Eleventh Annual Rutgers Brain Health Institute Symposium

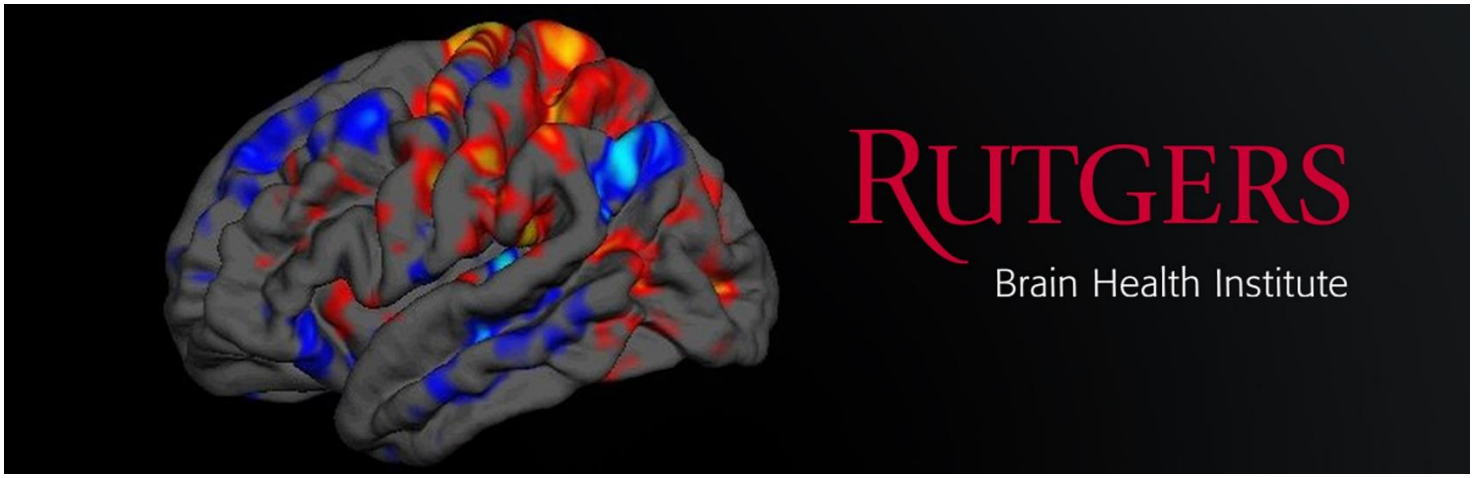
Wednesday, December 3, 2025

Busch Student Center
Piscataway, NJ

8.15 AM	Registration and Continental Breakfast
8.45 AM – 9.00 AM	***Dr. Brian Strom (Chancellor of Rutgers Health)*** *"Welcome Remarks"*
9.00 AM – 9.15 AM	***Gary Aston-Jones, PhD (Director of BHI)*** *"Brain Health Institute updates"*
9.20 AM –9.30 AM	***Detlev Boison, PhD (Professor, RWJMS-Neurosurgery/BHI)*** *"What Turns a Healthy Brain into a Diseased Brain ?"*
9.35 AM – 9.45 AM	***David Barker, PhD (Assistant Professor, RU-NB-Psychology/BHI)*** *"Reversing the Lasting Impacts of Traumatic Stress on Pain and Fentanyl Use Through Lateral Habenula Inhibition"*
9.50 AM – 10.00 AM	***Juan Mena Segovia. PhD (RU-NB-Psychology/BHI)*** *"Multiplexed Motor Functions of the Midbrain"*
10.05 AM – 10.15 AM	***Ioana Carcea, MD PhD (Assistant Professor, NJMS-Pharm, Phys, Neuro/BHI)*** *"Mechanistic Links Between Social Isolation & Glucose Homeostasis"*
10.20 AM – 10.35 AM	Coffee/Tea Break
10.40 AM – 10.50 AM	***Luciana Mascarenhas Fonseca, PhD (RWJMS-Psychiatry/BHI/IFH/KKARC)*** *"Rethinking Early Cognitive Markers of Dementia: From Traditional Testing to Intraindividual and Momentary Approaches"*
10.55 AM – 11.05 AM	***Suchismita Ray, PhD (Associate Professor, SHP-Health Informatics/BHI)*** *"Addiction Neuroimaging, Motivation, and Interventions (ANMI) Lab: Past, Present, and Future Directions"*

11.10 AM – 11.20 AM	***Morgan Firestein, PhD (Assistant Professor, RWJMS-Pediatrics/BHI/CHI/RUCARES)*** *"Prenatal Origins of Neurodevelopment"*
11.30 AM – 12.30 PM	***Keynote- Moses V. Chao, PhD*** ***(NYU Grossman School of Medicine)*** *"Mysteries of Neurotrophic Factors"*
12.30 PM – 1.30 PM	Hot Buffet Lunch
1.30 PM – 1.40 PM	***Hyung Jin Ahn, PhD (Assistant Professor, NJMS-Pharm, Phys, Neuro/BHI)*** *"Vascular and Pathological Mechanisms of Motor Dysfunction in AD/ADRD"*
1.45 PM – 1.55 PM	***Pabitra Sahoo, PhD (RU-Newark- Biological Sciences)*** *"Local Translation in Regeneration, Trauma, and Developmental Disorders"*
2.00 PM – 2.10 PM	***Miriam Bocarsly, PhD (Assistant Professor, NJMS-Pharm, Phys, Neuro/BHI)*** *"Brain Insulin Signaling Rescues Striatal Dopamine Deficits in Overweight Mice"*
2.15 PM – 3.00 PM	***Lewis Burke*** *"Special presentation: "Current Federal Funding Landscape"*
3.15 PM – 4.15 PM	Two-Minute Trainee Flash Talks *"25 Selected talks by trainees from across Rutgers"*
4.15 PM – 5.30 PM	Trainee Poster Session *"More than 55 posters in the Fireside & International Lounge"*
4.00 PM – 5.30 PM	Wine and Cheese & Reception Awards Ceremony

BHI would like to thank Metware Biotechnology Inc, RWD Life Science Co and Bristlecone Solutions LLC for partially sponsoring this 11th Annual BHI Symposium. Please visit their vendor tables & posters for more information.



Mission: The Rutgers Brain Health Institute (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 mission of BHI is to advance brain health through pioneering research, training, and innovation at Rutgers by:

- Promoting multidisciplinary collaborative neuroscience and brain health research.
- Fostering innovative translational studies.
- Cultivating a supportive and inclusive training environment.
- Creating and linking multiple levels of research, from basic discovery through clinical trials.

Vision: *Advancing Brain Health Through Cutting-Edge Research*.

Current Overview and Focus Areas:

- ***Neurodevelopment*** (e.g., autism spectrum disorder, schizophrenia, Tourette's);
- ***Neurodegeneration and Injury*** (e.g., Alzheimer's disease, multiple sclerosis, Parkinson's disease, spinal cord and traumatic brain injury);
- ***Cognitive and Sensory Neuroscience*** (e.g., age-related dementias, pain, auditory disorders);
- ***Motivational and Affective Neuroscience*** (e.g., addiction, eating disorders, obesity, depression, anxiety).

