons&quot;</td>
</tr>
<tr>
<td>9.15 AM – 9.25 AM</td>
<td><em>Dr. John Pintar</em> &quot;A novel ligand receptor system for treatment of chronic stress”</td>
</tr>
<tr>
<td>9.30 AM – 9.40 AM</td>
<td><em>Dr. Vanessa Routh</em> &quot;The role of orexin in binge eating behavior and ventral tegmental glutamate plasticity”</td>
</tr>
<tr>
<td>9.45 AM – 9.55 AM</td>
<td><em>Dr. Rafael Benoliel</em> &quot;Use of DREADDs (designer receptor exclusively activated by designer drugs) for pain control”</td>
</tr>
<tr>
<td>10.00 AM – 10.10 AM</td>
<td><em>Dr. Joshua Berlin</em> &quot;A novel cellular approach to study acute hyperexcitability in TBI”</td>
</tr>
<tr>
<td>10.15 AM – 10.25 AM</td>
<td><em>Dr. Radek Dobrowolski</em> &quot;The amino acid metabolite homocysteine activates mTORC1 to inhibit autophagy and form abnormal proteins in human neurons and mice”</td>
</tr>
<tr>
<td>10.30 AM – 10.45 AM</td>
<td>Refreshment Break</td>
</tr>
<tr>
<td>10.45 AM – 10.55 AM</td>
<td><em>Dr. Tracy Tran</em> &quot;The role of Neuropilin 2 in excitatory versus inhibitory neuron development and contribution to Autism Spectrum Disorder”</td>
</tr>
<tr>
<td>Time</td>
<td>Session</td>
</tr>
<tr>
<td>--------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| 11.00 AM – 11.10 AM | Dr. David Margolis  
“Role of primary sensory cortex in behavioral inhibition”  
Dr. Brian Keane  
“Therapeutic effects of transcranial current stimulation in schizophrenia”  
Keynote: Dr. György Buzsáki, M.D., Ph.D., Biggs Professor of Neuroscience, NYU School of Medicine.  
“Emergence of cognition from action” |
| 11.30 AM – 12.30 PM | Buffet Lunch  
Matthew Scarnati (PhD student in Dr. Kenneth Paradiso’s lab)  
“Evidence for the presence of local protein synthesis at a presynaptic nerve terminal in the brain, and its requirement to maintain sustained synaptic transmission”  
Anna Giarratana (MD, PhD student in Dr. Janet Alder’s lab)  
“The Role of Genetic Polymorphisms in a Mouse Model of Traumatic Brain Injury and Personalized Treatment Approaches”  
Rutgers Neuroengineering group (RU-NEG) mini talks  
Dr. Joachim Kohn: “A newly designed nerve wrap as a central part of a comprehensive solution to long-gap, peripheral nerve repair.”  
Dr. Hilton Kaplan: “Engineering Autologous Human Vascularized Nerve Grafts from Decellularized Xenogeneic Neurovascular Bundles”  
Dr. Antonio Merolli: “Single cell tracking of the myelination of artificial fibers” |
| 3.00 PM – 4.30 PM | Post-doc and Student Poster Session  
4.30 PM – 5.30 PM | Wine & Cheese Reception and Best Poster Awards |
Mission Statement

The goal of the Brain Health Institute (BHI) is to develop neuroscience at Rutgers to become a highly translational and internationally preeminent research enterprise. New tools are transforming neuroscience, and these afford an unprecedented opportunity to create new treatments for central nervous system disorders. Neuroscience has been identified by Rutgers University as one of five signature areas for future focus and development. As part of this strategic plan, the BHI was established to become an internationally recognized center for basic, translational, and clinical research into the biological bases of human brain function and dysfunction. The BHI is the home for the overall Rutgers neuroscience initiative, and is a growing interdisciplinary institute consisting of more than 250 principal investigators with neuroscience laboratories across various campuses of Rutgers University and Rutgers Biomedical and Health Sciences. By supporting and coordinating neuroscience across all campuses, the BHI will unite Rutgers University’s dynamic and diverse neuroscience community toward common goals:

- To create research programs focused on the biological underpinnings of the central nervous system’s function and dysfunction.
- To develop treatments for these disorders using novel neuroscience tools.
- To establish a rich neuroscience resource in New Jersey that educates the public, clinicians, faculty, and students, as well as state, national, and international health officials.

BHI Strategic Plan

Initial focus for development of neuroscience via the BHI at Rutgers will be on four areas and associated disorders: Neurodevelopment, Neurodegeneration and Injury, Cognitive and Sensory Neuroscience, and Motivational and Affective Neuroscience. The selection of the focus areas was based on an analysis of strengths at Rutgers currently, as well as the recognition of prevalent nervous system disorders with a large need for novel treatments. A major goal will be to identify potential teams within these areas of focus, where targeted recruitments would have a significant impact on multi-investigator translational research.

A further area of focus for the Brain Health Institute will be to utilize new techniques in basic neuroscience to develop novel therapies for brain and spinal disorders. Over the past 7 years, developments in viral vector neurotransduction, optogenetics, and chemogenetics (designer receptors), among other areas, are revolutionizing neuroscience. These new methods have proven effective in altering brain function and dysfunction in highly specific ways in animal models, indicating that such methods may lead to a new generation of neurotherapeutics. Indeed, viral vectors are already being used in clinical trials to treat Alzheimer’s and Parkinson’s diseases by expressing growth factors to halt degeneration of neurons in the basal forebrain and midbrain. Similar viral vectors can be used to express opsins or designer receptors in a cell type-specific manner to allow control of selective populations of brain or spinal neurons with unprecedented specificity. This will allow new therapies, based upon knowledge from basic neuroscience research, with many fewer side effects compared to almost any current treatment. By studying different disorders in parallel, we can identify commonalities for the underpinnings of disease. The goal is to identify the genetic, environmental, and other aspects related to neuropathology and repair so that effective strategies can be developed for prevention and treatment.

**Director:** Gary Aston-Jones, PhD: [gsa35@ca.rutgers.edu](mailto:gsa35@ca.rutgers.edu)

**Associate Director:** Robin Davis, PhD: [Rldavis@dls.rutgers.edu](mailto:Rldavis@dls.rutgers.edu)

**Managing Director:** Eldo Kuzhikandathil, PhD: [kuzhikey@ca.rutgers.edu](mailto:kuzhikey@ca.rutgers.edu)

**Administrative Services Manager:** Lena Fullem, CRA: [fullem@ca.rutgers.edu](mailto:fullem@ca.rutgers.edu)

**Secretary:** Louise Petrone: [lp544@ca.rutgers.edu](mailto:lp544@ca.rutgers.edu)

Brain Health Institute
683 Hoes Lane West, Office 259A
Piscataway, NJ 08854
Phone: 732-235-4767; e-mail: [bhi@ca.rutgers.edu](mailto:bhi@ca.rutgers.edu); Web site: [https://brainhealthinstitute.rutgers.edu/](https://brainhealthinstitute.rutgers.edu/)
The Second Annual Rutgers Brain Health Institute Symposium
SPEAKER ABSTRACTS
“Emergence of cognition from action”

A fundamental function of the brain is to predict the future. More complex brains evolved multiple hierarchical loops between their outputs and inputs to make prediction more reliable in more complex environments and at longer time scales. With extensive training these prediction mechanisms have become internalized. At the center of this model are self-propagating loops of neuronal coalitions connected by modifiable synapses that can be propelled forward without external cues. The implication of this conjecture is that brain networks are endowed with internal mechanisms that can generate a perpetually changing neuronal activity even in the absence of environmental inputs. I will discuss examples and mechanisms of this framework.

Dr. Buzsáki is the Biggs Professor of Neuroscience at NYU School of Medicine. He was previously a Board of Governors Professor at the Center for Molecular and Behavioral Neuroscience, Rutgers University, Newark. Dr. Buzsáki has made major contributions to the understanding of neuronal oscillations focusing on their relationships to each other, to behavior and to neuronal action potential generation and information coding in the brain. He is the author of Rhythms of the Brain, a book detailing the current neuroscientific understanding of brain rhythms, and of more than 300 peer reviewed papers. He is distinguished as a "highly cited researcher" by Thomson Reuters. He was the winner of the inaugural Grete Lundbeck European Brain Research Prize in 2011 together with Tamas Freund and Peter Somogyi for their work describing organization of neurons in the hippocampus and the cortex. Dr. Buzsáki is an elected Fellow of the American Association for the Advancement of Science and a member of the Hungarian Academy of Sciences, and he sits on the editorial boards of several leading neuroscience journals, including Science and Neuron. He has won numerous awards and given several named lectures.

Dr. Buzsáki research focuses on the working hypothesis that in brain networks, especially those serving cognitive functions, the packaging and segmentation of neural information is supported by the numerous self-organized rhythms the brain generates. His lab focuses largely on the generation of various brain oscillations, their spatial and temporal relationships, and the role of inhibition in the enforcement of syntactic rules.
Bin Tian, PhD
Professor
Department of Microbiology, Biochemistry and Molecular Genetics
Rutgers-New Jersey Medical School

Regulation of gene expression and mRNA isoform abundance in Down Syndrome patient-derived neurons

Aysegul Guvenek¹, Heather Mcgowan², Dinghai Zheng¹, Zhiping Pang², Bin Tian¹
¹Department of Microbiology, Biochemistry and Molecular Genetics, Rutgers New Jersey Medical School, Newark, NJ
²Department of Neuroscience and Cell Biology, Rutgers University Robert Wood Johnson Medical School, New Brunswick, NJ

Down syndrome (DS) is a genetic disorder caused by an extra copy of chromosome 21, resulting in intellectual disability along with other pathological features. Several studies have reported widespread gene regulation in DS cells. However, how mRNA isoforms are regulated in DS has never been examined. Here using 3’ region extraction and deep sequencing (3’READS), our recently developed method to examine gene expression and alternative polyadenylation (APA) isoforms, we analyzed RNAs from normal and DS patient-derived neurons. These human neurons were generated using induced neuronal (iN) cell technology by viral mediated expression of Neurognin2. Our analysis identified over 1,000 genes with differential expression in DS neurons. Consistent with trisomy of the disease, genes on chromosome 21 are globally upregulated. However, 93% of regulated genes are from other chromosomes, indicating significant impact of genes from chromosome 21 on other genes. Gene Ontology analysis identified several key pathways that are affected in DS neurons. Interestingly, we also identified over 1,000 genes that have alternative polyadenylation (APA) isoform changes across different chromosomes. APA is a widespread mechanism in mammalian genes, which regulates the 3’ untranslated (3’UTR) region or coding region. Since the 3’UTR contains regulatory elements for RNA metabolism, such as stability, localization and translation, APA plays an important in gene expression. The potential significance of APA isoform regulation in DS neurons will be discussed.

John Pintar, PhD
Professor
Department of Neuroscience and Cell Biology
Rutgers-Robert Wood Johnson Medical School

A novel ligand receptor system for treatment of chronic stress

John Pintar, Dept. of Neuroscience and Cell Biology, Rutgers-RBHS-RWJMS, and Alex Kusnecov, Dept. of Psychology, Rutgers New Brunswick

The ORL-1/OFQ receptor ligand system is the most recently described component of the endogenous opioid system and our previous work has shown its importance in a variety of behaviors including modulating analgesic responses and feeding behavior. We have obtained preliminary evidence from gene-targeted mice produced in house that the orphanin FQ peptide, one of the opioid family of peptides, limits detrimental cytokine responses that accompany chronic stress. Thus agonists for this peptide may represent novel treatments for this condition. Here we will present emerging data to support this hypothesis. First we will present data indicating that increased IL-1β levels accompany OFQ deletion in C57Bl6/J OFQ male mice when subjected to chronic unpredictable stress and present progress toward investigating a potential cellular basis for this response by producing mice with microglia labeled with eGFP. We will also present preliminary results from mice exposed to unpredictable stress and then used for a focused sequential battery of behavioral tests to evaluate anxiety. Extension of these experiments are expected to provide critical information regarding the role of the OFQ system in response to chronic stress and to identify the most promising approaches for future grant applications when additional cytokines, microglia responses, and behaviors can be evaluated in conjunction with OFQ system agonists and antagonists.
Vanessa Routh, PhD  
Professor  
Department of Pharmacology, Physiology & Neuroscience  
Rutgers-New Jersey Medical School

*The role of orexin in binge eating behavior and ventral tegmental glutamate plasticity*

**Vanessa Routh**, Pharmacology, Physiology & Neuroscience, Rutgers-NJMS,  
**Nicholas T. Bello**, Animal Sciences, Rutgers-SEBS

The prevalence of eating disorders in the United States population is estimated to be 4%. Effective long-term treatment for eating disorders involves cognitive behavioral strategies. However, the 5-year recovery rate for bulimia nervosa (BN) is only about 50% for most clinics and binge eating disorder (BED) is a major obstacle in the treatment of obesity. Although BN and BED are different psychiatric disorders, repeated episodes of prolonged calorie restriction (i.e. bouts of fasting) for BN or intermittent dieting for BED has been associated with the entrainment of binge eating. The goal of our project is to specifically determine how repeated intermittent caloric restriction contributes to the sustaining neurobiology that promotes the binge eating of sugary fat pleasurable foods. Our focus is the interaction between the lateral hypothalamic area (LHA) orexin glucose-inhibited (GI) neurons and the ventral tegmental area (VTA) dopamine neurons. Ventral tegmental area (VTA) dopamine neurons play a key role in reward-based feeding. The LHA orexin GI neurons enhance excitatory glutamate input to the VTA dopamine neurons and may be an important link between caloric restriction and binge eating. We have recently shown that fasting reduces the inhibitory effect of glucose on LHA orexin-GI neurons leading to increased activation in low glucose. Our preliminary data suggest that low glucose alters glutamate synaptic plasticity on VTA dopamine neurons in an orexin-dependent manner. Moreover, LHA glucose dialysis is negatively correlated with reward-based feeding. This project tests the hypothesis that intermittent caloric restriction alters the activity and glucose sensitivity of LHA orexin-GI neurons leading to persistent enhancement of glutamate transmission onto VTA DA neurons. This enhanced glutamate transmission then contributes to the development of binge eating.

Rafael Benoliel, BDS  
Professor, Associate Dean for Research  
Department of Diagnostic Sciences  
Rutgers-School of Dental Medicine

*Use of DREADDs for pain control*

**Rafael Benoliel**, Koczeniewska, O., Diagnostic Sciences, Rutgers-SDM,  
**Gary Aston-Jones**, Morgan J., Brain Health Institute

We hypothesized that activation of hM4Di-DREADDs transfected into trigeminal ganglion neurons will reduce behavioral and molecular markers of pain in recognized rat models of acute and chronic trigeminal neuropathic pain. Employing a stereotactic approach we successfully injected and transfected neurons in the trigeminal ganglion (TG) with the inhibitory hM4Di DREADD. Using immunocytochemistry we observed widespread transfection of hM4Di receptors across all neurons within the TG. Additionally, hM4Di receptors are transported to proximal fibers of TG neurons, which presents the transformative possibility of stimulating hM4Di receptors in the terminal branches of the trigeminal nerve on the face. Our behavioral experiments, carried out in over 60 animals, indicate that activation of hM4Di receptors expressed in TG neurons by the DREADD agonist clozapine N-oxide (CNO) is a highly effective method to reduce behavioral markers of pain in the orofacial formalin test. CNO-induced activation of hM4Di receptors expressed in TG neurons reduced facial grooming following injection of formalin into the rat vibrissal pad. A similar trend was observed across both sexes, but was more pronounced in female rats. We are now testing the effects of this approach on chronic trigeminal neuropathic pain and examining the use of alternative promoters such as the TAC1 promoter which control expression of tachykinin peptides and is specifically expressed in Substance P neuronal subpopulations in the TG.
A novel cellular approach to study acute hyperexcitability in traumatic brain injury

Joshua R Berlin\textsuperscript{1}, Dino Magou\textsuperscript{2}, and Bryan J Pfister\textsuperscript{2}, \textsuperscript{1}Pharmacology, Physiology & Neuroscience, Rutgers-NJMS, \textsuperscript{2}Biomedical Engineering, NJIT.