BHI Centers of Excellence:

The Rutgers University Center for Autism Research, Education and Services (RUCARES):

The new center was created by BHI in 2020 and led by inaugural Director, Dr. Wayne Fisher, till July 2025. We are currently recruiting a new Director for RUCARES. The center is responsible for coordinating and fostering basic and clinical research at Rutgers focused on diagnosing, treating, and supporting patients with autism spectrum disorder (ASD). The goal is to develop a world-class autism research center engaged in cutting-edge basic research to identify mechanisms and biomarkers, developing novel interventional behavioral therapies, creating new technologies and services to support both pediatric and adult ASD patient population. BHI partnered with Children's Specialized Hospital (CSH) to form CSH-RUCARES focused on treating autistic children with severe behavior disorders at its clinic on 888 Easton Ave., Somerset, NJ. Since the clinic opened in 2020, it has provided services to more than 200 patients with severe behavior and their caregivers. CSH-RUCARES and the severe behavior program is directed by Dr. Brian Greer, Associate Professor of Pediatrics in RWJMS. RUCARES is the first autism center of its kind in New Jersey dedicated to innovative research, education, and services. The programs focus on diagnosing, treating, and supporting children and adults with ASD. This broad-ranging initiative provides the opportunity for multidisciplinary researchers to partner on care and research for those with ASD with significantly challenging behaviors throughout their lifespan.

Kreiger Klein Alzheimer's Research Center (KKARC):

KKARC was originally started by BHI in 2017 with the recruitment of Dr. Luciano D'Adamio, Krieger Klein Endowed Chair and Professor of Pharmacology, Physiology & Neuroscience at NJMS, spearheading basic and translational research into Alzheimer's disease. In 2023, Dr. Michal Beeri, was recruited by BHI as the Krieger Klein Endowed Chair and Professor of Neurology at RWJMS, and the Director of the Herbert and Jacqueline Krieger Klein Alzheimer's Research Center. The new center conducts observational studies and host clinical trials with the intent of testing new medications and interventions to change the course of Alzheimer's disease and related disorders (ADRD). The center is committed to translational research and fosters collaborations to facilitate a bidirectional flow of research findings, from clinical observational studies in humans to basic science research in various disease models to unravel mechanisms underlying Alzheimer's. The center serves as a hub for collaboration, innovation, and transformative research, with the mission of making meaningful strides in the prevention, diagnosis, and care of ADRD.

The Rutgers Addiction Research Center (RARC):

The RARC at BHI is a new center launched in 2022 developed to help build collaborations among scientists with the multidisciplinary expertise required to advance our understanding of the causes of opioid addiction and other addictive and substance use disorders. Housed within the BHI, RARC is directed by Dr. Danielle Dick, Greg Brown Endowed Chair and Professor of Psychiatry. The center is composed of faculty and trainees across all Rutgers schools and campuses with expertise in addiction prevention, research, treatment, education, and public policy. These include faculty and trainees from RWJMS, NJMS, School of Arts and Sciences, Center for Alcohol Studies, Center for Tobacco Studies, School of Public Health, School of Social Work, School of Nursing, University Behavioral Health Care, School of Pharmacy, and the Institute for Health, Health Care Policy, and Aging Research. RARC is the only comprehensive addiction center in NJ with the capacity to impact the addiction epidemic through the diverse strengths of its members.

The Rutgers-Princeton Computational Cognitive Neuropsychiatry Center (CCNP):

The CCNP was formed by BHI in 2016 to leverage the computational neuropsychiatry expertise in Princeton's Department of Psychology and Neuroscience Institute, and in Rutgers' Departments of Psychology, Psychiatry and Computer Science, Rutgers University Behavioral Health Care, Robert Wood Johnson Hospital, and the BHI, in a major collaborative initiative. The center with its human behavior testing facility is housed in the Research Tower on the Rutgers Busch campus in Piscataway, is co-directed by Dr. Anna Konova from Rutgers (RWJMS/Psychiatry/UBHC) and Dr. Yael Niv from Princeton University.

The Rutgers Center for Advanced Human Brain Imaging Research (CAHBIR):

To fill a critical infrastructure gap, BHI developed CAHBIR a new human brain imaging center located in the Staged Research Building on Busch campus in Piscataway. The center opened July 2021 and houses a state-of-the-art 3T Siemens Prisma MRI that is dedicated for human brain imaging research purposes. The core facility, which now includes equipment for EEG and TMS, is available for use to neuroscientists from across Rutgers and neighboring institutions. The center is directed by Dr. David Zald, Henry Rutgers Term Chair and Professor of Psychiatry in RWJMS. CAHBIR is fully staffed to support the human brain imaging needs of new and experienced users.

BHI is led by Director, Dr. Gary Aston-Jones, PhD, Distinguished Professor of Psychiatry and Strongwater Endowed Chair. BHI staff include- Dr. Eldo Kuzhikandathil, PhD, Executive Director of Administration, Dr. Latha Devi, PhD, Program Manager, Jacqueline Fung, Grant Administrator, Louise Petrone, Program Coordinator, Rose Smith, Business Liaison, Serena Claiborne, Business Liaison, Tongyue (Lily) Zhang, Program Coordinator of Marketing & Communications, and Andrea Dzioba, Secretary.

SPEAKER ABSTRACTS

Keynote Speaker

Moses Chao, PhD

Professor
Department of Cell Biology, Physiology & Neuroscience and Psychiatry
NYU Grossman School of Medicine

"Mysteries of neurotrophic factors"

Dr. Chao's research is focused on how growth factors affect neuronal and glial cell function. Neurotrophic factors, such as NGF and BDNF (Brain-derived Neurotropic Factor), are potent signaling molecules that were originally characterized for their ability to regulate neuronal growth, survival and differentiation during early development. However, in adults, trophic factors are more critical for synaptic plasticity and in neuropsychiatric disorders, including anxiety, depression, pain, injury and mood disorders. Levels of BDNF have also been directly linked to metabolic syndromes, such as obesity and hyperphagia. Recently it has been confirmed that mature post-mitotic neurons possess the ability to become independent of trophic factors for survival. The future aims are to understand how independence from trophic factors is achieved, which may represent a key factor in the longevity of the nervous system, as well as in neurodegenerative diseases

Moses V. Chao PhD is Professor of Cell Biology, Neuroscience & Physiology and Psychiatry at the NYU Langone Medical Center. He received his BA degree from Pomona College and his PhD in biochemistry from UCLA. After postdoctoral research at Columbia University with Richard Axel, he started a faculty position at Weill Cornell Medical School, where he defined the genes encoding the NGF receptor and identified mechanisms used by trophic factors to change synaptic plasticity.

Chao served as Senior Editor for the *Journal of Neuroscience* for eleven years and is presently an editor for *PNAS* and *eLife*. As a member of the Scientific Advisory Boards for Target ALS, the Pershing Square Foundation and the Pritzker Foundation, he has been a frequent reviewer for the NIH, the Simons Foundation and the Weizmann Institute. Chao was President of the Society for Neuroscience in 2012 and was elected to the National Academy of Sciences in 2023. He is a Fellow of the AAAS and a recipient of a Zenith Award from the Alzheimer's Association, an NIH Jacob Javits Neuroscience Investigator Award, a Guggenheim Fellowship and the Julius Axelrod Prize.

Detlev Boison, PhD

Professor
Department of Neurosurgery and BHI
Vice Chair of Research & Training
Rutgers Robert Wood Johnson Medical School, Piscataway, NJ.

What Turns a Healthy Brain into a Diseased Brain?

Most patients with epilepsy and neurodegenerative conditions present with acquired (i.e., sporadic/non-familial) forms of the disease. For example, it is estimated that 95% of all Alzheimer's (AD), 90% of all Parkinson's (PD), and >60% of all adult epilepsy cases are acquired and thought to be triggered by a precipitating injury to the brain. Understanding the mechanisms that turn a healthy brain into a diseased brain provides the foundation for the development of disease-modifying and preventative therapies. There is growing evidence that various types of brain injury such as traumatic brain injury (TBI), status epilepticus (SE). stroke, or exposure to pathogens or chemicals, trigger common pathways and mechanisms thought to contribute to disease development during a 'laten" period, which can be months to years in humans and weeks to months in rodents. These mechanisms include neuroinflammation, disruption of the blood brain barrier, activation of microglia and astrocytes, cellular restructuring and metabolic and epigenomic reprogramming. Importantly, resulting pathology shares striking comorbidities. Thus, people with epilepsy have cognitive impairment as frequent comorbidity, whereas epileptic seizures are a frequent comorbidity in AD. Both conditions also share depression, anxiety, and sleep changes as common comorbidities. This lecture will discuss brain injury-induced disease-causing pathways and mechanisms, which are common to multiple types of injuries and implicated in the etiology of multiple neurological symptoms and how this information can be used for the discovery of novel disease and preventative therapeutic treatments for multiple conditions.

David Barker, PhD

Assistant Professor
Department of Psychology and BHI
Rutgers-New Brunswick School of Arts and Sciences, Piscataway, NJ.

Reversing the Lasting Impacts of Traumatic Stress on Pain and Fentanyl Use Through Lateral Habenula Inhibition

Traumatic stress is a powerful driver of long-lasting vulnerability to pain, mood disorders, and substance use. Understanding the brain mechanisms that translate acute trauma into persistent sensory and affective dysfunction is critical for developing new treatments. While many limbic circuits have been implicated, the lateral habenula (LHb), a glutamatergic hub for aversion and reward processing, has emerged as a promising candidate. Here, we tested whether LHb activity mediates vulnerability following traumatic stress. Using chemogenetic manipulations combined with behavioral assays of pain, affect, and reward, we show that LHb signaling is necessary for stress to produce enduring hypersensitivity and negative affect. We then asked whether the LHb could serve as a therapeutically relevant target. Using sustained inhibition of LHb neurons via oral delivery of the DREADD agonist J60, we found that stress-induced hyperalgesia could be reversed, providing proof-of-concept for circuit-specific, non-opioid strategies. Together, these findings establish the LHb as a key node linking traumatic stress to maladaptive outcomes and highlight its potential as a translational target for interventions aimed at stress-related pain and affective disorders.

Juan Mena Segovia, PhD

Associate Professor
Center for Molecular and Behavioral Neuroscience
Rutgers University, Newark, NJ

Multiplexed Motor Functions of the Midbrain

The midbrain integrates sensory, motivational, and motor information to shape purposeful behavior. Within this region, the pedunculopontine nucleus (PPN) contains distinct neuronal subtypes that contribute to movement in parallel and complementary ways. My research combines cell-type–specific recording, stimulation, and imaging approaches to understand how these populations coordinate motor preparation, initiation, and execution. We find that cholinergic, glutamatergic, and GABAergic neurons each engage selectively depending on behavioral context, linking internal state and external demands to the form and vigor of movement. When these neurons are disrupted, either experimentally or through disease, animals show specific deficits in the organization of action rather than a simple loss of motor output, suggesting that the PPN operates as a modulatory hub for flexible behavior. This framework helps explain why disorders that affect the PPN, such as Parkinson's disease, produce complex changes in motor and cognitive performance. Ongoing work extends this approach to define how midbrain circuits integrate with cortical and basal ganglia loops. Together, these studies reveal how parallel cell-type–specific pathways in the midbrain encode and control distinct components of action, and how their imbalance leads to characteristic motor and cognitive disturbances.

Ioana Carcea, MD PhD

Assistant Professor
Department of Pharmacology, Physiology and Neuroscience and BHI
Rutgers New Jersey Medical School, Newark, NJ

Mechanistic Links Between Social Isolation and Glucose Homeostasis

Loneliness and social isolation are strongly associated with increased incidence of diabetes in humans. However, it remains unclear if lack of meaningful social interactions represents a cause or a symptom of disease. In rodents, social isolation leads to metabolic dysregulation, however the dynamics and contributing factors remain poorly understood. Here we show that single-housing young adult male mice for at least three weeks led to fasting hyperglycemia, an effect that was maintained for the duration of single-housing but was corrected within a week of reverting mice to co-housing conditions. Single-housing did not affect glucose regulation in intact or ovariectomized females. Instead, sex differences in glucose regulation during single-housing were driven by testicular factors, as orchiectomy prevented isolation-induced fasting hyperglycemia in males. Consistently, single-housing induced increased levels of glucagon post-fasting in intact but not orchiectomized males. Additionally, the inability to thermoregulate via social interactions that mice initiate at sub-thermoneutral ambient temperatures, contributed significantly to isolation-induced fasting hyperglycemia. Taken together, we show that social isolation can affect glucose regulation via a combination of gonadal and thermoregulatory mechanisms.

Luciana Mascarenhas Fonseca, PhD

Assistant Professor
Department of Psychiatry and BHI/IFH/KKARC
Rutgers-Robert Wood Johnson Medical School, Piscataway, NJ

Rethinking Early Cognitive Markers of Dementia: From Traditional Testing to Intraindividual and Momentary Approaches

Understanding the earliest signs of cognitive decline is essential for timely intervention and improved outcomes in dementia care. My research centers on advancing cognitive assessment methods to detect Alzheimer's disease (AD) and related disorders earlier and more accurately, and to understand mechanisms related to resilience and resistance to pathology. Traditional neuropsychological tests remain valuable, but their limitations in capturing early, subtle changes, especially in populations with atypical development or aging trajectories, necessitate new approaches. I will briefly highlight emerging strategies that shift the focus from static, one-time performance to dynamic, person-centered metrics. These include intraindividual cognitive variability, the within person variability in cognitive tests or domains, measured at a single time point, that provides an opportunity to go beyond mean comparisons with a normative sample, alongside digital cognitive assessments, which offer scalable and frequent measurement opportunities; and ecological momentary assessment (EMA), which captures cognition and behavior in real-time contexts. I will conclude by outlining how these methods can inform future clinical trial design and personalized interventions, as well as my goals to contribute to a precision aging research framework that integrates diverse tools and populations. This evolving approach holds promise for identifying risk earlier and tailoring care across the lifespan.

Suchismita Ray, PhD

Associate Professor
Department of Health Informatics and BHI
Rutgers School of Health Professions, Newark, NJ.

Addiction Neuroimaging, Motivation, and Interventions (ANMI) Lab: Past, Present, and Future Directions

I am a Cognitive Psychologist by training and completed my post-doctoral training in Addiction Neuroscience. My lab activities started with an independent line of research while at Rutgers Center of Alcohol Studies by obtaining an NIH/NIDA K01 grant that used cognitive experimental tasks and functional magnetic resonance imaging (fMRI) to examine brain function in chronic cocaine smokers. Results were the first demonstration in the addiction field of a causal interaction between brain regions within a large-scale reward network that gives rise to drug seeking behavior. Through my NIH/NIDA I/START grant, I demonstrated that individuals with opioid use disorder show impaired brain structure and function within a large-scale reward network. During 2020, I became interested in mindfulness intervention research with the goal of improving the behavior and brain structure and function of the individuals with substance use disorders to enhance the treatment outcomes. I conducted a couple of Rutgers internal grant funded pilot studies on mindfulness. The results of the pilot projects led to an NIH funded R61/R33 (Contact PI; 2024) grant that is examining the efficacy of a combined Guanfacine pharmacotherapy and mindfulness behavioral intervention on opioid craving and opioid use outcomes in individuals with opioid use disorder. In another NIH funded multi-site R01 clinical trial grant (Site PI; 2024) I am examining the efficacy of Guanfacine pharmacotherapy in individuals with alcohol use disorder. In the next 5 years, I will continue submitting NIH grants with an intention to continue understanding of the neurocognitive mechanisms underlying various drugs of addiction and develop individual patient specific interventions.