The basis for acute seizures following traumatic brain injury (TBI) remains unclear. To study the cellular mechanisms initiating acute seizure-like activity after injury, we measure spontaneous electrical activity in cultured neocortical neurons subjected to a localized uni-axial stretch using whole-cell current clamp techniques. This \textit{in vitro} injury model yields a region of stretch injured neurons and adjacent regions of non-stretched neurons that do not directly experience stretch injury. Interestingly, 30-60 minutes after a stretch maneuver, non-stretched neurons display a marked increase in spontaneous action potential firing and bursting activity, while stretch injured neurons display dramatically lower numbers of action potentials. These results demonstrate that acute hyperexcitability can be observed in non-stretched neurons located in regions adjacent to the site of stretch injury, consistent with reports that seizure activity can arise from regions surrounding the site of localized brain injury. To understand, how hyperexcitability arises in these non-stretched neurons, we are developing an optical system to measure spontaneous electrical activity in populations of cultured neurons. This system utilizes recent advances in genetically-encoded voltage indicators that allow spontaneous action potentials to be measured in populations of neurons so that we can study network properties prior to and after localized stretch injury. We plan to use pharmacological and immunohistological tools to identify what types of neurons show stretch-induced alterations in electrical activity and then use network analysis tools to identify mechanisms underlying alterations in spontaneous activity.

Homocysteine activates mTORC1 to inhibit autophagy and form abnormal proteins in human neurons and mice.

Khoosheh Khatayi, K.\textsuperscript{1}, Antikainen, H.\textsuperscript{1}, Bonder, E.M.\textsuperscript{1}, Weber, G.F.\textsuperscript{1}, Kruger, W.D.\textsuperscript{2}, Hieronim Jakubowski\textsuperscript{3}, and Radek Dobrowolski\textsuperscript{1}, \textsuperscript{1}Rutgers-Newark; \textsuperscript{2}Fox Chase Cancer Center, Philadelphia; \textsuperscript{3}Rutgers-New Jersey Medical School

The molecular mechanisms leading to and responsible for age-related, sporadic Alzheimer’s disease (AD) remain largely unknown. It is well-documented that aging patients with elevated levels of the amino acid metabolite Homocysteine (Hcy) are at high risk of developing AD. We investigated the impact of Hcy on molecular clearance pathways in mammalian cells including \textit{in vitro} cultured iPSC-derived forebrain neurons and \textit{in vivo} neurons in mouse brains. Exposure to Hcy resulted in up-regulation of the mechanistic target of Rapamycin complex 1 (mTORC1), one of the major kinases in cells which is tightly linked to anabolic and catabolic pathways. Hcy is sensed by a constitutive protein complex composed of leucyl-tRNA-synthetase and Folliculin which regulates mTOR tethering to lysosomal membranes. In hyper-homocysteinemic human cells and cystathionine beta-synthase (Cbs)-deficient mouse brains, we find an acute and chronic inhibition of the molecular clearance of protein products resulting in a buildup of abnormal proteins including beta-Amyloid and phospho-Tau. Formation of these protein aggregates lead to AD-like neurodegeneration this pathology can be prevented by inhibition of mTORC1 or by induction of autophagy. We conclude that an increase of intracellular Hcy levels predisposes neurons to develop abnormal protein aggregates that are hallmarks of AD and its associated onset and pathophysiology with age.
The proper development of neuronal populations and dendritic morphologies are critical for patterning of synaptic connections and transmission, which affect circuit activity, and ultimately impact behavior and mental function. Many developmental disorders, including but not limited to Autism Spectrum Disorder (ASD) and childhood epilepsies prominently feature aberrant dendritic morphology and abnormal circuit function. However, the relationship between changes in dendritic morphology and resulting alterations in neural circuit activity and behavior is unclear. Previously, we demonstrated that the obligated binding receptor for the secreted ligand semaphorin 3F (Sema3F), Neuropilin-2 (Nrp2), is a key player in regulating dendritic spine number, size and distribution in layer 5 cortical neurons and granule neurons of the hippocampus. The Nrp2 is uniquely enriched in the apical dendritic compartment of cortical neurons in vitro and in vivo, and can serve to restrain excess spines and excitatory synapse development in pyramidal cortical and granule hippocampal neurons. The loss of Nrp2 enhanced excitatory synaptic transmission in both cortical and hippocampal neurons. Additionally, we conducted the first behavioral study of mice harboring a mutation of the Nrp2 gene, focusing on behaviors known to depend on cortical and hippocampal circuitry. Nrp2-/- mutants showed ASD-associated behavior impairments in object recognition memory, no preference for social novelty, and increased grooming behavior compared to age-matched controls. Interestingly, Sema3F-Nrp2 signaling is also linked to seizures. The high co-occurrence of epileptic seizures in ASD (30%) and ASD with epilepsy (>30%) suggests shared developmental pathological mechanisms, and Sema3F-Nrp2 may be the converging signaling pathway. Nrp2 is also required for cortical and hippocampal inhibitory neuron development. Furthermore, epilepsy-autism syndromes are proposed to result from compromised excitation-inhibition balance leading to changes in neuronal circuit connections and functions. Therefore, the main goal of our research program is to test the fundamental premise that deficits in the Nrp2 mutant to properly establish specific excitatory and inhibitory neuronal populations and their circuits during development underlie the shared outcome of autism and epilepsy. Using the conditional flox Nrp2 mouse crossed to an inducible Cre line driven by either CAMK2a or Nkx2.1 promoter, we will specifically delete Nrp2 in excitatory or inhibitory neuron progenitors, respectively, at early embryonic versus postnatal time points and analyze neuronal numbers, morphology, physiology and behaviors associated with ASD. Our ongoing study will directly examine the development of epilepsy and autistic behaviors in models with selective excitatory or inhibitory neuron deletion of Nrp2 to resolve the principal trigger for circuit changes underlying epilepsy and autism.
**Role of primary sensory cortex in behavioral inhibition.**

Lee, C.R.¹, James M. Tepper², David J. Margolis¹; ¹Cell Biology and Neuroscience, Rutgers–Piscataway; ²CMBN, Rutgers–Newark

The primary somatosensory cortex (S1) has an increasingly appreciated role in sensorimotor behavior and motor control. While signaling from prefrontal cortex to striatum is largely responsible for behavioral response inhibition, other cortical areas including S1 provide massive projections to the striatum that could play important functional roles, especially during specific behavioral contexts. We tested the hypothesis that S1 is involved in sensory-driven behavioral response inhibition through differential connectivity with identified cell populations of the dorsal neostriatum (DStr). We first tested whether optogenetic activation of S1 projections to DStr induces behavioral response inhibition in mice performing a tactile decision-making task. Preliminary data indicate that channelrhodopsin-2 (ChR2) stimulation decreases the occurrence of trials with responses (Go and False Alarm) and increases the occurrence of trials with no response (NoGo and Miss), consistent with an increase in response inhibition. Second, we used ex vivo patch clamp recordings to determine potential corticostriatal circuit mechanisms underlying this effect. Specifically, we measured excitatory postsynaptic potentials (EPSPs) from S1 to both medium spiny projection neurons and fast-spiking (PV+) interneurons in DStr, and compared results with inputs from primary motor cortex (M1). We found that optogenetically-activated S1-DStr inputs induce an approximately 7-fold larger EPSP in fast-spiking interneurons than in medium spiny projection neurons. In contrast, M1-DStr projections induce an EPSP that is of equal magnitude in fast spiking interneurons and medium spiny neurons. Preliminary behavioral experiments in mice expressing ChR2 in fast spiking interneurons reveal an increase in response inhibition when fast spiking interneurons are stimulated directly, similar to the effect seen with activation of S1 input to DStr. Together, our optogenetic and electrophysiological data support the idea that S1-DStr signaling could play an important role in behavioral response inhibition via biased activation of fast-spiking interneurons.

**Therapeutic effects of transcranial current stimulation in schizophrenia**

Brian P. Keane¹, and Bart Krekelberg²; ¹Rutgers-RWJMS; ²CMBN, Rutgers-Newark

Schizophrenia (Sz) is a severe psychiatric disorder defined by delusions, hallucinations, disorganized thought, and functional decline, among other features. Cognitive and perceptual deficits also strongly characterize the illness; these can be functionally debilitating and subjectively unnerving, but they rarely respond to antipsychotic treatment. Our long term goal is to develop transcranial current stimulation (TCS) as a novel, non-invasive therapy for impaired visual cognition in Sz. Our study aims to combine high-density EEG recordings with high-density TCS to boost neural dynamics that are typically deficient in Sz and to restore normal performance on visual cognitive tasks in Sz. Our initial emphasis has been on bringing together three lines of research. In the first, we established that TCS modulates neural activity in a nonhuman primate model and used the model to explore the wide parameter space of TCS. We have found that TCS can reduce spike frequency adaptation and boost neural synchrony. In the second, we are developing the technical capability to record and stimulate the human brain transcranially with high density electrode grids. In the third, we are probing deficits in visual cognition and their neural correlates in patients with Sz and have identified a specific visual cognitive task (contour integration) that is reliably impaired in Sz. Merging these lines of research into a multidisciplinary approach allows us to build on a novel understanding of basic neural mechanisms to develop new therapeutic approaches for a major psychiatric disease.
**Objective:** Peripheral nerve regeneration across large gaps of > 5 cm rarely results in full functional recovery. Current therapies, including autografts, cannot address the three significant hurdles to full recovery: First, improved, kink- and crush-resistant nerve wraps have to be available to protect the newly growing nerve. Second, some biological guidance is needed to accelerate the growth of axons through the zone of injury and toward their ultimate target. Finally, atrophy of the target muscle needs to be prevented while the newly grown axons slowly grow towards their target. We are developing a comprehensive solution that can address these three hurdles simultaneously.

**Methods:** The comprehensive solution consists of a newly designed, kink- and crush-resistant nerve wrap, tissue engineered nerve grafts (TENGs) that support accelerated axon growth across the zone of nerve damage, and an electrical stimulation system that will reduce the rate of atrophy of the distal muscle targets while axons slowly extend towards their ultimate target.

**Results:** In this talk, preliminary results will be described for the kink- and crush-resistant nerve wrap. Made of a newly designed, fully degradable polymer, this wrap adsorbs endogenous proteins such as laminin, fibronectin, and collagen, thereby creating a neurotrophic environment that supports axon growth across the nerve gap. In vitro results are supported by extensive in vivo data, showing excellent functional recovery in short nerve gaps (1 cm) in rat and mouse models. Recent studies have shown that TENGs can be incorporated into these wraps, allowing nerve regeneration over 5 cm gaps in a clinically useful porcine model. In the final step of our research, we will treat long nerve gaps simultaneously with the wrapped TENGs and the electrical stimulation system.

**Summary:** If successful, the proposed comprehensive solution to peripheral nerve repair will be a game changing, new therapy for millions of patients suffering from peripheral nerve injuries.
Hilton Kaplan MBCh, FCSSA, PhD
Associate Director, New Jersey Center for Biomaterials
Research Associate Professor,
Rutgers, The State University of New Jersey.
Piscataway

Engineering autologous human vascularized nerve grafts from decellularized xenogeneic neurovascular bundles

Objectives: Autografts remain the "gold-standard" for treating peripheral nerve gaps, despite poor functional outcomes. These grafts remain viable by longitudinal inosculation and revascularization from the surrounding tissue bed. However, large-caliber (>2-3 mm diameter) and long length (>10 cm) grafts are at risk for central necrosis due to ischemia, if not vascularized. Vascularized nerve grafts have been used successfully for bridging scarred wound beds and minimizing central ischemia. Two factors limit their use: Donor site morbidity (for autografts); and risks of immunosuppression/rejection (for allografts). To overcome these, we propose novel xenograft decellularized neurovascular bundles (NVBs) that can be recellularized with recipient human endothelial cells (ECs) and Schwann cells (SCs) to create "autologous" vascularized nerves.

Methods: Femoral NVBs have been harvested from rat (2.0 cm), rabbit (3.5 cm), and pig (7 cm); and brachial NVBs have been harvested from non-human primates (10.0 cm). Tissues were decellularized (SDS, Triton-X) and characterized (histology, immunohistochemistry, PicoGreen® DNA assay, collagenase assay, and differential scanning calorimetry (DSC)). Samples were recellularized with human ECs and SCs, for 5 days in a bioreactor.

Results: Regenerated NVBs demonstrated vessels with intact human vascular endothelium, and nerves with viable human SCs. Specimens are currently being prepared for in vivo re-implantation in a nude rat model.

Significance: We have developed techniques for harvesting and decellularizing xenogeneic NVBs, as scaffolds for the successful regeneration of vascularized human nerve grafts. These novel "autologous" off-the-shelf NVBs will play an important role as free vascularized nerve grafts, while avoiding donor site morbidity and immunosuppression.
Single cell tracking of the myelination of artificial fibers

**Objectives:** *In vitro* models currently used to study myelination on artificial fibers are limited to cell population studies and have difficulty dissecting individual contributions to the process. We established a novel model that does not include axons or neuronal factors. Its single wire construct allowed us to create a map to locate every cell precisely and track individual cells day-by-day.

**Methods:** Our model uses a single carbon fiber, suspended in culture media, to provide an elongated structure of defined diameter with 360-degree of surface available for Human Schwann cells to wrap around. Microscopic observation was performed by phase-contrast and fluorescence analysis once a day for a period of nine days. After fixation, scaffolds were incubated with anti-MAG antibody and anti-MBP antibody.

**Results:** Cell elongation along the wire became obvious starting on day 2. We also observed cells, which apparently wrapped around the fiber completely and changed morphology during the following days, exhibiting outtolding (blebbing) in a timely fashion. The expression of MBP and MAG was evaluated at Day 9 and distinct MBP or MAG staining was observed along the fiber.

**Conclusions:** We observed cell attachment, elongation and enwrapping over a period of 9 days. Cells are alive and express MBP and MAG as expected. Natural/artificial molecules and external physical factors may be tested as possible regulators of myelination by this new model. The possibility to study myelination by Oligodendrocytes with this model is going to be evaluated.
POSTER ABSTRACTS
**Poster #1**

Strain Differences in Sensitivity to Cuprizone Induced Demyelination

**Authors**
Qili Yu, Ryan Hui, Alexander Kusnecov, Cheryl F. Dreyfus, and Renping Zhou

**PI Name:** Renping Zhou

Multiple Sclerosis (MS) is a severe neurological disorder caused by demyelination of the central nervous system (CNS) and affects about 2.5 million people worldwide. However, the molecular mechanisms underlying the pathogenesis of the disease remain unclear. We investigated the role of genetic differences in contributing to demyelination by using the cuprizone toxicity model with mice of different genetic background (CD1 and C57BL/6). We demonstrate using black gold staining and luxol fast blue (LFB) staining that exposure to diet containing 0.2% cuprizone treatment resulted in less severe demyelination in CD1 mice than C57BL/6 mice. With continuous cuprizone administration, demyelination in CD1 mice was not prominent until after 7 weeks of treatment, in contrast to C57BL/6 mice, in which demyelination was already prominent at week 4 of exposure. Concomitantly, immunohistochemical analysis of the cuprizone treated brain sections of the corpus callosum overlying the fimbria fornix demonstrated significantly more GST-pi+ oligodendrocytes in the CD1 mice relative to C57BL/6 mice. Moreover, CD1 mice exhibit fewer GFAP+ astrocytes and Iba1+ microglia. In order to rule out the issue of potential difference in diet consumption between strains, we measured food intake per body weight between CD1 mice and C57BL/6 mice and report no significant differences. Thus, genetic background factors appear to influence the susceptibility to cuprizone induced demyelination, and our findings might provide new insight into the detailed mechanism of the demyelination process.