Morgan Firestein, PhD

Assistant Professor
Department of Pediatrics
Child Health Institute and BHI/ RUCARES
Robert Wood Johnson Medical School, Piscataway, NJ

Prenatal Origins of Neurodevelopment

Her research program applies translational and clinical research approaches to identify and examine early emerging biological mechanisms during pregnancy that contribute to the etiology of neurodevelopmental disorders, including autism. Stemming from the Developmental Origins of Health and Disease (DOHaD) framework, Dr. Firestein has developed a fetal ecological systems model and will provide an overview of her lab's current and prior studies that have examined how the intrauterine environment shapes the developing brain with long-term implications for neurobehavioral outcomes. Most recently, Dr. Firestein's lab has investigated associations between risk for neurodevelopmental disorders and aberrant levels of prenatal sex steroid hormones and extracellular vesicles of maternal, fetal, and placental origin.

Hyung Jin Ahn, PhD

Assistant Professor
Department of Pharm/Phys/Neuroscience and BHI/KKARC
Rutgers-New Jersey Medical School, Newark, NJ

Vascular and Pathological Mechanisms of Motor Dysfunction in AD/ADRD

Motor dysfunction is an increasingly recognized but understudied feature of Alzheimer's disease (AD) and related dementias (ADRD). Although severe impairment typically arises in late stages, 10–40% of patients exhibit motor deficits much earlier, where they correlate with accelerated cognitive decline, disease severity, and mortality. Despite their clinical significance, the mechanisms driving motor dysfunction in AD remain unclear. White matter lesions (WMLs), characterized by disrupted myelination, are strongly linked to motor impairment and heightened AD risk, suggesting a vascular contribution. To explore this relationship, we examined aged AD mouse models with motor deficits and observed fibrin deposition, demyelination, axonal degeneration, and reduced vascular density in the striatum—findings that implicate vascular pathology in motor decline.

Familial Danish Dementia (FDD), a rare autosomal dominant neurodegenerative disease caused by ITM2b mutations, also presents with WMLs, vascular compromise, and progressive motor dysfunction, making it a powerful model to probe shared pathogenic mechanisms. Using a novel FDD knock-in (FDD-KI) rat model, we identified age-dependent deposition of Danish amyloid (ADan) and amyloid-β cerebral amyloid angiopathy (CAA), alongside myelin disruption, axonal loss, and widespread vascular permeability with extravascular fibrinogen leakage. These vascular and white matter pathologies were closely associated with progressive motor and gait abnormalities, paralleling clinical features of FDD.

Together, our findings indicate convergent vascular and white matter mechanisms underlying motor dysfunction in both AD and FDD, offering new insights into disease progression and potential therapeutic targets.

Pabitra Sahoo, PhD

Assistant Professor
Department of Biological Sciences
Rutgers School of Arts and Science, Newark, NJ

Local Translation in Regeneration, Trauma, and Developmental Disorders

Neurons rely not only on transcriptional programs but also on precise posttranscriptional regulation of gene expression to respond to injury and developmental cues. A key regulator of this process is the stress granule (SG), a dynamic RNA-protein condensate that governs the sequestration and translational fate of specific mRNAs. By modulating local translation, SGs influence whether axons can regenerate or succumb to dysfunction. Our lab investigates how G3BP1-positive SGs control mRNA availability during axonal repair. In models of spinal cord, peripheral nerve injury, and traumatic brain injury, we study how SG assembly and disassembly regulate the local translation of regeneration-associated transcripts. We further extend these insights to neurodevelopmental disorders, where mutations in SG components, including G3BP1, are linked to disrupted synaptic development and autism spectrum-related phenotypes. Our long-term goal is to leverage this knowledge to enhance regeneration after trauma and uncover mechanisms underlying neurodevelopmental disorders.

Miriam Bocarsly, PhD

Assistant Professor
Department of Pharm/Phys/Neuroscience and BHI
Rutgers New Jersey Medical School, Newark, NJ

Brain Insulin Signaling Rescues Striatal Dopamine Deficits in Overweight Mice

Obesity is associated with insulin resistance and motivational deficits. In the brain, lower levels of striatal dopamine D2-receptor availability have been associated with obesity, although inconsistently. The mechanisms linking weight gain and insulin sensitivity with D2-receptor levels and motivational deficits are not fully identified. Here we probe the causal and mechanistic link through direct manipulations to striatal dopamine receptor levels. We have found that male, but not female, mice with low striatal D2-receptors consumed more food and gained more weight than littermate controls on standard diet. Consistent with an obesity phenotype, mice also exhibited metabolic signs of peripheral insulin resistance. Motivational deficits that preceded weight gain included a delayed circadian onset of locomotor activity, reduced physical activity, and lower effort to obtain food. In the brain, male mice with low D2- receptors showed reduced striatal dopamine release capacity and age-dependent changes in insulin sensitivity. Before weight gain, insulin responses were blunted, compared to controls where insulin increased cholinergic interneuron firing and acetylcholine amplitude to potentiate dopamine signaling. However, once overweight, these mice exhibited a hypersensitive brain response to insulin, whereby insulin strongly potentiated dopamine release, effectively reversing the baseline deficiency. Together, our findings show that male mice with reduced D2- receptors exhibit obesity-linked behavioral and circuit alterations before weight gain, possibly driven by striatal insulin receptor dysregulation.

Top 25 Two-Minute Trainee Flash Talks

Name	Title	Poster #	Lab- PI
Anastasia Balatsky	*Role of a Potassium Ion Channel Mutant in Neuronal Development*	**6**	Federico Sesti
Jose Cevallos	*Lipid droplets promote the aberrant liquid-liquid phase separation of alpha-synuclein in Parkinson's disease leading to impaired energy homeostasis*	**38**	Eleanna Kara
Sarah Delcourte	*Retinal Regulation of Locus Coeruleus: A Chemogenetic Approach to Treat Neurodegenerative Disorders*	**32**	Gary Aston-Jones
Yicong Le	*AAV-Mediated BDNF and GAS6 Muscle Delivery Delays Disease Onset in SOD1G93A ALS Mice*	**20**	Renping Zhou
Joshua Stein	*Advanced Biomaterial Delivery of Hypoxia-Conditioned Extracellular Vesicles (EVs) as a Therapeutic Platform for Traumatic Brain Injury*	**54**	KiBum Lee
Yan Tong	*Altered Bioenergetic Profiles in Neurons with Frontotemporal Dementia-Associated MAPT Mutation.*	**16**	Ching-On Wong
Ian Biluck	*Strain and sex effects on behavioral responses to model blast-induced traumatic brain injury.*	**5**	Bruce Citron
Olivia DePasquale	*Examining Therapeutic Potential of Lateral Habenula Inhibition for Chronic Pain and Stress-induced hyperalgesia.*	**8**	David Barker
Raymond Chien	*Mapping morphine-induced changes of global protein phosphorylation in brain regions of a mouse tolerance model using tandem mass tag-based phosphoproteomics/proteomics.*	**17**	Ying-Xian Pan
Nusrath Yusuf	*The role of Tex15 in stochastic olfactory receptor choice.*	**43**	Kevin Monahan
Joel Raymond	*Duel-edged Dosing: Dexamphetamine and Lisdexamfetamine Suppress Binge-like Eating but Disrupt Sleep in Female Rats*	**22**	Morgan James
Tyler Kays	*Reducing neuronal nitric oxide synthase (nNOS) expression in the ventromedial hypothalamus (VMH) increases body weight and blood glucose levels while decreasing body temperature in male mice.*	**30**	Vanessa Routh