Research partially supported by 2PO1HD023315 (RZ), RO1EY019012 to RZ, P30ES005022

**Poster #2**

Dysregulation of Protein Phosphatase 2A in Dementia with Lewy Bodies

**Authors**
Hye-Jin Park, Kang-Woo Lee, Eun S. Park, Stephanie Oh, Run Yan, Jie Zhang, M. Maral Mouradian

**Center for Neurodegenerative and Neuroimmunologic Diseases, Department of Neurology, Rutgers Robert Wood Johnson Medical School**

**PI Name:** M. Maral Mouradian

Protein phosphatase 2A (PP2A) functions as a master regulator of cellular phosphoregulatory networks in the brain, controlling key processes required for protein homeostasis and cell survival. The heterotrimeric holoenzyme is composed of a highly conserved catalytic C subunit, a scaffold-like A subunit, and one of several regulatory B subunits that confer substrate specificity. The assembly and activity of PP2A is regulated by reversible carboxyl methylation of the C subunit. α-Synuclein, which misfolds and aggregates in hallmark pathologic lesions of Dementia with Lewy Bodies (DLB), is phosphorylated at Serine 129, and PP2A containing B55α subunit is a major phospho-Ser129 phosphatase. To investigate the possibility that PP2A dysregulation could play a role in the pathogenesis of DLB, we have compared the state of PP2A methylation, as well as the expression of its methylaing enzyme, leucine carboxyl methyltransferase (LCMT-1), and demethylating enzyme, protein phosphatase methylesterase (PME-1), in postmortem brain tissues from 8 DLB cases and an equal number of age-matched non-neurological controls. Immunohistochemical studies showed that LCMT-1 is significantly reduced in both frontal cortex and substantia nigra of DLB cases compared to Controls, while PME-1 is not significantly altered. This is associated with a marked reduction in the ratio of methylated PP2A to demethylated PP2A in DLB. These findings support the hypothesis that PP2A dysregulation in α-synucleinopathies may contribute to the accumulation of hyperphosphorylated α-synuclein and to the disease process, raising the possibility that pharmacological means to enhance PP2A phosphatase activity may be a useful disease modifying therapeutic approach.

This research was supported by a grant from the Michael J. Fox Foundation for Parkinson’s Research.
**Poster #3**

MicroRNA-7 regulates the function of mitochondrial permeability transition pore by targeting VDAC1

**Authors**
Myungsik Yoo, Amrita Datta Chaudhuri, Doo Chul Choi, Savan Kabaria, Alan Tran and Eunsung Junn

**PI Name:** Eunsung Junn

Mitochondrial dysfunction is one of the major contributors to the neurodegenerative disorders including Parkinson’s disease (PD). The mitochondrial permeability transition pore (PTP) is a protein complex located on the mitochondrial membrane. Under cellular stress, the pore opens, increasing the release of pro-apoptotic proteins, and ultimately resulting in cell death. MicroRNA-7 (miR-7) is a small non-coding RNA that has been found to exhibit a protective role in the cellular models of PD. In the present study, miR-7 was predicted to regulate the function of mitochondria, according to gene ontology (GO) analysis of proteins that are downregulated by miR-7. Indeed, miR-7 overexpression inhibited mitochondrial fragmentation, mitochondrial depolarization, cytochrome c release, reactive oxygen species (ROS) generation and release of mitochondrial calcium in response to 1-methyl-4-phenylpyridinium (MPP+) in human neuroblastoma SH-SY5Y cells. In addition, several of these findings were confirmed in mouse primary neurons. Among the mitochondrial proteins identified by GO analysis, the expression of voltage dependent anion channel 1 (VDAC1), a constituent of the mitochondrial PTP, was downregulated by miR-7 through targeting 3'-untranslated region of VDAC1 mRNA. Similar to miR-7 overexpression, knockdown of VDAC1 also led to a decrease in intracellular ROS generation and subsequent cellular protection against MPP+. Notably, overexpression of VDAC1 without the 3'-UTR significantly abolished the protective effects of miR-7 against MPP+-induced cytotoxicity and mitochondrial dysfunction, suggesting that the protective effect of miR-7 is partly exerted through promoting mitochondrial function by targeting VDAC1 expression. These findings point to a novel mechanism by which miR-7 accomplishes neuroprotection by improving mitochondrial health.

This work was supported by National Institutes of Health Grant (NIH) NS070898 to E.J.

**Poster #4**

Basolateral amygdala nucleus responses to appetitive conditioned stimuli correlate with variations in conditioned behavior

Seung-Chan Lee, Alon Amir, Drew B. Headley, Darrell Haufler & Denis Pare

*Center for Molecular and Behavioral Neuroscience, Rutgers, The State University of New Jersey, Newark, NJ 07650*

**PI Name:** Denis Pare

In the lateral amygdala (LA), training-induced increases in neuronal responsiveness to conditioned stimuli (CSs) reflect potentiated sensory responses that drive conditioned behaviours (CRs) via LA’s targets. The basolateral nucleus of the amygdala (BL) receives LA inputs and projects to various subcortical sites that can drive aversive and appetitive CRs. Consistent with this, BL neurons also develop increased responses to CSs that predict rewarding or aversive outcomes. This increased BL activity is thought to reflect the potentiated sensory responses of LA neurons. Here we contrast the CS-related activity of BL neurons when rats produced the expected CR or not, to show that cells activated by appetitive CSs mainly encode behavioural output, not CS identity. The strong dependence of BL activity on behaviour irrespective of CS identity suggests that feedforward connectivity from LA to BL can be overridden by other BL inputs.
**Poster #5**

The Role of Genetic Polymorphisms in a Mouse Model of Traumatic Brain Injury and Personalized Treatment Approaches

Anna Giarratana, Lauren Fish, Rene Schloss, Smita Thakker-Varia, Martin Yarmush, Janet Alder

**PI Name:** Janet Alder

Traumatic Brain Injury (TBI) is a serious and potentially life threatening clinical problem. Clinicians have long noticed that certain patients recover better after TBI, and identifying what makes some patients more susceptible is a vital step in understanding the underlying mechanisms through which TBI causes its deleterious effects. The goal of this study was to determine the effect of specific single nucleotide polymorphisms (SNPs) which may lend insight into whether individuals with these genetic alleles might be at higher risk than the general population for poor recovery following TBI and to explore approaches to treating them. We have investigated behavioral and cellular outcomes in genetically engineered mice with the ApoE4 and BDNF Val66Val polymorphisms following repeated, mild TBI. We have found that ApoE4 and Val66Met mice trend towards having a larger injury volume as assessed by MRI and increased levels of neurodegeneration, apoptosis, and gliosis compared to ApoE3 and Val66Val mice. We have also begun to identify a personalized approach to treating genetically susceptible individuals by targeting the pathway altered in those genotypes. Human mesenchymal stromal cells have been shown to secrete neurotrophins such as BDNF. We have utilized different approaches such as encapsulation and pre-treatment with different factors in order to increase their therapeutic efficacy. We have found that encapsulation and pretreatment of the MSCs with forskolin may help skew their secretome towards a more positive one. This study lays the groundwork for further investigation into the genetics that play a role in recovery after TBI and potential therapeutics.

Fellowship from the New Jersey Commission on Brain Injury Repair

**Poster #6**

CHARIS - Cerebral Hemodynamic Autoregulatory Information System

Bianca Pineda, Maria Qadri, Colin Kosinski, Nam Kim, William Craelius

**PI Name:** William Craelius

Acute brain injury (ABI) is a devastating event requiring intensive acute treatment and post-injury rehabilitation, both delivered for indeterminate periods of time. For severe ABIs, acute treatment is aimed at stabilizing the patient to prevent secondary brain injury from ischemia and swelling. This requires a balance between adequate levels of cerebral blood flow and safely low intracranial pressure (ICP) - a task normally done by autoregulatory (AR) processes of the brain. In the absence of normal AR, hemodynamic stability is difficult to maintain because there are no reliable predictors to guide treatment. Thus, guidelines for triage and discharge are somewhat arbitrary for ABI and the need for a clinical decision support system (CDSS) for neurotrauma is widely recognized. Prerequisite to a CDSS is a large database of patient records and efficient means to extract meaningful information from them, neither of which are available. These requirements are being addressed by consortia such as MIMIC III in the U.S. and BrainIT in Europe, but more effort is needed. Our contribution, CHARIS, will systematize the analysis of relevant physiological signals, and will embody data-driven algorithms to search for potential predictors of acute clinical events. Specifically, we will produce: (1) a processing system for organizing, identifying and preconditioning raw data prior to analysis, (2) a process to discriminate clinically relevant events from artifacts in physiological recordings, and (3) algorithms for efficient computation of selected AR parameters.

New Jersey Commission on Brain Injury Research
**Poster #7**
Low Dose Ethanol Selectively Excites Raphe- and VTA- Projecting Lateral Habenula Neurons in the Rat

Rao Fu, Wanhong Zuo, Jing Li, Haifeng Zhang, Seungwoo Kang, Jiang-Hong Ye

**PI Name:** Jiang-Hong Ye

It is unclear how ethanol affects the brain that eventually leads to addiction in susceptible subjects. Recent work from our laboratory has shown that many neurons in the lateral habenula (LHb) are activated by ethanol with an EC50 of ~1 mM. The LHb is a highly heterogenetic complex, consists of many subregions. The LHb receives afferents from and sends efferents to many different brain regions. The current study in adult male Sprague-Dawley rats sought to identify the LHb subregions and circuitries that are activated by low dose ethanol. Systemic administration of ethanol (0.25g/kg, I.P.), resulting in a peak blood alcohol concentration of 5.6 mM, significantly increased the number of c-Fos-immunoreactive (IR) neurons in the parvocellular part and central part of median division of the LHb (LHbMPC and LHbMC). Further c-Fos experiments in conjunction with retrograde tracing approach showed that ethanol-activated LHbMPC and LHbMC neurons preferentially project to the median raphe, dorsal raphe and the VTA, but less to the rostromedial tegmental nucleus. Finally, injections of the anterograde tracer AAV-GFP in the lateral hypothalamus lead to numerous anterogradely labeled fibers with bouton-like structures impinging on LHb c-Fos-IR neurons that project to dorsal raphe and were activated by ethanol. These data indicate that low dose ethanol selectively excites the raphe- and VTA-projecting neurons in the medial part of the LHb, and that the LHb-raphe projecting neurons that are activated by ethanol receive projections from the lateral hypothalamus.

Funded by NIH grant AA022292

**Poster #8**
Investigating the dichotomy of accumbens core and shell in response to food-predictive cues

Daniel Quintin, Alexei Taylor, Joshua Stamos, Anthony Pawlak, Mark West

**PI Name:** Mark West

Neuropsychobiological processes influence abnormal eating-related behaviors such as binge eating. Particularly, hyperstimulation of the nucleus accumbens (NAc) may play a role in affecting such behaviors. The inherently multifactorial nature of hedonic pathologies represents a challenge in identifying the etiology and developing treatments. The purpose of this study is to 1) examine changes in NAc firing in relation to cue processing, 2) elucidate how changes in NAc processing as influenced by an exteroceptive cue in animals with a history of binge eating reflect the feed-forward organization of the striatonigrostriatal pathway and 3) investigate how rewards and predictive cues may prompt and/or modify aberrant behaviors. Naïve, young adult, female rats underwent a 6-week pretreatment of twice-per-week bingeing on sweetened fat in comparison to chow controls. All rats then underwent stereotaxic surgery and had 16 microwires implanted directly into core and shell subregions of the NAc. Following recovery, subjects individually were trained in a 10-day Pavlovian task with a conditioned stimulus CS paired with a 32% sucrose reward. All rats learned the task and exhibited positive affect as indicated by elevated 50 kHz ultrasonic vocalizations. Collectively, NAc neurons acquired responsiveness to the tone CS. Importantly, shell neurons but not core neurons exhibited greater responsiveness in BE than CC animals. These findings are consistent with anatomical data indicating that limbic signals regarding biologically relevant cues initially affect NAc shell firing, and moreover, provide the first evidence of its kind that such firing is altered by a history of binge eating.

Supported by Rutgers Aresty Undergraduate Fellowship
**Poster #9**

Gut-dysbiosis breaks the immune tolerance in MBP-specific T cells

Sudhir K. Yadav, Naoko Ito, John E. Mindur, Martin Mathay, Amale Laouar, Suhayl Dhib-Jalbut

**PI Name:** Kouichi Ito

Multiple sclerosis (MS) is an immune mediated disease targeting the myelin protein in the central nervous system (CNS). It is proposed that breakage of immune tolerance in myelin protein -specific autoreactive T cells initiates the CNS autoimmunity. Interestingly, recent studies indicated gut dysbiosis as one of the important risk factors for breakage of immune tolerance and development of MS; however, the mechanism remains elusive.

In this study, we investigated the role of gut dysbiosis in breaking peripheral immune tolerance and the development of CNS autoimmunity, using spontaneous experimental autoimmune encephalomyelitis (EAE) transgenic mice model. We show here that induction of gut dysbiosis triggers the development of EAE. Furthermore, gut dysbiosis up-regulates the expression of complement C3 gene and subsequently down-regulates the expression of Foxp3 gene in Treg cells and anergy-related E3-ubiquitine ligase genes, CBLB and ITCH, in CD4+CD25- T cells. As a consequence, gut dysbiosis triggers the development of encephalotogenic T cells and their expansion. This data suggests that gut dysbiosis-mediated up-regulation of complement C3 increases the risk of CNS autoimmunity.

This study was supported by Department of Defense- Multiple Sclerosis Research Program Idea Award (MS110174).

**Poster #10**

Enhanced AMPA receptor trafficking mediates anorexigenic effect of endogenous glucagon like peptide-1 in the paraventricular hypothalamus

Ji Liu, Peng Zhang, Varoth Lilascharoen, Byung Kook Lim, Randy Seeley, Julius J. Zhu, Michael M. Scott, Zhiping P. Pang

**PI Name:** Zhiping Pang

Glucagon Like Peptide 1 (GLP-1) plays a pivotal role in central control of energy metabolism and its analogs are clinically proven to be effective in treating obesity. However, the neural basis underlying the regulation of feeding behavior by endogenous GLP-1 remains unknown. Here, we found that specific stimulation of GLP-1 afferent fibers within the paraventricular nucleus of the hypothalamus (PVN) is sufficient to suppress food intake, and conversely, depletion of PVN GLP-1 receptor (GLP-1R) GLP-1R increases food intake and causes obesity. GLP-1R activation augments excitatory synaptic strength in PVN corticotropin-releasing hormone (CRH) neurons. Moreover, a protein kinase A (PKA) dependent signaling cascade is initiated by GLP-1R activation, which results in phosphorylation of serine S845 of GluA1 AMPA receptors and subsequently promotes GluA1 trafficking to the plasma membrane. This study provides a comprehensive multilevel (circuit-, synaptic-, and molecular-) explanation of how food intake behavior and body weight is regulated by endogenous central GLP-1.

Supported by American Heart Association Postdoc Fellowship
**Poster #11**  
Mammalian Target of Rapamycin - mediated mechanisms underlying oligodendrocyte process extension  
Aminat Saliu and Teresa Wood  

**PI Name:** Teresa Wood  

Oligodendrocyte development occurs in multiple stages from initial precursor cells (OPC) to mature myelinating cells. During development, OPCs morphologically transition during differentiation to extend a network of processes through a sequence of events involving cytoskeletal organization. Our lab has previously shown that oligodendrocyte differentiation is regulated by the mammalian target of rapamycin (mTOR), a downstream target of the PI3K/Akt pathway. Pharmacological inhibition of mTOR by rapamycin resulted in a significant reduction in process extension. Moreover, our in vivo studies demonstrate that conditional knockout of mTOR (mTOR cKO) in oligodendrocytes results in hypomyelination in the spinal cord reflected by a reduction in the number of myelinated axons as well as in thickness of myelin membrane around axons. Thus, both in vitro and in vivo studies support the hypothesis that normal mTOR signaling is crucial for proper morphological development of oligodendrocytes. Extension of cellular processes during morphological differentiation of oligodendrocytes involves protrusion at the leading edge initiated by actin filaments positioned immediately beneath the cell membrane. Dynamics of these actin filaments are regulated by profilin, an actin polymerizing factor, and cofilin, an actin depolymerizing factor. Western immunoblot analysis of primary cell cultures revealed a significant reduction in total-profilin2 and phospho-cofilin in mTOR-inhibited cells undergoing differentiation. This reduction was apparent in the first two days of differentiation. In vivo, we also found a reduction in total-profilin2 positive oligodendrocytes in the mTOR cKO, at the peak of differentiation. This suggests deficits in axon ensheathment after differentiation and supports our previous data showing a reduction in total number of myelinated axons. These preliminary data demonstrate a role for mTOR in cytoskeleton reorganization and modulation of cytoskeleton associated protein expression specifically early during morphological differentiation and initiation of myelination.

**Poster #12**  
Mechanisms underlying neuronal development and recovery from injury  
Tomas Kasza, Frank Kung, Chen Liang, Harita Menon, Anton Omelchenko, Kate M. O’Neill, Mihir Patel, Ana R. Rodriguez, Kirsten Svane, Przemyslaw Switakowski, and Bonnie L. Firestein.  

**PI Name:** Dr. Bonnie Firestein  

In the mammalian central nervous system, individual neurons have elaborate dendrite networks, reflecting the density and complexity of incoming presynaptic contacts. The extent of dendrite branching is influenced by learning, which increases branching, or injury, which decreases branching. Our laboratory studies the regulation of dendrite morphology and neuronal function during development and in response to injury or neurocognitive aberrations at the cellular, network, and whole animal levels. We assess the role of the phosphatidylinositol-4,5-bisphosphate 3-kinase/Akt/ mammalian target of rapamycin (PI3K/Akt/mTOR) pathway and brain-derived neurotrophic factor in recovery from traumatic brain injury (TBI). Similarly, we use compounds, such as uric acid and inosine, to aid in recovery from spinal cord injury (SCI). We also focus on the proteins PSD-95 and the cytosolic PSD-95 interactor (cypin) as therapeutic targets for recovery from both TBI and SCI and study these proteins in neurodevelopment. In a parallel project, we focus on the role of NOS1AP, encoded by a schizophrenia susceptibility gene, in neurodevelopment and altered neuronal connectivity. We use multiple techniques, including primary neuronal culture, biochemistry, electrophysiology, microelectrode array analysis, and neurobehavior, to determine how the pathways we study shape the nervous system and its repair. Furthermore, we develop software to aid in the analysis of morphological and microelectrode array data. Our integrative approach uncovers molecular mechanisms that underlie synaptic plasticity, which we aim to harness to improve central nervous system function.  

Supported by National Science Foundation, New Jersey Commission on Brain Injury Research, New Jersey Commission for Spinal Cord Research, NIH Biotechnology Training Grant.
**Poster #13**

NEUROG1 Function is Dependent on Changes in H3K9ac and H3K9me3

Zhichao Song, Azadeh Jadali, and Kelvin Y. Kwan

**PI Name:** Kelvin Kwan

Loss of spiral ganglion neurons (SGNs) due to loud noise significantly contributes to hearing loss. Currently, there are no viable treatments for auditory neuropathies. Understanding how key transcription factors promote SGN differentiation in stem cells will accelerate efforts for replacement therapies. A pro-neural transcription factor, NEUROGENIN1 (NEUROG1), is essential of SGN development and is a potent factor that promotes differentiation of pluripotent stem cells and mouse embryonic fibroblasts to become functional excitatory neurons. Using an immortalized multipotent otic progenitor (iMOP) cell line that can self-renew and differentiate into otic neurons, NEUROG1 bound to the promoter of both cell cycle (CDK2) and neuronal differentiation (NEUROD1) genes. Changes in H3K9ac and H3K9me3 deposition at the promoters altered the NEUROG1 dependent transcription of CDK2 and NEUROD1. In self-renewing iMOP cells, overexpression of NEUROG1 increased CDK2 expression to drive proliferation while knockdown of NEUROG1 decreased CDK2 and reduced proliferation. In contrast, changes in NEUROG1 levels in iMOP-derived neurons did not affect CDK2 but instead, altered transcript levels of NEUROD1. Our findings showed that changes in H3K9ac and H3K9me3 marks affects whether NEUROG1 promotes proliferation or neuronal differentiation.

Supported by Duncan and Nancy MacMillan Faculty Development Chair Endowment Fund and R01 DC015000.

**Poster #14**

Histone Modification is a Novel Epigenetic Mechanism to Up-Regulate Human Blood-Brain Barrier Transporters

Dahea You1, Xia Wen2,3, Ayeshia Morris1, Jason R. Richardson4, Lauren M. Aleksunes2,3.

1Joint Graduate Program in Toxicology, Rutgers University, Piscataway, NJ, 2Environmental and Occupational Health Sciences Institute, Rutgers University, Piscataway, NJ, 3Department of Pharmacology and Toxicology, Rutgers University, Piscataway, NJ, 4Northeast Ohio Medical University, Rootstown, OH

**PI Name:** Lauren M. Aleksunes

Multidrug Resistance Protein 1 (MDR1, ABCB1) and the Breast Cancer Resistance Protein (BCRP, ABCG2) expressed at the blood-brain barrier (BBB) are key efflux transporters which regulate the efficacy and/or toxicity of chemicals in the brain. Prior studies in cancer cells have pointed to the ability of histone deacetylase (HDAC) inhibitors to modulate the expression and function of MDR1 and BCRP. Here we sought to test whether HDAC inhibitors could potentially alter expression and function of MDR1 and BCRP at the BBB. To test this, we treated immortalized human brain capillary endothelial (hCMEC/D3) cells, a model of the BBB, with six different HDAC inhibitors, valproic acid (VPA), sodium butyrate (NaB), romidepsin, apicidin, suberoylanilide hydroxamic acid (SAHA), and trichostatin A (TSA), and assessed for expression and function of MDR1 and BCRP. HDAC inhibition following treatment was confirmed by increased levels of acetylated histone H3 protein. After 12 h of treatment, VPA, apicidin, SAHA, and TSA up-regulated MDR1 mRNA levels between 50% and 200%. All six HDAC inhibitors significantly induced BCRP mRNA levels between 100% and 270%. Similarly, the protein expression of MDR1 and BCRP transporters was up-regulated about two-fold at 24 h. Enhanced MDR1 expression corresponded with reduced intracellular accumulation of the substrate rhodamine 123. Collectively, these results demonstrate that HDAC inhibitors up-regulate MDR1 and BCRP transporters at the BBB by modifying histone acetylation. The clinical use of HDAC inhibitors may enhance efflux transporter activity at the BBB and restrict access of xenobiotics to the brain.

Supported by R01ES021800, P30ES005022.
**Poster #15**
Effects of a Toll-like Receptor 9 Antagonist on Spinal Cord Astrocyte Function, *in vitro*

Lun Li, Li Ni, Eliseo A. Eugenin, Robert F. Heary, Stella Elkabes

**PI Name:** Stella Elkabes & Robert F. Heary

The glial scar is considered as an impediment to axonal regeneration, albeit studies show that astrocytes are necessary for axonal re-growth following spinal cord injury (SCI). The principal cell type in the glial scar are proliferating and migrating reactive astrocytes. Modulation of astroglial function can alter the properties of the glial scar and influence the outcomes of SCI. Toll like receptors (TLRs), which are expressed by SC cells including astrocytes, play important roles in SCI. Our earlier studies have shown that a TLR9 antagonist improves the functional deficits following SCI. The current studies were undertaken to determine whether the TLR9 antagonist modulates astroglial functions pertinent to glial scar formation such as proliferation and migration. Astrocytes were isolated from mixed glial cultures, derived from the SC of postnatal day 3 mice and were passage 3 times to 99% purity. Astrocyte cultures were maintained in medium containing 1% fetal bovine serum, in the presence or absence of CpG ODN 2088, after addition of EdU which labels proliferating cells. A scratch-wound assay and live cell imaging were utilized to assess astroglial migration. CpG ODN 2088 significantly reduced by 40% the number of proliferating astrocytes (p<0.001; n=4) and attenuated astroglial migration into the gap formed by the scratch. These effects necessitated TLR9 since CpG ODN 2088 did not have effects on TLR9−/− astrocytes.

Our studies indicate that astroglial TLR9 antagonism inhibits both proliferation and migration, *in vitro*. Thus, CpG ODN 2088 has the potential of targeting astrocyte functions pertinent to glial scar formation.

Supported by The New Jersey Commission on Spinal Cord Research Grant CSCR12IRG007 and The Reynolds Family Spine Laboratory Funds

---

**Poster #16**
Systemic deficiency of GM1 ganglioside as major risk factor in sporadic Parkinson’s disease leading to failure of GDNF neurotrophic signaling.

1Gusheng Wu, 1Zi-Hua Lu, 2Roy Alcalay, 1Marielle Torres, 3Shawn DeFrees, 1Robert Ledeen

1Rutgers New Jersey Medical School, Dept. Pharmacology, Physiology & Neurosciences, Newark, 07103; 2Columbia University, Dept. of Neurology, New York, NY; 3Seneb BioSciences, Inc., Doylestown, PA

**PI Name:** Robert Ledeen

**Objectives:** (a) To assess GM1 levels in several tissues from sporadic PD patients. (b) To determine efficacy of GDNF signaling in dopaminergic (DA) neurons of substantia nigra pars compact (SNpc). (c) To study GM1 replacement therapy in a mouse PD model, *B4galnt1*(+/−), based on deficient GM1.

**Methods:** (a) PD tissues obtained from PD tissue banks were analyzed for GM1 and GD1α (metabolic precursor) employing HPTLC. GM1 in DA neurons of the SNpc were quantified via immunohistochemistry (IHC). (b) GDNF neurotrophic signaling was assessed by IHC determination of pRET. (c) Use of GM1 analogs as GM1 replacement therapy was assessed with GM1-deficient mice which manifest numerous motor and non-motor symptoms of PD.

**Results:** (a) All examined anatomical regions of PD showed significant GM1 deficiency. Those studied by HPTLC (occipital cortex, colon, heart) were 34-48% below age-matched controls, approximating the reduced levels in the heterozygous *B4galnt1*(+/−) parkinsonian mouse. Preliminary findings for PBMC (lymphocytes) and fibroblasts were similar. IHC revealed severe GM1 deficiency in DA neurons of the SNpc. (b) pRET levels in DA neurons of the SNpc were significantly below age-matched controls, similar to the above mice. (c) Administration of GM1 analogs to the above mice restored GDNF signaling and ameliorated the other PD symptoms.

**Conclusions:** Systemic GM1 deficiency is suggested as major risk factor in sporadic PD, consistent with the extensive PD phenotype in GM1-deficient *B4galnt1*(+/−) mice. A major consequence is failed GDNF neurotrophic support, suggesting the need for clinical testing of potent GM1 analogs capable of restoring effective neurotrophic signaling.
**Poster #17**  
Age dependent effects of ALK5 inhibition and mechanism of neuroprotection in neonatal hypoxic-ischemic brain injury  
Brian H. Kim, Mariano Guardia Clausi, Israel C. Nnah, Chaitali Saqcena, Radek Dobrowolski and Steven W. Levison

**PI Name:** Steven W. Levison

Neonatal encephalopathy due to hypoxic-ischemic (HI) brain injury triggers a wave of neuroinflammation attributed to progressive degeneration and functional deficits seen weeks after initial insult. We previously evaluated the therapeutic efficacy of an antagonist for activin-like kinase 5 (ALK5), TGFβ receptor in a rat model of perinatal HI, and extended those studies of ALK5 antagonism in postnatal day 6 (P6) and P9 HI rat pups with and without hypothermia. ALK5 receptor antagonist SB505124 was administered systemically 3 days following HI via osmotic pump. SB505124-treated rats injured on P6 sustained less damage to their hippocampi and had improved performance on Morris water maze when tested at P60 versus vehicle-treated animals. However, SB505124 did not improve sensorimotor deficits and exacerbated hippocampal/thalamic volume loss when administered to P9 pups. These rats tended to perform worse than their vehicle-treated counterparts on Morris water maze, and treatment did not improve outcome when combined with hypothermia. To elucidate the mechanism of reduced neuronal cell death in the P6 HI model, we assessed components of autophagy in neurons of the hippocampus and thalamus, and found that SB505124 increased levels of two key proteins indicative of active autophagic flux: p62 and LC3. Our results demonstrate a dynamic switch in the CNS response to TGF-β1 occurring before P9 in which TGFβ signaling inhibition worsens functional outcomes. We conclude that attenuating TGF-β1 signaling will likely be an effective treatment for HI-related encephalopathy in pre-term infants, offering protection of the hippocampal and thalamic regions that suffer degeneration even with therapeutic hypothermia.

This work was supported by grants from the National Institutes of Health HD052064 and the Leducq Foundation awarded to S.W.L., and by The International Alzheimer’s Association (NIRG-305325) and the New Jersey Commission on Brain Injury (CBIR14PIL001) awarded to R.D.

---

**Poster #18**  
Role of stathmin-dependent microtubule stability during postpartum depression-like behaviors.  

**PI Name:** Gleb Shumyatsky

One in seven women experiences depression following their baby’s birth. The etiology of postpartum depression (PPD), an episode of major depression disorder (MDD), is poorly understood. Stathmin, a protein that blocks microtubule (MT) formation by binding to tubulin, has a role in fear, post-traumatic stress disorder (PTSD), and maternal and social behaviors. We found that stathmin phosphorylation increases in the dentate gyrus (DG) of wild type mice during pregnancy and postpartum, suggesting increased MT stability. We use mice expressing a mutated form of stathmin (Stat4A) that binds irreversibly to tubulin, causing MTs to be unstable. Our work suggests that eliminating stathmin functionality leads to PPD-like symptoms, such as increased anxiety and decreased pup retrieval. Also, Stat4A male mice show decreased spine density in the DG, similar to what is found in MDD. To better understand the role of stathmin-dependent MT dynamics in PPD-like behaviors we look at dendritic spine changes in the prefrontal cortex (PFC), central amygdala (CeA) and DG of both Stat4A and WT female mice in virgin and postpartum states. To explore long-term PPD-like phenotype, mice were tested 35 to 40 days after delivery of pups. Only Stat4A-PP female mice showed signs of depression-like behavior in sucrose preference test and forced swim test. To identify brain structures responsible for PPD we will use injections of AAV-teto-HA-Stat4A virus into selected brain regions to induce Stat4A expression. Viral injections in GRP-tTA mice, will allow turning on/off Stat4A expression with doxycycline and studying maternal and PPD-like behavior.

G.P.S. was supported by the Whitehall Foundation and March of Dimes. I.C.T supported by NARSAD award. Research funded in part by the New Jersey Governor’s Council for Autism.
Poster #19
Multi-Scale Analysis of the Dynamics of Brain Functional Connectivity using EEG.
Ali Haddad and Laleh Najafizadeh.

PI Name: Laleh Najafizadeh.

We present a new approach to investigate the dynamics of functional connectivity at multiple temporal scales from the recordings obtained through electroencephalography (EEG). Discrete wavelet transform (DWT) is utilized to decompose the recorded signals into several frequency bands, and signals corresponding to each frequency band (referred to as subband components) are reconstructed. Using our proposed source-informed segmentation technique, the subband components are then segmented into intervals during which the spatial distribution of the underlying ensembles of active and functionally connected neurons is expected to stay quasi-stationary. This is achieved through monitoring the running dominant left singular subspace of each subband component matrix for statistically significant shifts in its span over time. Finally, for each identified segment, the source matrix is analyzed for ensembles of functionally connected cortical points. These ensembles are localized through correlating their temporal activity to the dominant right singular vectors of the corresponding source matrix, during the segment. Depending on the number of extracted dominant right singular vectors, multiple ensembles of functionally connected cortical points can be localized per segment and subband. The proposed method is then used to explore the temporal evolution of functional connectivity during the execution of a visual oddball task, at scales spanning δ-, θ-, and α-bands. Results are presented.