Xijun Wang	*Autism- and Schizophrenia-Associated 3q29 CNV Deletion Drives Transient Hyperproliferation of Neural Precursor Cells During Cortical Neurogenesis*	**34**	Emanuel DiCicco-Bloom
Jack DeLucia	*Purkinje cell development, survival, and adaptive motor behavior in mice require the redundant function of the small GTPases Rab11a and Rab11b*	**27**	Tracy Tran
Abigail Heller	*Expression Level Impact of Developmental Cc2d1a Reduction as a Sex-Specific Mouse Model of ASD/ID and growth deficits.*	**53**	Chiara Manzini
Sol Diaz de Leon-Guerrero	*Loss of function mutations in neurodevelopmental and psychiatric risk genes alter neuronal morphology and synaptic transmission in human neurons*	**46**	Zhiping Pang
Tatyanna Landell	*Perceived Stress in Autistic vs Non-Autistic Adolescents*	**19**	Kristina Jackson
Andrew Boreland	*Ethanol induces neuroimmune dysregulation and soluble TREM2 generation in a human iPSC neuron, astrocyte, microglia triculture model.*	**51**	Ron Hart
Jason Sherman	*Phosphodiesterase 2A Modulates Aβ-induced Oxidative Stress & Neuroinflammation in HT22 cells*	**35**	Ying Xu
Rouba Houbeika	*Perinatal IL-6 elevation changes hippocampal plasticity and memory via postsynaptic mechanisms*	**31**	Ozlem Gunal
Rebecca Zaritsky	*The maternal inflammation associated cytokine Interleukin-6 alters gene expression in neural progenitors.*	**36**	Steve Levison
Jeremy Willekens	*CSF Metabolomic Signature During Therapy for Childhood Acute Lymphoblastic Leukemia Predicts Subsequent Working Memory Impairment*	**2**	Peter Cole
Aishwarya Dodda	*The Interaction Between Physical and Mental Fatigue*	**37**	Glenn Wylie
Li Xin Lim	*Effort to Engage, Effort to Adapt: The Brain Near Criticality*	**40**	Andrew Westbrook
I-Tzu Hung	*Genetic Liability for Substance Use Explains Variations in Suicidal Ideation and Behaviors: Findings from the Adolescent Brain Cognitive Development Study*	**23**	Jill Rabinowitz

POSTER ABSTRACTS

Poster #1

TRPM3 inhibition by Gβγ is a key mediator of opioid-induced analgesia in inflammatory pain.

Authors

Carter Burton, Nawoo Kim, Yevgen Yudin, Huijuan Hu, Tibor Rohacs

PI Name: Tibor Rohacs

TRPM3 is a heat-activated cation channel that is an important detector of noxious heat in peripheral nociceptors and TRPM3 activation is sufficient to induce pain. Activation of G_i-coupled G-protein coupled receptors (GPCRs) causes TRPM3 inhibition via a direct interaction between TRPM3 and Gβγ. The physiological relevance of TRPM3 inhibition by Gβγ has not previously been established in a behavioral model. Using CRISPR-Cas9 genome editing, we generated a mouse line ($TRPM3^{\Delta Ex17}$) without Trpm3 exon 17, which encodes the Gβγ binding site, thus disinhibiting TRPM3 following activation of G_i-coupled GPCRs. Calcium imaging of dorsal root ganglion (DRG) neurons isolated from $TRPM3^{\Delta Ex17}$ mice shows TRPM3 disinhibition following activation of G_i-coupled mu-opioid receptors (MOR). Using $TRPM3^{\Delta Ex17}$ mice, we show that inhibition of TRPM3 is an important mediator of exogenous-opioid-induced analgesia in heat hyperalgesia in the Complete Freund's Adjuvant (CFA) model of inflammatory pain. Based on this finding, we developed a second novel mouse line ($TRPM3\text{-}Ex17^{fl/fl}$) in which Trpm3 exon 17 is flanked by *loxP* sites to permit cell-type-specific disinhibition of TRPM3. We crossed $TRPM3\text{-}Ex17^{fl/fl}$ mice with $Trpv1^{Cre}$ mice to selectively disinhibit TRPM3 in peripheral nociceptors and validated exon 17 deletion using calcium imaging of DRG neurons. $Trpv1^{Cre}$; $TRPM3\text{-}Ex17^{fl/fl}$ mice show significant reductions in opioid-induced analgesia to heat hyperalgesia caused by CFA-induced inflammation when TRPM3 in peripheral nociceptors is disinhibited. Collectively, our data show that TRPM3 inhibition in peripheral nociceptors is a key mediator of opioid-induced analgesia in inflammatory heat hyperalgesia.

Supported by NIH grant R01-NS055159

Poster #2

CSF Metabolomic Signature During Therapy for Childhood Acute Lymphoblastic Leukemia Predicts Subsequent Working Memory Impairment.

Authors

J. Willekens, S.A. Sands, M.A. Burns, L.B. Silverman, P.D. Cole

PI Name: Peter D. Cole

Background: Although typically curative, treatment for pediatric acute lymphoblastic leukemia (ALL) is associated with neurotoxicity and leads to chemotherapy-related cognitive impairment (CRCI) in 40–70% of survivors. CSF routinely collected during intrathecal chemotherapy, offers a direct window into brain metabolism. This study characterizes longitudinal metabolic changes in the CSF of pediatric patients undergoing chemotherapy for ALL. Methods: CSF samples from pediatric patients enrolled in the DFCI ALL Consortium Protocol 16-001 were collected at 5 standardized timepoints over the first 20 weeks of treatment and analyzed using untargeted metabolomics. Cognitive outcomes were assessed post-treatment, with the Working Memory Index (WMI) serving as the primary cognitive measure. Patients with WMI scores at least 1 SD above or below the mean were selected for metabolomic comparison. Results: Our analysis revealed a profound reorganization of the CSF metabolome during the first 18 days of treatment, spanning the induction phase of chemotherapy and early leukemia remission. This shift was characterized by alterations in amino acid, phospholipid, and one-carbon metabolism. Of note, we identified a metabolomic signature predictive of low post-treatment WMI, implicating metabolic dysregulation in CRCI susceptibility. Conclusions: These findings highlight the dynamic impact of chemotherapy on the CSF metabolome and support its utility as a matrix for monitoring neurotoxicity during pediatric ALL therapy. CSF metabolomics may enable the early identification of patients at risk for CRCI through predictive biomarkers, and guide future neuroprotective interventions.

Supported by NIH/NCI (R01 CA220568), New Jersey Pediatric Hematology/Oncology Research Center of Excellence (NJ PHORCE)

Poster#3

Establishing a Gerbil Model to Study the Interaction Between Hearing Loss and Alzheimer's Disease Pathology.