This work is supported by DARPA.

Poster #20
Does Sexual Trauma in the Past Alter One’s Sense of Time?

Emma Millon, Michelle Chang, Tracey Shors

PI Name: Tracey Shors

Sexual violence affects over 25% of women worldwide (World Health Organization, 2013), with one in five university students reporting sexual violence (Cantor et al., 2015). Survivors of sexual violence often reharse negative thoughts about the past, while worrying about the future (Buchholz et al., 2016; Falsetti and Resnick, 1997; Nixon et al., 2004). In the present experiment, we hypothesized that women with a history of sexual violence would process time differently (i.e., experience time as moving faster or slower) compared to women without a history. We further predicted that they would ruminate more about the past while expressing anxiety and fear about the future. Women students with (n=16) and without (n=10) sexual violence history were recruited from Rutgers University. Trauma history was assessed with the Structured Clinical Interview for DSM-5 (SCID-5), rumination with the Ruminative Responses Scale (RRS) and anxiety symptoms with Beck Anxiety Inventory (BAI). Temporal perspective and sensitivity was assessed with the Temporal Bisection Task, during which participants make judgements about time duration while viewing presentations of computer-generated stimuli. In this preliminary study, women with sexual trauma history reported significantly more anxiety symptoms compared to women without sexual trauma (p<0.05). Across the entire sample, individuals who reported more reflective ruminative thoughts processed time as moving faster (r=0.79). Overall, these data suggest that the anxious thoughts and memories of sexual violence may alter one’s sense of time.
**Poster #21**

Inhibition of transient receptor potential melastatin 3 ion channels by G-protein beta-gamma subunits

Yevgen Yudin, Doreen Badheka, Istvan Borbiro, Aysenur Yazici, Tibor Rohacs

**PI Name:** Tibor Rohacs

Transient Receptor Potential Melastatin 3 (TRPM3) channels are activated by heat, and chemical ligands such as pregnenolone sulphate and CIM0216. Here we show that activation of receptors coupled to heterotrimeric Gi/o proteins inhibits TRPM3 currents. This inhibition was alleviated by co-expression of proteins that act as sinks for the βγ subunits of heterotrimeric G-proteins (Gβγ). Co-expression of Gβγ, but not constitutively active Gαi or Gαo, inhibited TRPM3 currents. TRPM3 coimmunoprecipitated with Gβ, and purified Gβγ proteins applied to excised inside-out patches inhibited TRPM3 currents, indicating a direct effect. Baclofen and somatostatin, agonists of Gi-coupled receptors, inhibited Ca²⁺ signals induced by pregnenolone sulphate and CIM0216 in dorsal root ganglion (DRG) neurons. The GABAB receptor agonist baclofen also inhibited inward currents induced by CIM0216 in DRG neurons, and nocifensive responses elicited by this TRPM3 agonist in vivo. Our data uncover a novel signaling mechanism regulating TRPM3 channels.

Funded by NIH grants R01-NS055159, R01-GM093290

---

**Poster #22**

Wide-field imaging of sensory-evoked and behavior-related cortical activity in GCaMP6 transgenic mice

Christian R. Lee, Juhi H. Farooqui, Marissa B. DelRocini, David J. Margolis

**PI Name:** David J. Margolis

Advances in fluorescent calcium indicator proteins have allowed imaging of neural activity from specific cell types in various brain regions. We used wide-field transcranial imaging in new generation TITL-GCaMP6f reporter mice to measure calcium signals from excitatory neurons across a large portion of the dorsal neocortex covering visual, somatosensory and parts of auditory, motor and association areas in awake, head-restrained mice. Signal to noise of calcium signals was high with spontaneous events reaching up to 30% amplitude and averaged whisker-evoked responses in primary somatosensory cortex (S1) reaching between 5-10% amplitude. Spatially, mechanical stimulation of single whiskers resulted in early S1 signals followed closely by signals in secondary somatosensory cortex (S2) and motor cortex (M1). We and others have recently reported changes in pupil diameter that occur either spontaneously or during cognitive or motor tasks and reflect changes in brain activity. We therefore determined whether recording GCaMP6 fluorescence from wide areas of neocortex can resolve changes in brain state that occur during either spontaneous or behavior-related pupil dilation. These experiments revealed a resting state network that is associated with constricted pupil and high-amplitude, rhythmic activity in anterior lateral neocortex. This activity is interrupted and replaced by lower amplitude activity in other cortical areas during either spontaneous pupil dilation or dilation induced in a cued-reward behavioral task. Our results indicate that new generation GCaMP6f reporter mice enable mapping the functional properties of cortical networks related to sensory processing, arousal, and sensory-driven behaviors.

Supported by the Whitehall Foundation, the Brain and Behavior Research Foundation, the Rutgers Busch Biomedical Research award, the Rutgers Brain Health Institute Pilot Grant, the New Jersey Commission on Brain Injury Research and the National Institutes of Health.
Changes in cytoplasmic FMRP interacting protein 1 (CYFIP1) dosage contribute toward or exacerbate several neurodevelopmental brain disorders. We previously showed that Cyfip1+/- mice exhibit enhanced mGluR-dependent long-term depression (LTD) in the hippocampus (Bozdagi et al., 2012), which is a shared phenotype with mice lacking Fmrp (Fmr1 KO) but effects of genetic interaction are not known. To test the genetic interaction of Cyfip1 and Fmr1 we generated Cyfip1+/-/Fmr1-/-y mice, and compared mGluR-LTD. mGluR-LTD was enhanced in adult double mutant mice similar to each of the single mutants. However, Fmrp and Cyfip1 levels are not regulated by each other. We next tested Cyfip1+/-/Fmr1-/-y mice for additional synaptic phenotypes that could arise as a consequence of genetic interaction. Adult double mutant mice show no difference in paired pulse facilitation. However, the mice show significantly diminished long-term potentiation and decreased GluN-LTD, which are the phenotypes not seen in either of the single mutants, which suggests an additive or interactive effect of Fmrp and Cyfip1. GluN receptor activity is known to be regulated by actin. Since our previous data show that F-actin levels are increased relative to G-actin in Cyfip1+/- mice (Hsiao et al., 2016), now we are investigating the possibility that GluN-related phenotypes may be related to altered actin dynamics. These data demonstrate that Cyfip1 reduction has an interactive effect with Fmrp in the regulation of postsynaptic function in mature synapses. Cyfip1 may affect the mechanisms underlying a developmentally regulated switch from pre- to postsynaptic phenotypes and contribute to increased disease severity.

This work was supported by the Beatrice and Samuel A. Seaver Foundation, and National Institute of Mental Health to O.G.
**Poster #25**
The KRK motif of the PlexinA4 receptor is required for Sema3A-mediated dendritic arborization *in vitro* and *in vivo*.

**Edward Martinez**, Ron Goldner, **Oday Abushalbag**, Irena Gokhman, Avraham Yaron, Tracy S. Tran

**PI Name:** Tracy S. Tran

The proper wiring of the nervous system during development is largely regulated by extracellular cues and their ability to induce diverse cellular responses through the activation of receptors on the surface of the developing neurons. The Semaphorins are a large family of cues that signal through the Plexin family of receptors, directly or through heteromeric complex with the Neuropilins. Previously, semaphorin (Sema) 3A signaling through Neuropilin-1/Plexin-A4 receptor had been demonstrated to promote basal dendrite arborization of cortical pyramidal neurons, and to induce growth cone collapse of DRG sensory neurons. How this same ligand-receptor pair can trigger diverse cellular responses is largely unknown. Using neuronal cultures and a structure-function analysis approach, we showed that control of the diverse responses could be found at the receptor level, within the cytoplasmic domain of Plexin-A4. Furthermore, we demonstrated that the conserved triplet of basic amino acids, KRK, in the cytoplasmic domain of Plexin-A4, is required for Sema3A induced dendritic arborization of cortical neurons but dispensable for growth cone collapse of DRG sensory neurons. To elucidate the role of the KRK motif in Plexin-A4 signaling *in vivo* we have generated a mutant mouse that harbors KRK to AAA substitution by Crispr/Cas9 technology. We are currently examining multiple neurodevelopmental processes that are regulated by the Sema3A/Plexin-A4 pathway both *in vivo* and *in vitro* in this mouse line. Overall, results from our study will shed new light on the mechanisms that allow the same ligand-receptor pair to trigger different responses in different cell types.

Supported by NSF (IOS1556968) and Busch Biomedical Award to TST, and Israel BSF funding to AY.

**Poster #26**
The Selective mGluR Group I Agonist, 2-chloro-5-hydroxyphenylglycine (CHPG), Increases BDNF and Myelin Proteins following Demyelination in Multiple Disease Models

Kyle Saitta¹,², April DeStefano¹,³, Talia Planas¹,², Maria Isaac¹,³, Yangyang Huang¹, Lauren Lercher¹, and Cheryl F. Dreyfus¹

¹Department of Neuroscience and Cell Biology, Rutgers-RWJMS; ²Joint Graduate Program in Toxicology, Rutgers University; ³Rutgers-GSBS at RWJMS, Piscataway, NJ

**PI Name:** Cheryl F. Dreyfus

Our previous studies indicated that injection of metabotropic glutamate receptor (mGluR) agonists into the cuprizone-lesioned corpus callosum reverses deficits in myelin proteins through the action of astrocyte-derived BDNF. Recent studies have used a more selective mGluR agonist, CHPG, and determined that a reversal in myelin protein deficits in the cuprizone model is elicited through Group I mGluRs. To define the effect of a more clinically relevant approach, CHPG was injected intraperitoneally (ip; 20 or 40 mg/kg at 6 hours and 24 hours prior to analysis). Increases in BDNF and myelin proteins are evident 3 days after the initial injection. To determine if CHPG is effective in a second demyelinating model that includes an immune component, experimental autoimmune encephalomyelitis (EAE) was elicited and CHPG was injected every other day (20 mg/kg) either prior to the appearance of clinical signs of disease or after disease onset. In both cases, CHPG either prevented disease progression or reversed the clinical signs. These effects were accompanied with the reversal of BDNF and myelin protein deficits apparent in EAE. To determine effects in a third disease model, 12-month 3xTg-Alzheimer’s Disease (AD) mice were similarly treated with CHPG (40 mg/kg ip 3X over 1 week). Consistent with previous data, CHPG reversed deficits in BDNF and myelin proteins. Our data suggest that Group I mGluR agonists can be explored as a therapeutic approach to demyelinating effects associated with multiple brain dysfunctions.

Supported by: NIH NS036647; T32ES007148 and NMSS RG 4257B4/1)
Poster #27  
The mechanisms of single-neuron phase maintenance in an oscillatory network

Haroon Anwar, Diana Martinez, Farzan Nadim

Pl Name: Farzan Nadim

Many oscillatory activities require precise firing of neurons. In particular, neurons generating rhythmic motor activity often maintain a constant activity phase, despite large changes in frequency, to produce meaningful behavior. We aim to understand how intrinsic mechanisms and synaptic inputs interact to produce phase stability in a single neuron embedded in an oscillatory network. The activity phases of neurons in the crab pyloric network are maintained across animals oscillating at different frequencies despite the variability in the levels of voltage-gated ionic conductances and synaptic currents. We therefore tested the hypothesis that the synaptic input to a neuron is precisely matched to its intrinsic properties to produce a constant activity phase. To test our hypothesis, we investigated whether 1) the shape or amplitude of the synaptic input is correlated with voltage-gated ionic conductances, 2) the synaptic amplitude influences the activity phase, and 3) the synaptic shape affects the activity phase. In an identified lateral pyloric (LP) bursting neuron, we found no linear correlations between any of the measured ionic conductances and IPSC parameters across animals. We also found that inhibition strength only weakly influenced the time to first spike. However, we found that the shape of IPSC most significantly affected LP burst onset phase. Altogether our results show that phase maintenance emerges from mechanisms, which do not simply rely on linear correlations between synaptic input and intrinsic properties. Moreover, the temporal dynamics of synaptic input, rather than its strength, are most effective in determining the activity phase of a neuron.

Supported by NIH grant MH060605

Poster #28  
Histone modification enables song-specific auditory memories in an avian model.
Efe Soyman, Brittany Bell, Mingwen Dong, Mimi L. Phan, Syed Zammam Saad, David S. Vicario, Kasia M. Bieszczad

Pl Name: David Vicario

Vocal communication relies on the brain’s ability to process, learn and remember important sounds. Long-term memories (LTM) of salient sounds require the expression of genes that subserve stable changes in neural function and connectivity in the auditory areas of the brain. Here we investigate the role of epigenetic mechanisms in auditory memory formation. Following the demonstration that rats treated with a selective inhibitor of a histone de-acetylase (HDAC3-i) acquired a specific and unusually detailed auditory associative memory for pure tones (Bieszczad et al., 2015), we now test the hypothesis that HDAC3-I can enable long-term neuronal memories for natural communication signals: the unique songs of individual songbirds. The songbird caudomedial nidopallium (NCM) is analogous to a secondary auditory cortex and subserves auditory discrimination and song memory. Normally, 200 exposures of a conspecific song will lead to LTM that can be measured electrophysiologically 20h later in NCM by comparing neuronal responses to novel and previously heard songs. In this study, adult male zebra finches heard only 20 repetitions (< the usual threshold for memory formation), followed by systemic injection of specific HDAC3 inhibitor RGFP966 or vehicle control. Neural responses in NCM were recorded 20h later. Multi-electrode recordings in RGFP966 birds showed a significant neuronal memory, which was not seen in vehicle birds, as expected. In addition, immunohistochemistry revealed increased acetylation in NCM of RGFP966 birds. These differences suggest that HDAC3-i lowered the threshold of exposure events required for memory, transforming a sub-threshold auditory experience into neuronal LTM for specific song stimuli.

Supported by R03-DC014753(KMB); R15-D085102(MLP); Rutgers University: Aresty Research Center, SAS Honors Program(SM)
**Poster #29**  
The enhancement of toll like receptor 4 signaling on somatostatin Interneurons mediates granule cell hyperexcitability after FPI

Ying Li, Arielle Kasnetz, Viji Santhakumar

**PI Name:** Viji Santhakumar

TBI is a common causes of acquired epilepsy. We previously reported that TBI induced cell loss, enhancement of toll-like receptor 4 (TLR4) and hyperexcitability of granule cells in dentate gyrus. This hyperexcitability of granule cells were mediated by activation of TLR4 in a NMDA-independent mechanism that likely involves complex cell-type specific modulation of excitatory and inhibitory circuits. Here we examine the effect of TLR4 signaling on inhibitory circuits. The amplitude of evoked IPSC of granule cells decreased after brain injury. LPS-RS ultra, a TLR4 antagonist reversed reduction in granule cell eIPSC after FPI but decreased eIPSC in controls. Somatostatin interneurons expressedTLR4. LPS-RS Ultra selectively reduced somatostatin interneurons death during a high potassium challenge in vitro, after experimental brain injury but not in controls. In whole cell recordings, acute LPS-RS treatment reduced injury-induced increase in frequency of spontaneous EPSC of somatostatin interneurons. Brain injury decreased the rectification index of non-NMDA voltage currents in somatostatin interneurons which was reversed by LPS-RS ultra. However, the rectification index of PV interneurons was not affected by LPSRS ultra. TLR4 signaling leads to differential modulation of GABA currents in granule cells from control and brain injured rats. TLR4 actively modulates spontaneous excitatory synaptic activities and Calcium Permeable AMPA currents in somatostatin interneurons early after brain injury. TLR4 antagonist treatment early after brain injury has the potential to limit excitotoxic inhibitory neuronal loss and maintain GABAergic inhibition in the dentate gyrus.

Supported by NJCBIR CBIR14IRG024 and NIH/NINDS R01NS097750

**Poster #30**  
The role of motivation and safety signals in pathological avoidance in anxiety-vulnerable Wistar-Kyoto rats

Kevin M. Spiegler, Ashley M. Fortress, Jacquelyn N. Tomaio, Jennifer E.C. Fragale, & Kevin C.H. Pang

**PI Name:** Kevin C.H. Pang

Pathological avoidance behavior is a hallmark of all anxiety disorders. Wistar-Kyoto (WKY) rats, which display rapid avoidance acquisition and impaired extinction compared to outbred Sprague-Dawley (SD) controls, are a model of such pathological behavior. The behavior seen in the WKY strain may be due to increased motivation to avoid. Additionally, these potential motivational differences may be attributed to danger and safety signals. The present study utilized a novel avoidance progressive ratio task to determine whether differences in motivation and learned cues can account for strain differences in avoidance learning. WKY and SD rats received 12 sessions of avoidance training, in which a warning tone preceded foot shock. Lever pressing either during the tone (avoidance) or the shock (escape) resulted in the termination of the tone/shock, and onset of a flashing light signaling safety during the intertrial interval. After training, rats were placed in a progressive ratio task in which the number of lever presses needed to avoid shock steadily increased. WKY rats demonstrated increased motivation to avoid foot shock and greater hedonic value of avoiding compared to SD rats. Subsequently, animals were placed in a progressive ratio session without the safety signal. Removal of the safety signal reduced motivation to avoid in SD rats but not WKY rats, suggesting that anxiety vulnerable WKY rats may not process the safety signal. We conclude that WKY rats display increased avoidance motivation that is at least partially attributed to attentional differences to safety signals, implicating impaired safety signal processing in anxiety disorders.

Supported by Biomedical Laboratory Research & Development Service, U.S. Department of Veterans Affairs, VA Office of Research & Development (grant I01BX000132 to KCHP, grant IK2BX-003196-01 to AMF)
Poster #31
The Role of Dentate Gyrus Activin Signaling in Antidepressant Treatment Response

Christine Yohn, Marjorie R. Levinstein, René Hen, Benjamin Adam Samuels

PI Name: Benjamin Samuels

Approximately 32-35 million adults in the US population (16%) experience an episode of major depression in their lifetime, and commonly used treatments, such as selective serotonin reuptake inhibitors (SSRIs), are not ideal since only a subset of patients (~33%) achieves remission with initial treatment. The reasons why some individuals remit to antidepressant treatments while others do not are unknown. Our overall research program addresses this question by assessing antidepressant treatment resistance in mice. Proper assessment of the antidepressant response in mice first requires manipulations that will yield behaviors associated with negative valence constructs that can then be reversed by antidepressant treatment. Chronic treatment of mice with corticosterone (CORT) effectively induces multiple changes in behavior associated with enhanced responses to potential harm and sustained threats. Subsequent chronic treatment with antidepressants such as fluoxetine (FLX) reverses these behavioral changes in some, but not all, of the mice, permitting stratification into responders and non-responders to FLX. We found that there are differences in expression of Activin signaling-related genes between responders and non-responders to FLX in the dentate gyrus, a region that we recently reported is critical for the beneficial effects of FLX on behavior and the hypothalamic-pituitary-adrenal (HPA) axis (Samuels et al 2015).

This work is funded by a NARSAD Young Investigator Award from the Brain & Behavior Research Foundation.

Poster #32
In Vitro Analysis of Microglial Inflammatory Responses

Meenal Paul, David Crockett

PI Name: David Crockett

CNS injury induces an inflammatory response that involves invasions of the injury site by microglia and astrocytes. Microglia are brain’s immune cell and following injury become activated. This leads to these cells migrating towards the injury site. As part of the response, the cells show morphological changes that do not heal. The response afterwards is quick; the beginning of inflammation ranges from minutes to hours post-injury. The concern is that once elicited, the inflammatory response continues since the cytokines become neurotoxic, which leads to the additional loss of cells. My project is to understand the mechanisms, determine what factors elicit the responses, what maintains the response, and what exactly terminates the response. Once we grasp an understanding of this, we would be able to develop pharmacological therapies to control the responses and mitigate against continued inflammation. We assessed for changes in HDAC inhibitors, such as Valproic acid. Valproic acid (VPA), a widely prescribed drug for seizures and bipolar disorder, has been shown to be an inhibitor of histone deacetylase (HDAC). Previous studies in the Crockett lab suggest that HDAC may play a role in eliciting or maintaining glial response. For example, HDAC7 appears to show increased expression in microglia and astrocytes following TBI. There was an increase in microglial inflammation when VPA was administered pre-injury. However, post-injury, with every subsequent hour, the inflammation went down. The cells are rounder, hence more inflamed. There is also reduced intensity of HDAC7, however, we couldn’t quantify it.

Supported by Rutgers Aresty, Douglass Residential College
Poster #33
The role of the ribosome signature in postnatal neocortical circuit development

Tatiana Popovichenko, Anjani Patel, Nicholas Page, Patty Ibrahimian, Mladen-Roko Rasin

PI Name: Mladen-Roko Rasin

The ribosome signature is the distinct combination of ribosomal proteins (R-proteins), large (i.e. Rpl7) and small (i.e. Rps5), active in translation in a given cell. We have been exploring the role of the ribosome signature in development, both at prenatal and postnatal stages. We hypothesize that distinct ribosome signatures dictate gene expression at the protein synthesis step. We found that in vitro knockdown of the Ribosomal protein large 7 (Rpl7) regulates mRNA translation of Foxp2, which is expressed in a subpopulation of glutamatergic neurons in lower layers. However, it has no effect on Foxp1 mRNA, expressed in a subpopulation of glutamatergic upper and lower layer neurons. Both of these are genes are associated with Autism Spectrum Disorder (ASD). In parallel we found, in vivo in a wild-type P22 mouse, that Rpl7 is preferentially translationally active in lower layers of the neocortex. Collectively, these findings suggest that Rpl7 regulates specification and circuitry of lower layers. Indeed, silencing of Rpl7 in vivo resulted in loss of Foxp2 expression, while overexpression of Rpl7 in upper layers downregulates expression of the upper layer neuronal marker Satb2 and increases neurite length. Therefore, we think that the ribosome signature, characterized by distinct R-proteins, is playing a key regulatory role in the translation of gene transcripts driving neurite development and neuronal specification.

Supported by NIH and Aresty

Poster #34
Effects of an 8-week moderate-intensity aerobic exercise intervention on conflict monitoring processes in major depressive disorder

Christopher J. Brush, Ryan L. Olson, Peter J. Ehmann, Brandon L. Alderman

PI Name: Brandon L. Alderman

Major depressive disorder (MDD) is characterized by a number of behavioral, emotional, and cognitive symptoms. Despite the wide variety of treatments for MDD, cognitive impairment remains a common residual side effect, regardless of clinical outcome. Thus, there is a need for more evidence-based alternative treatments that target cognitive dysfunction in depression. The aim of this study was to assess the effects of an 8-week aerobic exercise intervention on depressive symptoms and neurocognitive function in individuals with MDD. Thirty participants (age = 21.1 ± 2.0 years) were stratified by depressive symptoms and randomized to 8-weeks of aerobic exercise (AE) or placebo exercise (PE). AE consisted of three 45 min sessions/week of moderate-intensity exercise, while PE consisted of three sessions/week of light-intensity stretching. Depressive symptoms and N2 event-related brain potentials were assessed at baseline and post-intervention. Following both AE and PE, symptoms of depression decreased, with AE resulting in larger reductions (ES = 0.57; 58% decrease) relative to PE (ES = 0.24; 22% decrease). Compared to PE, AE resulted in improvements in neurocognitive function as demonstrated by larger N2 amplitudes post-intervention (ρ = 0.05, η² = 0.13). Exploratory mediation analyses indicated that changes in N2 amplitudes failed to mediate pre-to-post reductions in depressive symptoms. The findings suggest that an 8-week AE program results in improved neurocognitive function and reduced depressive symptoms amongst individuals with MDD. Future research should examine the influence of exercise in combination with conventional treatments on neurocognitive function in MDD.

Supported by the Charles and Johanna Busch Memorial Fund at Rutgers University
Poster #35
Intermittent access to cocaine increases demand for cocaine in an orexin/hypocretin-dependent manner

Morgan H James, Colin M Stopper, Nikki E Koll, Benjamin A Zimmer, Shayna O'Connor & Gary Aston-Jones

PI Name: Gary Aston-Jones

For almost 20 years, researchers have utilized the so-called ‘long access’ self-administration model to promote compulsive drug seeking behavior in rats. In this model, rats are given continuous access to cocaine for 6 hours per day, every day, for two or more weeks. Recently, our laboratory has adopted an ‘intermittent access’ paradigm, whereby rats are repeatedly given brief (5 min) periods of access to cocaine followed by 25 min periods of abstinence. This pattern of use results in a ‘spiking’ pattern of brain cocaine levels, which more closely resembles the pattern of drug use seen in experienced cocaine addicts. Here, we compared rats given either intermittent or long access to cocaine on a range of addiction behaviors. Further, because our lab has previously shown that the orexin/hypocretin system is particularly important for highly-motivated behavior, we also tested whether an orexin receptor-1 antagonist could reverse any changes in motivation induced by intermittent or long access to cocaine. Compared to long access rats, intermittent access rats showed significantly higher motivation for cocaine, higher levels of compulsive responding for cocaine and higher levels of relapse. Remarkably, these effects persisted for up to 3 months following intermittent access. Further, these behaviors could be reversed by pretreatment with an orexin receptor-1 antagonist. Together, these findings indicate that intermittent access to cocaine results in a stronger addiction-like phenotype in rats, and that orexin-based therapies may represent a possible treatment option for addicted individuals.

Supported by PHS grant 1-R01 DA006214 and NHMRC fellowship 1072706.

Poster #36
Evidence for the presence of local protein synthesis at a presynaptic nerve terminal in the brain, and its requirement to maintain sustained synaptic transmission.

Matthew Scarnati and Kenneth Paradiso

PI Name: Kenneth Paradiso

Neurotransmitter release is fundamental to brain function, and sustained synaptic transmission requires the interaction and maintenance of thousands of proteins. While some synaptic proteins, ion channels for example, are stable for days, others undergo faster turnover. The rate of protein synthesis that is required to replenish the proteins that are necessary to maintain ongoing synaptic activity is not fully understood. We examine this question using electrophysiological recordings and fluorescent imaging at the Calyx of Held, a large synapse in the mammalian auditory brainstem that allows presynaptic and postsynaptic electrical recordings of synaptic transmission. To study synaptic activity, we stimulate the presynaptic axon of this nerve terminal and record the postsynaptic response. We find that blocking protein synthesis by bath application of anisomycin causes a large increase in the number of synaptic failures. We also find an approximately 2-fold increase in the latency, delay, between the presynaptic stimulation time and the postsynaptic response, and an approximately 50% reduction in the expected peak amplitude of the EPSC but prolonged duration compared to control recordings. These results are consistent with a presynaptic effect, and indicate the need for newly synthesized proteins in the presynaptic terminal to maintain synaptic transmission. In agreement with this, using fluorescent noncanonical amino acid tagging (FUNCAT) to fluorescently label newly synthesized proteins, we find evidence of local translation in the presynaptic nerve terminal. In addition, we also find evidence for the presence of ribosomes in the presynaptic terminal. Taken together, our results indicate that local protein synthesis occurs at nerve terminals, and that newly synthesized presynaptic proteins are required to maintain synaptic transmission.

This work was supported by the National Institutes of Health (Grant R00 NS051401-42) to K.G.P.
Poster #37
Serotoninergic signaling in a transgenic mouse model of Huntington’s disease
Samar K. Alselehdar, Elizabeth D. Abercrombie

PI Name: Elizabeth D. Abercrombie

Affective disorders, such as anxiety and depression, commonly manifest in individuals with Huntington’s disease (HD). In HD, anxiety and depression predominantly are treated with pharmacological agents that enhance serotonin (5-HT) neurotransmission. Therefore, we hypothesize that input from the serotonergic system to target structures involved in affective control is altered in HD. In order to study whether serotonergic signaling is altered in HD, we quantified 5-HT neurotransmission using in vivo microdialysis in young BACHD transgenic mice to measure extracellular 5-HT efflux in the ventral hippocampus. This region has a well-documented involvement in the etiology of affective behaviors. We previously have shown that the BACHD model exhibits anxiety-like and depressive-like symptoms at 8 weeks old, and that these symptoms manifest prior to motor deficits assessed via rotarod testing. At this age, we found that 5-HT efflux is decreased in the ventral hippocampus of BACHD mice. These findings support our hypothesis that serotonergic synaptic signaling is disrupted as a result of the HD mutation, and that this pathology may be critically involved in driving the observed behavioral phenotype in BACHD mice. Given our findings, as well as evidence of altered serotonergic neurotransmission in HD patients, we are investigating whether altered reuptake or metabolic processes of the 5-HT system may explain our results. Such an approach may provide insight into the altered synaptic mechanisms of 5-HT signaling that we hypothesize to result in impaired 5-HT neurotransmission and changes in affective circuits in humans with HD.

This research was funded by NIH GRANT NS059921 (EDA).

Poster #38
Epigenetic mechanisms gate the sensory specificity of associative memory and learning-induced plasticity in sensory cortex
Andrea Shang, Sooraz Bylipudi, Kasia M Bieszczad

PI Name: Kasia Bieszczad

Epigenetic mechanisms, such as histone acetylation via acetyltransferases (HATs) and histone deacetylases (HDACs), remodel chromatin. Blocking HDAC activity opens chromatin structure, thereby enabling gene expression, which can facilitate various forms of long-term memory by “releasing the molecular brakes” on learning-induced plasticity (McQuown & Wood 2011). We hypothesize that HDAC3 regulates the specificity and strength of newly formed associative memories by controlling sensory cortical plasticity. Bieszczad et al., (2015) showed that pharmacologically inhibiting HDAC3 (via RGFP966) in rats learning an association between sound and reward enhanced highly-specific tonotopic plasticity for the behaviorally-salient sound-frequency in primary auditory cortex (A1). Here, we sought to determine the behavioral role of HDAC3 function on cortical plasticity for frequency-specific auditory information. Using an instrumental learning paradigm for a two-tone frequency discrimination (2TD), we asked whether rats learned more specific information about sound-signals of different frequencies (5.0 kHz, CS+; 11.5 kHz, CS-) with RGFP966 treatment. If so, then HDAC3 regulates the specificity (vs. generalization) of memory formation. Rats treated with RGFP966 (n=6) acquired frequency-discrimination faster than performance-matched rats given Vehicle (n=6). All animals ultimately acquired the 2TD task to the same performance level, however later memory tests indicated rats given RGFP966 early in 2TD training had stronger, and more frequency-specific memory for both CS+ and CS-sounds. These data support that HDAC3 is a key regulator of memory specificity and strength. Therefore, a role for epigenetic mechanisms in memory formation may be to gate the sensory specificity (vs. generalization) of memory by enabling plasticity in sensory cortices.