Authors

Marissa Calvano, Kasia Bieszczad, Justin D. Yao

PI Name: Justin Yao

Alzheimer's disease (AD) is a neurodegenerative disorder marked by the accumulation of amyloid-beta (Aβ) oligomers and hyperphosphorylated tau tangles. Hearing loss is epidemiologically linked to increased AD risk and accelerated cognitive decline, but mechanisms remain poorly understood. We used gerbils- ideal due to human-like hearing and identical Aβ40/Aβ42 sequences – for modelling. To model AD pathology, we oligomerized human Aβ1-42 and injected it into the lateral ventricle of adult gerbils. After a 4-week recovery period, brains were processed and analyzed via immunofluorescence. We quantified mean fluorescence intensity (MFI) of Aβ (6E10) and phosphorylated tau (p-Tau, AT8) in the hippocampus (HPC), auditory cortex (AC), and parietal cortex (PC). Comparisons were made across groups of normal-hearing controls versus animals injected with Aβ, and normal-hearing controls versus animals with noise-induced hearing loss (NIHL). Our preliminary findings reveal distinct regional effects. Aβ-injected animals exhibited significantly elevated levels of 6E10 expression in all regions compared to controls (HPC: $p<0.05$, AC: $p<0.05$, and PC: $p<0.01$). p-Tau expression was elevated in PC ($p<0.05$) but not in HPC or AC. In NIHL animals, compared to controls, 6E10 expression was significantly higher in AC ($p<0.05$) and PC ($p<0.005$), but not in HPC. Furthermore, p-Tau levels were elevated across all regions in NIHL compared to controls (HPC: $p<0.005$, AC: $p<0.0005$, and PC: $p<0.0001$). The region-specific effects of NIHL on Aβ and p-Tau accumulation, particularly the pronounced increase in p-Tau in auditory-related regions, suggest that chronic sensory deprivation may exacerbate AD pathology in a region-specific manner. Hearing loss thus exacerbates AD -related pathology, particularly in auditory and parietal cortex. Gerbils thus offer a robust model to explore mechanistic link between sensory loss and AD progression.

Poster#4

Effects of developmental dieldrin exposure on inflammasome pathways.

Authors

Perel Rose, Briana De Miranda, Alison I Bernstein

PI Name: Alison I Bernstein

Exposure to the organochlorine pesticide, dieldrin, increases the risk of Parkinson's disease (PD). Since PD occurs later in life, PD-related exposures can accumulate throughout the lifetime long before symptom onset. Two-hit models enable identification of pre-degenerative changes that increase PD susceptibility. Our lab has developed a two-hit model that combines developmental dieldrin exposure with the alpha-synuclein preformed fibril (PFF) model of PD, in which exposure causes a male-specific exacerbation of PFF-induced toxicity. In this model, we identified longitudinal sex-specific epigenetic and gene expression changes in genes related to neuroinflammation that occur before PFF injection, suggesting that epigenetic changes to transcriptional regulation of neuroinflammatory genes could contribute to increased PD susceptibility in our two-hit model. Here, we tested the hypothesis that developmental dieldrin exposure causes dysregulation of neuroinflammatory and inflammasome-related genes, leading to exacerbation of the neuroinflammatory response to synucleinopathy. Starting at 8 weeks of age, female C57BL/6 mice were exposed orally to 0.3 mg/kg dieldrin twice a week throughout mating, lactation, and weaning. At 12 weeks of age, the F1 pups received intrastriatal PFF or saline injections. Male and female mice from independent litters were sacrificed 2 months post-injection, and expression of neuroinflammatory and inflammasome-related proteins (Iba-1, GFAP, IL-1β, ASC) was assessed by immunofluorescent staining. Dieldrin alone did not affect the expression of these proteins, while PFF induced increased IL-1β expression in astrocytes and ASC in microglia. This is consistent with previous reports on neuroinflammatory activation in response to synucleinopathy. Here, we assessed whether dieldrin exposure exacerbates this response.

Supported by T32ES007148, PF-VSA-1477479, NIEHS R01ES031237

Poster #5

Strain and sex effects on behavioral responses to model blast-induced traumatic brain injury.

Authors

Ian M. Biluck, Kathleen E. Murray, Aileigh E. Daniel, Anya E. Mausoof, Tara P. Cominski, Victoria A. Stiritz, Bryan J. Pfister, Kevin D. Beck, Vedad Delic, and Bruce A. Citron

PI Name: Bruce A. Citron

Blast-induced mild traumatic brain injury (bTBI) is one of the most common types of mild TBI experienced by active-duty military personnel. To identify potential genetic influences on the responses to bTBI, we assessed the sex- and strain-dependent neurobehavioral effects of a single 180-kPa blast on C57Bl6/J and 129S1/SvImJ mice using the Morris Water Maze and compared these results to acoustic startle tests. We observed that, at 1-week post-injury, blasted male C57Bl6/J mice displayed a 47% increased swim distance to the target platform ($p<0.005$) not observed in female C57Bl6/J or 129S1/SvImJ mice at the first hidden platform session. During the 2-hour Morris Water Maze probe trial, only female C57Bl6/J mice exhibited a significantly reduced time in the target zone. During acoustic startle testing, female, but not male, C57BL/6J mice exhibited greater prepulse inhbition of the startle response at 1-month post-injury. Testing at 1-month and 3-months post-injury will allow for comparison of strain-and sex-specific chronic phase responses to bTBI. These findings suggest that both sex and genetic differences contribute to a differential response to bTBI on learning and memory neurobehavior. Future studies will aim to identify mechanistic targets that could be exploited to develop therapies for Veterans with bTBI history.

Supported by Department of Veterans Affairs (Veterans Health Administration, Office of Research and Development, Biomedical Laboratory and Rehabilitation Research and Development I01BX005015 (BAC), I01BX004561 (KDB), IK2RX003253 (VD)), a VA Research Career Scientist award IK6BX006188 (BAC), and the Veterans Bio-Medical Research Institute.

Poster# 6

Role of a Potassium Ion Channel Mutant in Neuronal Development.

Authors

Anastasia Balatsky and Federico Sesti

PI Name: Federico Sesti

Potassium channels regulate neuronal excitability and can elicit intracellular signaling cascades in the central nervous system. Specifically, voltage-gated potassium channel subfamily B member 1 (KCNB1) forms complexes with integrins (Integrin K+ channel complexes, IKCs). These complexes convert the electrical properties of the channel into biochemical signals that modulate the regulation of cell proliferation, survival, and migration. In humans, mutations of KCNB1 are associated with epileptic disorders. In particular, a substitution mutation of Arginine to Histidine at position 312 in the KCNB1 gene ($KCNB1^{R312H}$) has been identified in children with developmental and epileptic and encephalopathies (DEEs). Children affected by these disorders present intellectual delays with or without seizures. To investigate this neurological condition, we generated a CRISPR knock-in (KI) murine model harboring the *Kcnb1*^{R312H} gene variant. The R312H KI mouse exhibits a DEEs-like phenotype, including behavioral deficits and spontaneous seizures. In addition, we found that the R312H pyramidal cells display morphological defects (shorter apical dendrites, hyper-arborization and immature spines) that are ameliorated when IKC signaling is rescued *in vitro* via activated integrins. Thus, these results unveil a previously unknown developmental role of KCNB1 through mechanisms that do not depend exclusively on its ionic function.

Supported by NSF (2030348) grant

Poster#7

Autophagy is Required for Nuclear Lipid Droplet Formation in Astrocytes.

Authors

Mohamed Asik Rajmohamed and Ching-On Wong

PI Name: Ching-On Wong

Astrocytes are highly metabolic glial cells that play crucial roles in lipid metabolism and neuroprotection. In response to aging or neuronal stress, astrocytes increase their lipid droplets (LDs) abundance. While LDs are commonly found in the cytoplasm of astrocytes and other glial cells, emerging studies revealed the presence of nuclear lipid droplets (nLDs) in other non-neural cell types. Here, we report that astrocytes also form nLDs under specific metabolic conditions. In human iPSC-derived astrocytes and primary mouse astrocytes, we found that nutrient deprivation, an established inducer of autophagy, robustly promotes nLD formation. Concordantly, inhibiting mTORC1, a negative regulator of autophagy, also enhanced nLD content, demonstrating an interaction between the autophagy pathway and nLD metabolism. Mechanistically, inhibition of autophagosome-lysosome fusion or blocking lysosomal calcium efflux markedly reduced nLD formation, confirming the requirement of autophagic flux for this process. Furthermore, APOE genotype significantly influenced nLD biogenesis, with APOE3 and APOE4 astrocytes exhibiting differential responses to autophagy induction. Together, our findings uncover an hitherto unrecognized aspect of lipid mobilization in astrocytes, and implicate that nLDs may represent an adaptive mechanism that regulates nuclear processes under metabolic stress. Understanding the regulation and function of astrocytic nLDs could open new directions in studying lipid homeostasis and neurodegeneration.