Supported by NIH/NIDCD R03 DC014753-01 and funding from SAS and Dept. of Psychology to KMB.
Poster #39
Reversible and Non-Destructive Clearing of Rat and Mouse Brains using Visikol HISTO Approach

Michael Johnson¹, Thomas Villani²,³

¹Department of Environmental Sciences; ²New Use Agriculture and Natural Plant Products Program, School of Environmental and Biological Sciences, and the New Jersey Agricultural Experiment Station (NJAES); ³Department of Medicinal Chemistry, Ernest Mario School of Pharmacy, Rutgers, The State University of New Jersey

Since the introduction of the CLARITY tissue clearing technique in 2013, interest in tissue clearing techniques and 3D histology have increased dramatically as they allow for the three-dimensional interrogation of complex and heterogeneously expressed biomarkers. While current tissue clearing techniques (CLARITY, i/3/uDISCO, Scale, SeeDB) enable researchers to add a new dimension to their research, these techniques have a fundamental limitation that has slowed their widespread adoption. These tissue clearing techniques have not been widely-adopted as they are destructive to tissues, making validation against the gold-standard two-dimensional histological approach challenging. Therefore, it is difficult to determine with current tissue clearing techniques whether a three-dimensional representation of a tissue is indicative of the tissue or of the processing technique used to generate the representation. To address this problem, a novel tissue clearing approach was developed called Visikol HISTO that can render tissues transparent through the process of refractive index matching without damaging underlying cellular morphology. It was demonstrated that with this approach whole-brain immunolabeling can be conducted with both mouse and rat brains for a wide range of immunolabels. Following tissue labeling and clearing it was shown that opacity can be returned to tissues with an ethanol gradient and that tissues can be processed using traditional 2D histology. Through traditional H&E and nissl histological processing it was demonstrated that the Visikol HISTO approach is non-destructive. This Visikol HISTO-based approach represents a shift in how tissue clearing is conducted and can potentially lead to a tool with wide-spread applicability.

Poster #40
Transcription factor regulation by mTOR during oligodendrocyte differentiation

Ornelas, IM; Wahl, SE; Khandker L; Wood, TL

PI Name: Terri Wood

Oligodendrocytes (OL) are generated from precursor cells that give rise to committed oligodendrocyte progenitor cells (OPCs), divide and migrate throughout the CNS. Differentiation of oligodendrocyte progenitor cells into mature oligodendrocytes requires extensive changes in gene expression. We have shown that mTOR is critical for OL differentiation in vitro and in vivo (Tyler et al, 2009, 2011; Wahl et al., 2014). Here we furthered that analysis by investigating how mTOR controls the transcription factor machinery that is essential for the OPCs progression through the OL lineage. In the spinal cord of mice lacking mTOR in the oligodendrocyte lineage (CNP-Cre, floxed-mTOR) we found a significant increase in the mRNA levels of the inhibitor of DNA binding-2 (Id2). Conditional knockout mice show an accumulation in early progenitors, PDGFRα+ cells, at the expense of O4+ cells in spinal cord at PND10. Moreover, Id2 mRNA expression is increased in sorted PDGFRα+ cells. Id2 is a negative regulator of transcription and differentiation in the OL lineage and is mainly regulated by BMP/Smad pathway (Samanta and Kessler, 2004). OPC cultures differentiated in the presence of rapamycin showed increased levels of phospho Smad1/5/8 compared to untreated control cells. Upon mTOR inhibition in vitro and in vivo we also observed a decrease in total levels of Sip1, which is a negative regulator of the Smads. Our preliminary results suggest that mTOR modulates Id2 expression through a complex regulation of the transcriptional machinery at its promoter, which is essential for OPCs to differentiate into mature oligodendrocyte.
**Poster #41**
Regulation of Alternative Cleavage and Polyadenylation During Hippocampal Long Term Potentiation

Aysegul Guvenek, Mariana Fortes, Dinghai Zheng, Yue Feng, Kelsey Martin, Bin Tian

**PI Name:** Bin Tian

In eukaryotes, cleavage and polyadenylation is an essential processing step for almost all mRNAs. More than 70% of mammalian mRNA genes exhibit alternative cleavage and polyadenylation (APA), which results in generation of different mRNA isoforms from a single gene. APA can alter either the 3’ untranslated (3’UTR) region or the coding region and may result in changes in mRNA stability, localization and translational efficiency. Studies have shown that APA regulation differs between cell types and varies under different biological conditions. Among the cell types, brain cells have the longest 3’UTRs, making it possible that APA is a key regulatory mechanism in the brain. Long term potentiation (LTP) is a form of synaptic plasticity, which is persistent strengthening of synapses in response to the repeated stimulations over time. Previous studies indicated that the polyA site (PAS) usage changes in an activity-dependent manner. In this study we examined regulation of APA in LTP-induced mouse hippocampus. Using the deep sequencing (3'READS), we found global shortening of 3’UTR after LTP induction and the shortening is dependent upon the distance between PASs in the 3’UTR. Our analysis also revealed that neuronal activity by LTP induces intronic PAS activation. Finally, we compared LTP induced hippocampal cells with seizure induced cells and observed a significant correlation between relative expression of PAS. Taken together, our results indicate widespread APA changes upon LTP which may impact gene functions in learning and memory.

This work was supported by NIH RO1GM084089 (to BT), NIH R01NS045324 (to KM)

---

**Poster #42**
Spatial and temporal deformation pattern of the brain from blunt trauma

Abdus Ali, Brian Swenson, Chen Miao, Namas Chandra, Bryan J. Pfister

**PI Name:** Bryan J. Pfister

It is widely accepted that under extreme loadings the soft tissue of the brain will deform inside the skull, creating large amounts of both stress and strain on the tissue. This can result in a focal injury, or in the case of acceleration and deceleration, diffuse injuries. Any attempt at understanding the underlying mechanisms and effects of TBI, have to start by focusing on what is actually occurring within the brain. Our objective is to use visual markers embedded within a head surrogate to extract strains (principal tension, principal compression, max shear) under various blunt loading conditions. Three parameters were varied for the experiments: velocity of impact (3 and 5 mph), impact location (crown and front impacts), and the makeup of the brain surrogate (10% and 20% ballistic gel). Varying the impact location and velocities both resulted in significantly different strain values (independent-samples t-test, t(238), p<0.01). To generalize, crown injuries caused higher strains than front impacts, and 5mph impacts caused larger strains than 3mph impacts. Contour maps of the maximum strains occurring in the brain revealed regional differences when comparing crown and front impacts. Using this first approximation, we constructed a procedure of extracting the strains of the brain under loads to improve our understanding of the deformation of the brain during TBI. Future directions include escalating levels of complexity and biofidelity of the model, both in terms of geometry and material properties.

This work was supported by NSF, NJIT, ARL
Does CDNF or MANF increase the ability of floor plate neuroepithelial cells to develop into dopaminergic neurons?

Asthya Saini, Nicola Francis, Zhiping Pang, Prabhas Moghe, Rick Cohen

PI Name: Rick Cohen

Induced pluripotent stem cells (IPSCs) are widely used to study mechanisms of various diseases, effects of novel drug therapies, and cases of regenerative medicine. Through the use of IPSCs, it is possible to investigate the early development of the CNS, to develop a cell based system to produce transplantable therapies, and to develop/screen compounds for stimulation of endogenous regeneration. CDNF and MANF, two relatively new neurotrophic factors (NTFs), are being considered for clinical trials in Parkinson’s disease. These factors have been observed to stop degeneration of DANs and induce neurite and axonal growth in vivo. In this study, we use CDNF and/or MANF to test the percentage of dopaminergic neurons generated from floor plate and if these two factors can help generate a conditional precursor stage that would allow us to amplify the number of TH+ cells prior to terminal differentiation of the dopaminergic neurons.

This work was supported by Aresty Research Center, PeproTech, NIH/R21 NS095082

The spatial and temporal deformation pattern of the brain from blunt trauma

Aswati Arvind, Mathew Long, Kevin Pang, Viji Santhakumar, Namas Chandra, Bryan Pfister

PI Name: Bryan Pfister

Injury from blunt impact is a multi-scale problem where impact forces transmit from the skull to the cellular level. We hypothesize the type and degree of trauma is related to mechanical loading and deformation patterns throughout the brain tissue where each trauma is unique to other types of head trauma. The span and degree of tissue deformation (corresponding to the level of injury) varies across different regions of the brain and is determined by the biomechanical parameters of the blunt injury event (velocity, momentum, direction) and the anatomical boundary conditions of the human head, brain and neck. In this study we developed a full scale skull-brain-neck model with a simulated brain to map the spatial and temporal distribution of deformation in various regions of the brain under blunt impact conditions. A skull filled with ballistic gelatin was be fixed to a biofidelic Hybrid 3 neck and exposed to controlled blunt impact on a Cadex droptower system. Markers spaced >5mm apart were painted onto the surface of a simulate brain to measure deformations patterns throughout the impact using a high speed 3D video imaging system operating at 1000 fps. A 5kg impactor was dropped from heights for impact velocities of 3&5 mph to the forehead and crown. Models were constructed to examine the brain deformation at various depths using a half, ¾ and full skull. Captured video of the motion of the brain markers was used to calculate the tissue level strains and rates of strains using ProAnalyst Software and a custom algorithm. While rotations of the head (not linear motions) are generally agreed to produce brain deformation, strains were found through the cranium under blunt impacts with primarily linear motion of the skull. Maximum strains of 15% were produced from the 5mph impact within the hippocampal region. Deformation maps show the measured motion extending to the brain skull interface. The strain values and location for the produced strain maps provide an important insight into the deformation pattern of the brain under blunt impact that will be useful in linking brain deformation to the type and degree of neuronal damage.

This work was supported by New Jersey Commission on Brain Injury Research
**Poster #45**

Preconception Alcohol Increases Offspring Vulnerability to Stress

Lucy G. Chastain, Shaima Jabbar, Omkaram Gangisetty, Miguel A. Cabrera, Dipak K. Sarkar

**PI Name:** Dipak K. Sarkar

While the detrimental effects of drinking during pregnancy are known, the effect of preconception drinking by the mother on the life-long health outcomes of her children is unknown. In this study using an animal model, we determined the impact of preconception alcohol drinking of the mother on offspring stress response during adulthood. In our preconception alcohol exposure model, adult female Fischer rats were fed with 6.7% alcohol in their diet for four weeks, went without alcohol for three weeks and were bred with untreated male rats to generate male and female offspring. Preconception alcohol exposed offsprings' birth weight, body growth, stress response, anxiety-like behaviors and changes in stress regulatory gene and protein hormone levels were evaluated. Additionally, roles of epigenetic mechanisms in preconception alcohol effects were determined. Alcohol feeding three weeks prior to conception significantly affected pregnancy outcomes of female rats, in respect to delivery period and birth weight of offspring, without affecting maternal care behaviors. Preconception alcohol negatively affected offspring adult health, including increased stress hormone response (corticosterone and ACTH) to an immune challenge (lipopolysaccharides, 0.1 mg/kg body weight) and increased anxiety behaviors in the open field and elevated plus maze tests. These health abnormalities were associated with changes in expression and methylation profiles of stress regulatory genes in various brain areas. Specifically, offspring of pre-conception alcohol exposed (PCAE) dams showed an increase in hypothalamic corticotrophin releasing factor (Crf) expression and a decrease in proopiomelanocortin (Pomc) expression. In addition, offspring of PCAE rats had suppressed hypothalamic Crf methylation of the Cpg dinucleotides at -232 in the proximal promoter as determined by pyrosequencing methods, and increased hypothalamic Pomc gene methylation as determined by pyrosequencing and methylation-specific PCR. These changes in stress regulatory genes and anxiety behaviors were normalized following treatment with a DNA methylation blocker (5-azadeoxycytidine, 5 mg/kg) during the postnatal period. These data highlight the novel possibility that preconception alcohol affects the inheritance of stress-related diseases possibly by epigenetic mechanisms.

**Poster #46**

Application of a scalable assay for rapid and unbiased discovery of novel neuronal ligand-receptor pairs

Sinem Ozgul, Fanomezana M. Ranaivoson, Sumie Kakehi, Davide Sereni, Sventja von Daake, Gaetano T. Montelione, Davide Comoletti

**PI Name:** Davide Comoletti

The synaptic cleft contains a dense material composed by hundreds of different proteins, such as receptors, adhesion proteins, etc., that can be defined as the proteome of the synaptic cleft. For the last half century, one of the key questions in neurobiology has been to understand how these proteins provide the specificity and connectivity to build and maintain trillions of synapses. To infer functional properties from molecular composition we need to understand the function and binding partners of these molecules. Thus, the challenge is to uncover as many new direct protein-protein interactions as possible and to map how these synaptic proteins work in pathways to form the molecular interactome of the synaptic cleft. This new information will be important to understand how these pathways function during brain development. We used a medium-throughput cloning and expression of over 250 cell surface neuronal proteins, and used these proteins to perform ELISA-based binary association studies to identify novel ligand-receptor pairs. In the past two years we have implemented numerous improvements that have considerably enhanced the speed and accuracy at which new ligand-receptor pair are being discovered and validated. We have performed >11,000 direct interactions and we have identified >40 interacting pairs. Of these, about half have never been published. Although this project has not been completed, these new data will lead to a better understanding of synaptic function and circuitry formation. Moreover, new pairs of interacting proteins will set the stage for new structural biology and molecular neuroscience projects.