This work was supported by NIH award R01AG081379.

Poster#8

Examining Therapeutic Potential of Lateral Habenula Inhibition for Chronic Pain and Stress-induced hyperalgesia.

Authors

DePasquale, O., Bryant, S., O'Brien, C., Barker, D

PI Name: David Barker

Chronic pain and stress-related disorders frequently co-occur, and maladaptive stress responses are known to heighten pain sensitivity and impede recovery. The lateral habenula (LHb), composed primarily of glutamatergic neurons, regulates aversive learning and affective state through projections to brainstem and midbrain centers that modulate pain perception. Dysregulated LHb activity has been implicated in both stress and chronic pain, suggesting that LHb hyperactivity may represent a common mechanism driving hypersensitivity across these conditions. We hypothesized that targeted inhibition of LHb glutamatergic neurons would reduce hyperalgesia arising from stress, nerve injury, or both. Using chemogenetic inhibition (hM4Di DREADDs), we selectively silenced LHb glutamatergic neurons during stress exposure, following stress exposure, or following a spared nerve injury. In the stress model, silencing LHb neurons during stress prevented the development of stress-induced hyperalgesia (SIH), while inhibition after stress reversed established SIH. In the spared nerve injury model of neuropathic pain, inhibition following nerve injury attenuated both mechanical and thermal hypersensitivity. Together, these findings demonstrate that LHb glutamatergic neuron activity is necessary for the expression and maintenance of hyperalgesia following stress or nerve injury, highlighting the LHb as a potential therapeutic target for interventions addressing both stress-related and neuropathic pain.

Supported by NIH R01-NS134981

Poster #9

Vestibulothalamic relay to the posterior parietal cortex and parietal corticostriatal connectivity suggests a canonical vestibular neuraxis.

Authors

Mark Aziz, Eran Peci, Todd Mowery

PI Name: Todd Mowery

The striatum is the primary input nucleus of the basal ganglia, integrating a dense plexus of inputs from the cerebral cortex and thalamus to regulate action selection and learning. Neuroanatomical mapping of the striatum and its sub compartments have been carried out extensively in rats and mice, nonhuman primates, and cats allowing comparative neuroanatomy studies to derive heuristics about striatal composition and function. Sensorimotor structures involving motor, somatosensory, auditory, and visual senses have been extensively studied; however, little work has focused on the vestibular sensory processing. Our recent study suggested that the vestibular nucleus has an important thalamostriatal relay through the perifascicular nucleus of the thalamus with extensive sensorimotor overlap and a compartmentalized region in the dorsoventral striatum. Here we used retrograde and anterograde AAV to systematically map a second vestibular pathway in the Mongolian Gerbil. We find that this pathway from medial vestibular nucleus to the lateral dorsal nucleus has a thalamocortical input to the posterior parietal cortex. Furthermore, PPC corticostriatal input shows a compartmentalized region in the dorsal lateral striatum and a similar pattern of overlapping input to what we have previously described for the MVN to PF to Striatum pathway. Together these results suggest that this pathway represents a canonical brainstem to thalamus to cortex neuraxis for the vestibular system in rodents and likely other species. This finding will allow novel studies to be carried out involving vestibular contributions to physiology, cross modal plasticity, and behavior.

Supported by NIH DC017163

Poster #10

Greater fidelity of neural patterns during reading is associated with lower frequency and concreteness ratings, and higher orthographic distance.

Authors

Cory McCabe, David Rothlein, Isabella Walsh, Noah Nixon, Lucia Zepeda Rivera, Michaela R. Brooks, William W. Graves, Donald J. Bolger, Jeremy J. Purcell

PI Name: William W. Graves

Representational similarity analyses (RSA) have been used to determine specific brain regions encoding word form and meaning (semantic) features among individuals. Such work does not address the reproducibility of feature-related neural patterns across individuals. We used fMRI and RSA to quantify representational fidelity (RF) -- the degree to which neural patterns are reliably structured across individuals. We hypothesize a relationship between RF and both word-form and semantic features within language processing regions. 32 participants underwent fMRI scanning while reading 160 words presented 4 times. For each participant, a representational similarity matrix (RSM) was generated by cross-correlating the activation patterns within a region of interest (ROI) for each word stimulus. RF was calculated for each word by correlating each participant's RSM with the group average RSM while leaving that participant out. 38 individual atlas-based ROIs representing left-hemisphere language regions were generated. Multiple regressions with RF as the dependent variable were performed for each ROI using word frequency, length, concreteness, and orthographic distance as covariates. ROI analyses revealed significant negative associations between word frequency and RF in the ventral occipito-temporal cortex (vOTC), inferior frontal (IFG), supramarginal (SMG), and middle temporal gyri (MTG). Additionally, there were negative associations between concreteness and RF in the angular gyrus, anterior temporal lobe, posterior cingulate cortex, and MTG. In contrast, there was a positive association between orthographic distance and RF in the vOTC, IFG, SMG, and MTG. By focusing on reproducibility, RF offers a novel approach for examining the neural components of the reading system.

Supported by 2025-2026 Rutgers Brain Health Institute Trainee Travel Award

Poster #11

Epsilon Toxin from Gut Microbiome Clostridium Perfringens Damages Human Oligodendrocytes.

Authors

Irva Patel, Dr. Hiroko Nobuta, Dr. Timothy Vartanian

PI Name: Hiroko Nobuta

Multiple sclerosis (MS) is a chronic demyelinating disorder of the central nervous system (CNS) characterized by areas of neuroinflammation, demyelination and axonal loss. MS causes damage to oligodendrocytes, the myelin producing cells in the CNS. The exact cause of MS remains unknown, with studies highlighting genetic and environmental factors. One potential trigger is Clostridium (C) perfringens's epsilon toxin (ETX). C. perfringens is a gram-positive bacterium which releases ETX, a pore-forming toxin , known to specifically target oligodendrocytes in livestock and murine samples. It is known to cause blood brain barrier disruption and dose and time dependent demyelination in mouse cerebellar slice cultures. Studies have found higher levels of C. perfringens in fecal samples of MS patients compared to healthy controls. However, the effects of ETX on human oligodendrocytes is still unknown. The goal of this project is to understand how ETX impacts human oligodendrocyte morphology and survival. Hence, we treated human oligodendrocyte samples with ETX (0,5,10,20,40 ,100nM) for 2 or 6 hours, and immunofluorescently stained with oligodendrocyte marker myelin basic protein. Images were captured and morphological analysis was conducted by tracing cellular processes (branches). We determined that ETX reduces the total path length of the processes in oligodendrocytes, shifting the individual path length distribution by increasing the short processes and decreasing long processes. Additionally, ETX increased the percentage of pyknotic cells in dose and time dependent manner indicating cell death. These findings imply that ETX alters human oligodendrocyte morphology and induces cell death, consistent with MS pathogenesis.

Poster #12

Striatal Insulin Sensitivity Augmented by Semaglutide in Obese Mice.