Supported by NSF BRAIN EAGERs. MCB-1450895
<table>
<thead>
<tr>
<th>Name</th>
<th>Email</th>
<th>Poster #</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ali Haddad</td>
<td><a href="mailto:ali.haddad@rutgers.edu">ali.haddad@rutgers.edu</a></td>
<td>19</td>
</tr>
<tr>
<td>Ali Yasrebi</td>
<td><a href="mailto:ali.yasrebi@rutgers.edu">ali.yasrebi@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Aminat Saliu</td>
<td><a href="mailto:aminatsal@gmail.com">aminatsal@gmail.com</a></td>
<td>11</td>
</tr>
<tr>
<td>Amy Kohtz</td>
<td><a href="mailto:askohtz@gmail.com">askohtz@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Andrea Shang</td>
<td><a href="mailto:andrea.shang@rutgers.edu">andrea.shang@rutgers.edu</a></td>
<td>38</td>
</tr>
<tr>
<td>Angelina Evangelou</td>
<td><a href="mailto:angelina.evangelou@rutgers.edu">angelina.evangelou@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Anna Giarratana</td>
<td><a href="mailto:agiarratan@gmail.com">agiarratan@gmail.com</a></td>
<td>5</td>
</tr>
<tr>
<td>Anna Zhukovskaya</td>
<td><a href="mailto:az3@princeton.edu">az3@princeton.edu</a></td>
<td></td>
</tr>
<tr>
<td>Antonio Merolli</td>
<td><a href="mailto:antonio.merolli@rutgers.edu">antonio.merolli@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>April A Benasich</td>
<td><a href="mailto:benasich@andromeda.rutgers.edu">benasich@andromeda.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>April DeStefano</td>
<td><a href="mailto:aprild1@gsbs.rutgers.edu">aprild1@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Astha Saini</td>
<td><a href="mailto:as1837@scarletmail.rutgers.edu">as1837@scarletmail.rutgers.edu</a></td>
<td>43</td>
</tr>
<tr>
<td>Aysegul Guvenek</td>
<td><a href="mailto:ag1194@gsbs.rutgers.edu">ag1194@gsbs.rutgers.edu</a></td>
<td>41</td>
</tr>
<tr>
<td>Bart Krekelberg</td>
<td><a href="mailto:bart@vision.rutgers.edu">bart@vision.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Benjamin Samuels</td>
<td><a href="mailto:ben.samuels@rutgers.edu">ben.samuels@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Bianca Pineda</td>
<td><a href="mailto:bianca.m.pineda@gmail.com">bianca.m.pineda@gmail.com</a></td>
<td>6</td>
</tr>
<tr>
<td>Biao Wang</td>
<td><a href="mailto:bw315@scarletmail.rutgers.edu">bw315@scarletmail.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Bilal A Khan</td>
<td><a href="mailto:bak127@rutgers.edu">bak127@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Bin Tian</td>
<td><a href="mailto:btian@rutgers.edu">btian@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Brandon Alderman</td>
<td><a href="mailto:alderman@rci.rutgers.edu">alderman@rci.rutgers.edu</a></td>
<td>34</td>
</tr>
<tr>
<td>Brian Kim</td>
<td><a href="mailto:bhk8619@gmail.com">bhk8619@gmail.com</a></td>
<td>17</td>
</tr>
<tr>
<td>Brian P. Keane</td>
<td><a href="mailto:brian.keane@rutgers.edu">brian.keane@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Bryan Pfister</td>
<td><a href="mailto:pfister@njit.edu">pfister@njit.edu</a></td>
<td>42, 44</td>
</tr>
<tr>
<td>Chaitali Saqcena</td>
<td><a href="mailto:cmsaqcena@gmail.com">cmsaqcena@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Charlene Wetterstrand</td>
<td><a href="mailto:bonescannr@gmail.com">bonescannr@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Cheryl F Dreyfus</td>
<td><a href="mailto:dreyfus@rwjms.rutgers.edu">dreyfus@rwjms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Christian Lee</td>
<td><a href="mailto:christian.r.lee@gmail.com">christian.r.lee@gmail.com</a></td>
<td>22</td>
</tr>
<tr>
<td>Christine Yohn</td>
<td><a href="mailto:Cy253@scarletmail.rutgers.edu">Cy253@scarletmail.rutgers.edu</a></td>
<td>31</td>
</tr>
<tr>
<td>Cynthia Roesler</td>
<td><a href="mailto:croesler@andromeda.rutgers.edu">croesler@andromeda.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Dahea You</td>
<td><a href="mailto:airen@scarletmail.rutgers.edu">airen@scarletmail.rutgers.edu</a></td>
<td>14</td>
</tr>
<tr>
<td>Name</td>
<td>Email</td>
<td>Poster #</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Daniel Quintin</td>
<td><a href="mailto:quintind94@gmail.com">quintind94@gmail.com</a></td>
<td>8</td>
</tr>
<tr>
<td>Danielle Gregor</td>
<td><a href="mailto:danielle.gregor6@gmail.com">danielle.gregor6@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>David Crockett</td>
<td><a href="mailto:crockett@rwjms.rutgers.edu">crockett@rwjms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>David Kimball</td>
<td><a href="mailto:kimball@rutgers.edu">kimball@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>David Margolis</td>
<td><a href="mailto:david.margolis@rutgers.edu">david.margolis@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>David Shreiber</td>
<td><a href="mailto:shreiber@soe.rutgers.edu">shreiber@soe.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>David Vicario</td>
<td><a href="mailto:vicario@rci.rutgers.edu">vicario@rci.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Davide Comoletti</td>
<td><a href="mailto:comoleda@rwjms.rutgers.edu">comoleda@rwjms.rutgers.edu</a></td>
<td>46</td>
</tr>
<tr>
<td>Deepak Subramanian</td>
<td><a href="mailto:deepak.subramanian@rutgers.edu">deepak.subramanian@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Dina Popova</td>
<td><a href="mailto:dina.popova@rutgers.edu">dina.popova@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Edward Martinez</td>
<td><a href="mailto:emart@scarletmail.rutgers.edu">emart@scarletmail.rutgers.edu</a></td>
<td>25</td>
</tr>
<tr>
<td>Efe Soyman</td>
<td><a href="mailto:efe.soyman@rutgers.edu">efe.soyman@rutgers.edu</a></td>
<td>28</td>
</tr>
<tr>
<td>Elena K. Rotondo</td>
<td><a href="mailto:elena.rotondo@rutgers.edu">elena.rotondo@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Ellia Miller</td>
<td><a href="mailto:elliam@princeton.edu">elliam@princeton.edu</a></td>
<td></td>
</tr>
<tr>
<td>Emma Millon</td>
<td><a href="mailto:emma.millon@gmail.com">emma.millon@gmail.com</a></td>
<td>20</td>
</tr>
<tr>
<td>Gary Aston-Jones</td>
<td><a href="mailto:gsa35@ca.rutgers.edu">gsa35@ca.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Gina Marrone</td>
<td><a href="mailto:gina.f.marrone@rutgers.edu">gina.f.marrone@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Gleb Shumyatsky</td>
<td><a href="mailto:gleb@dls.rutgers.edu">gleb@dls.rutgers.edu</a></td>
<td>18</td>
</tr>
<tr>
<td>Gwyndolin Vail</td>
<td><a href="mailto:gwyndolinvail@gmail.com">gwyndolinvail@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Hannah Bowrey</td>
<td><a href="mailto:Hannah.bowrey@rutgers.edu">Hannah.bowrey@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Haroon Anwar</td>
<td><a href="mailto:hanwar@njit.edu">hanwar@njit.edu</a></td>
<td>27</td>
</tr>
<tr>
<td>Henri Antikainen</td>
<td><a href="mailto:hpa8@scarletmail.rutgers.edu">hpa8@scarletmail.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Hieronim Jakubowski</td>
<td><a href="mailto:jakubows@rutgers.edu">jakubows@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Hilton Kaplan</td>
<td><a href="mailto:hilton.kaplan@rutgers.edu">hilton.kaplan@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Hye-Jin Park</td>
<td><a href="mailto:hp331@rwjms.rutgers.edu">hp331@rwjms.rutgers.edu</a></td>
<td>2</td>
</tr>
<tr>
<td>Isis Ornelas</td>
<td><a href="mailto:isis.ornelas@rutgers.edu">isis.ornelas@rutgers.edu</a></td>
<td>40</td>
</tr>
<tr>
<td>Janet Alder</td>
<td><a href="mailto:janet.alder@rutgers.edu">janet.alder@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Jennifer Fragale</td>
<td><a href="mailto:catuzzje@njms.rutgers.edu">catuzzje@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Ji Liu</td>
<td><a href="mailto:jl1852@rwjms.rutgers.edu">jl1852@rwjms.rutgers.edu</a></td>
<td>10</td>
</tr>
<tr>
<td>Jiang Ye</td>
<td><a href="mailto:ye.jianghong4@gmail.com">ye.jianghong4@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Jigna Patel</td>
<td><a href="mailto:jpatel816@comcast.net">jpatel816@comcast.net</a></td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Email</td>
<td>Poster #</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Jing Li</td>
<td><a href="mailto:jl1526@njms.rutgers.edu">jl1526@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Joachim Kohn</td>
<td><a href="mailto:kohn@dls.rutgers.edu">kohn@dls.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>John McClure</td>
<td><a href="mailto:hc.tmclure@gmail.com">hc.tmclure@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>John Pintar</td>
<td><a href="mailto:pintar@rwjms.rutgers.edu">pintar@rwjms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Joost Wiskerke</td>
<td><a href="mailto:joost.wiskerke@rutgers.edu">joost.wiskerke@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Josh Berlin</td>
<td><a href="mailto:berlinjr@njms.rutgers.edu">berlinjr@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Juan Pablo Zanin</td>
<td><a href="mailto:juanpablo.zanin@rutgers.edu">juanpablo.zanin@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Juhi Farooqui</td>
<td><a href="mailto:juhi.farooqui@gmail.com">juhi.farooqui@gmail.com</a></td>
<td>22</td>
</tr>
<tr>
<td>Kasia M Bieszczad</td>
<td><a href="mailto:Kasia.bie@rutgers.edu">Kasia.bie@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Katherine Wolfert</td>
<td><a href="mailto:katherine.wolfert@rutgers.edu">katherine.wolfert@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Ken Paradiso</td>
<td><a href="mailto:paradiso@dls.rutgers.edu">paradiso@dls.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Kevin Beck</td>
<td><a href="mailto:kevin.d.beck@njms.rutgers.edu">kevin.d.beck@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Kevin Spiegl er</td>
<td>spiegl <a href="mailto:er@njms.rutgers.edu">er@njms.rutgers.edu</a></td>
<td>30</td>
</tr>
<tr>
<td>Khoosheh Khayati</td>
<td><a href="mailto:kk294@njit.edu">kk294@njit.edu</a></td>
<td></td>
</tr>
<tr>
<td>Kristine Conde</td>
<td><a href="mailto:kmc450@gsbs.rutgers.edu">kmc450@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Kyle Saitta</td>
<td><a href="mailto:kyle.saitta@rutgers.edu">kyle.saitta@rutgers.edu</a></td>
<td>26</td>
</tr>
<tr>
<td>Laura Montroull</td>
<td><a href="mailto:lem@andromeda.rutgers.edu">lem@andromeda.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Lindsey M. Williams</td>
<td><a href="mailto:lindseymichellewilliams@gmail.com">lindseymichellewilliams@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Long-Jun Wu</td>
<td><a href="mailto:longjun.wu@gmail.com">longjun.wu@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Lucy G. Chastain</td>
<td><a href="mailto:guillory.lucy@gmail.com">guillory.lucy@gmail.com</a></td>
<td>45</td>
</tr>
<tr>
<td>Luipa Khandker</td>
<td><a href="mailto:luipak@gsbs.rutgers.edu">luipak@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Lun Li</td>
<td><a href="mailto:lil7@nims.rutgers.edu">lil7@nims.rutgers.edu</a></td>
<td>15</td>
</tr>
<tr>
<td>Maria Isaac</td>
<td><a href="mailto:marieisaac08@gmail.com">marieisaac08@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Marisa Jeffries</td>
<td><a href="mailto:mfa61@gsbs.rutgers.edu">mfa61@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Mark Gergues</td>
<td><a href="mailto:Markgergues@gmail.com">Markgergues@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Mark Plummer</td>
<td><a href="mailto:mplummer@rci.rutgers.edu">mplummer@rci.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Matthew Scarnati</td>
<td><a href="mailto:mss344@rutgers.edu">mss344@rutgers.edu</a></td>
<td>36</td>
</tr>
<tr>
<td>Meenal Paul</td>
<td><a href="mailto:meenal.paul@rutgers.edu">meenal.paul@rutgers.edu</a></td>
<td>32</td>
</tr>
<tr>
<td>Michael Johnson</td>
<td><a href="mailto:m.johnson.36.80@gmail.com">m.johnson.36.80@gmail.com</a></td>
<td>39</td>
</tr>
<tr>
<td>Michelle Chang</td>
<td><a href="mailto:quintessence.hmc@gmail.com">quintessence.hmc@gmail.com</a></td>
<td>20</td>
</tr>
<tr>
<td>Mike Kiledjian</td>
<td><a href="mailto:kiledjian@biology.rutgers.edu">kiledjian@biology.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Email</td>
<td>Poster #</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>--------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Mimi Phan</td>
<td><a href="mailto:mimi.phan@rutgers.edu">mimi.phan@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Mohsen Omrani</td>
<td><a href="mailto:omranim@gmail.com">omranim@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Morgan James</td>
<td><a href="mailto:Morgan.james@rutgers.edu">Morgan.james@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Myungsik Yoo</td>
<td><a href="mailto:msyoo523@gmail.com">msyoo523@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Nicholas Beacher</td>
<td><a href="mailto:nicholasjbeacher@gmail.com">nicholasjbeacher@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Nicholas Bello</td>
<td><a href="mailto:ntbello@SEBS.rutgers.edu">ntbello@SEBS.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Nivedita Rangarajan</td>
<td><a href="mailto:nivedita.rangarajan@princeton.edu">nivedita.rangarajan@princeton.edu</a></td>
<td></td>
</tr>
<tr>
<td>Nowrin Ahmed</td>
<td><a href="mailto:nowrinahmed.nishe@gmail.com">nowrinahmed.nishe@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Omar Itani</td>
<td><a href="mailto:oi7@njit.edu">oi7@njit.edu</a></td>
<td></td>
</tr>
<tr>
<td>Ozlem Gunal</td>
<td><a href="mailto:ozlem.gunal@rutgers.edu">ozlem.gunal@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Pierre-Olivier Polack</td>
<td><a href="mailto:polack.po@rutgers.edu">polack.po@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Qili Yu</td>
<td><a href="mailto:qiliyu@rutgers.edu">qiliyu@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Qinyin Qiu</td>
<td><a href="mailto:qiuqinyin@gmail.com">qiuqinyin@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Radek Dobrowolski</td>
<td><a href="mailto:r.dobrowolski@rutgers.edu">r.dobrowolski@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Rafael Benoliel</td>
<td><a href="mailto:rafael.benoliel@rutgers.edu">rafael.benoliel@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Robert Ledeen</td>
<td><a href="mailto:ledeenro@njms.rutgers.edu">ledeenro@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Robin L. Davis</td>
<td><a href="mailto:rldavis@rci.rutgers.edu">rldavis@rci.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Sabrina Walley</td>
<td><a href="mailto:swalleyy@gmail.com">swalleyy@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Samar Alselehdar</td>
<td><a href="mailto:samar.alselehdar@rutgers.edu">samar.alselehdar@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Sara McEwan</td>
<td><a href="mailto:sm1818@gsbs.rutgers.edu">sm1818@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Seung-Chan Lee</td>
<td><a href="mailto:indigvapr@gmail.com">indigvapr@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Seungwoo Kang</td>
<td><a href="mailto:sk1646@njms.rutgers.edu">sk1646@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Seza Ergun</td>
<td><a href="mailto:sezaergun@yandex.ru">sezaergun@yandex.ru</a></td>
<td></td>
</tr>
<tr>
<td>Smita Thakker-Varia</td>
<td><a href="mailto:varia@rutgers.edu">varia@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Steven Levison</td>
<td><a href="mailto:levisosw@njms.rutgers.edu">levisosw@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Subhashini Joshi</td>
<td><a href="mailto:sj582@scarletmail.rutgers.edu">sj582@scarletmail.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Sudhir Kumar Yadav</td>
<td><a href="mailto:yadavsk@rwjms.rutgers.edu">yadavsk@rwjms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Talia Planas</td>
<td><a href="mailto:talia.planas@gmail.com">talia.planas@gmail.com</a></td>
<td></td>
</tr>
<tr>
<td>Tatiana Popovitchenko</td>
<td><a href="mailto:t.popovitchenko@rutgers.edu">t.popovitchenko@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Teresa Realpe-Bonilla</td>
<td><a href="mailto:trealpe@andromeda.rutgers.edu">trealpe@andromeda.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Thushini Manuweera</td>
<td><a href="mailto:manuweth@gsbs.rutgers.edu">manuweth@gsbs.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Email</td>
<td>Poster #</td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>--------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Tibor Rohacs</td>
<td><a href="mailto:tibor.rohacs@rutgers.edu">tibor.rohacs@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Tomas Kasza</td>
<td><a href="mailto:tk389@scarletmail.rutgers.edu">tk389@scarletmail.rutgers.edu</a></td>
<td>12</td>
</tr>
<tr>
<td>Tracey Shors</td>
<td><a href="mailto:shors@rutgers.edu">shors@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Tracy S. Tran</td>
<td><a href="mailto:tstran@rutgers.edu">tstran@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Troy Roepke</td>
<td><a href="mailto:ta.roepke@rutgers.edu">ta.roepke@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Vaidhyanathan Mahaganapathy</td>
<td><a href="mailto:vaidhyanathan.m@rutgers.edu">vaidhyanathan.m@rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Vanessa Routh</td>
<td><a href="mailto:routhvh@njms.rutgers.edu">routhvh@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Vijayalakshmi Santhakumar</td>
<td><a href="mailto:santhavi@njms.rutgers.edu">santhavi@njms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Wanhong Zuo</td>
<td><a href="mailto:wanhongzuo@163.com">wanhongzuo@163.com</a></td>
<td>7</td>
</tr>
<tr>
<td>Wilma Friedman</td>
<td><a href="mailto:wilmaf@andromeda.rutgers.edu">wilmaf@andromeda.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Yangyang Huang</td>
<td><a href="mailto:huangy4@rwjms.rutgers.edu">huangy4@rwjms.rutgers.edu</a></td>
<td></td>
</tr>
<tr>
<td>Yevgen Yudin</td>
<td><a href="mailto:yyudin73@gmail.com">yyudin73@gmail.com</a></td>
<td>21</td>
</tr>
<tr>
<td>Ying Li</td>
<td><a href="mailto:yl691@njms.rutgers.edu">yl691@njms.rutgers.edu</a></td>
<td>29</td>
</tr>
<tr>
<td>Zhichao Song</td>
<td><a href="mailto:zhichaosong@icloud.com">zhichaosong@icloud.com</a></td>
<td>13</td>
</tr>
<tr>
<td>Zhiping Pang</td>
<td><a href="mailto:zhiping.pang@rutgers.edu">zhiping.pang@rutgers.edu</a></td>
<td></td>
</tr>
</tbody>
</table>