Authors

James Muldowney, Miriam Bocarsly

PI Name: Miriam Bocarsly

Obesity affects one third of American adults and is associated with peripheral and central insulin resistance. Insulin has multiple targets in the brain, including in the striatum, a brain region implicated in reward and motivation. Striatal cholinergic interneurons (ChIs) express insulin receptors, inducing dopamine transmission augmentation to regulate feeding behaviors. Glucagon-like peptide-1 (GLP-1) receptor agonists, particularly semaglutide, have gained popularity for weight loss and correction of dysfunctional insulin signaling. Peripheral insulin resistance and impulsive feeding behavior are ameliorated in patients treated with semaglutide. In rodents, semaglutide does not cross the blood brain barrier; therefore, the mechanism of the drug's effects is unknown. Semaglutide's effect on insulin sensitivity in the dopaminergic pathway is yet to be investigated. The long-term goal of this research is to identify significant molecular regulators of dopaminergic signaling alteration in obesity that are remediated by semaglutide treatment. Our preliminary data suggests a role for insulin action on dopamine transmission augmentation in mice subjected to a high fat diet (HFD) and treated with semaglutide. Through fast-scan cyclic voltammetry, we demonstrate that mice fed a HFD and administered semaglutide have increased evoked dopamine concentrations in the striatum. Striatal brain slices of these treated mice also present a significant increase in dopamine release when washed with insulin, an effect absent in HFD-fed, vehicle-treated mice. This data provides the basis for the hypothesis that semaglutide rescues insulin sensitivity in the striatum, increasing evoked dopamine concentrations, and normalizing feeding behaviors in obese mice.

Poster #13

Defining the role of B-Raf and mTOR Signaling in Spinal Cord Oligodendroglia.

Authors

Divyangi Kantak, Meghana Mulpuri, Terri Wood

PI Name: Terri Wood

CNS myelination is carefully orchestrated by multiple extracellular and intracellular signal transduction pathways that regulate the formation of myelin sheath during development and adulthood, maintain myelin and axonal integrity throughout life. Recent studies have shown that Raf/Mek/ERK1/2 (MAPK) and PI3K/Akt/mTOR pathway function autonomously and co-operatively to regulate oligodendrocyte differentiation, myelinogenesis and myelin maintenance. Interestingly, deletion of Raf kinases like B-Raf, direct upstream activators of ERKs in neural precursor cells (NPC) of mice results in a striking hypomyelinating and neurodegenerative phenotype and hinders OPC differentiation. Mice lacking mTOR in oligodendrocytes display significantly hypomyelinated spinal cord axons along with impaired oligodendrocyte maturation. However, how B-Raf and mTOR coordinately regulate myelination remains unexplored.

To elucidate the function of B-Raf and/or mTOR in oligodendroglia, we developed a rodent model in which Braf and/or Mtor floxed sequences are conditionally deleted in OPCs by Cre recombinase expression utilizing the CNPase (2',3'-Cyclic nucleotide 3'-phosphodiesterase) promoter. Gene expression analysis revealed significant downregulation of mRNA levels of several myelin proteins crucial for myelin sheath formation and axonal ensheathment in spinal cords of young adult mice lacking Braf or Braf;Mtor. We also observed fewer number of myelinated axons in the spinal cords of 12mo mice. Additionally, mice lacking both Braf and Mtor in oligodendroglia showed more severe motor deficits than mice with individual gene deletions. Our analyses aim to assess the impact of mTOR and/or B-Raf deletions on developmental and adult myelination and the mechanism of interaction between the two signaling pathways.

Supported by R37 NS082203 and BHI-trainee travel award 2025-2026 by Neurodevelopment FAWG

Poster #14

p75NTR mediates retrograde degeneration of Basal Forebrain Cholinergic neurons after cortical traumatic brain injury leading to long-term cognitive deficits.

Authors:

Mansi A. Pandya, Michael W. Shiflett, Wilma J. Friedman

PI Name: Wilma J. Friedman

Basal forebrain cholinergic neurons (BFCNs) extend long projections to multiple targets in the brain to regulate cognitive functions such as memory, and learning, and are compromised in numerous neurodegenerative disorders. We have previously demonstrated that moderate injury to the cortex elicits retrograde degeneration of afferent BFCNs in vivo. TBI promoted a significant increase in proneurotrophins in the damaged cortex, leading to extended local damage in the penumbra of the injury, as well as retrograde loss of BFCNs ipsilateral to the injury via the p75 neurotrophin receptor(p75NTR). While the loss of cortical neurons in the penumbra of the injury occurs within days following TBI, retrograde loss of BFCNs occurs over 1-2 weeks following the injury. To investigate whether there are functional deficits specifically associated with the loss of the BFCNs, we developed p75fl/fl:ChAT-cre mice to specifically delete p75NTR from the cholinergic populations. These mice were subjected to fluid percussion injury to the cortex. The p75fl/fl:ChAT-Cre mice showed sparing of basal forebrain neurons compared to WT mice, despite having similar cortical damage. Using the Barnes maze and operant conditioning to assess the acquisition of BFCN-specific cognitive functions, we observed that in mice with bilateral cortical TBI, the WT mice suffered long-term cognitive deficits which were not seen in the p75fl/fl:ChAT-cre mice. The knowledge of how the loss of BFCNs after TBI subsequently affects cognitive functions can help further our understanding of functional deficits due to TBI and neurodegeneration suggest that there may be functional benefits to sparing these neurons following TBI.

This study was supported by NIH/NINDS grant 1R01NS12789, and Rutgers Brain Health Institute

Poster #15

bioRNNS: toward more biologically plausible neural network models of the brain.

Authors

Ahmad Beyh, Jason Kim, Linden Parkes

PI Name: Linden Parkes

Recurrent neural networks (RNNs) are powerful tools for modeling neural dynamics, but standard architectures often lack biological realism in two key aspects: their all-to-all connectivity pattern where every node processes inputs and outputs directly, and their unconstrained hidden layer formation which can lead to biologically unrealistic architectures. We address these limitations by developing biophysical RNNs (bioRNNs) with two novel constraints: (1) input/output masking that restricts inputs to "sensory cortex" nodes and outputs to "association cortex" nodes, mirroring biological information flow, and (2) biophysical embedding based on distances along the sensory-association axis, which represents the functional gradient from sensory to higher-order cognitive regions. Our bioRNNs demonstrated more brain-like neural dynamics in their hidden layer timescales compared to standard RNNs. Furthermore, in the presence of I/O masking, bioRNNs outperformed standard RNNs across various behavioral tasks. These findings suggest that incorporating biophysical constraints in RNNs can create more realistic and interpretable brain models, enhancing their utility for both basic and clinical neuroscience research.

This study was supported by NIMH; BHI Travel Award

Poster #16

Altered Bioenergetic Profiles in Neurons with Frontotemporal Dementia-Associated MAPT Mutation.

Authors

Yan Tong, Anwar Nakhla, Christina Mansour, Alyssa Kenny, Rushi Makadia, Ching-On Wong

PI Name: Ching-On Wong

Frontotemporal dementia (FTD) is a neurodegenerative disorder affecting people in midlife or earlier. Dysregulated brain metabolism, including glucose hypometabolism, is a common hallmark of FTD, but how altered glucose metabolism impairs neuronal function remains unclear. Glucose oxidation via glycolysis is essential for meeting the bioenergetic demands of neuronal processes. Here, we investigated whether and how bioenergetics is disrupted in neurons carrying FTD-associated genetic variants. We employed human induced pluripotent stem cell-derived neurons (iNeurons) carrying *MAPT*^{R317W} alongside primary neurons isolated from *MAPT*^{P301L} *Drosophila* as cellular FTD models. Real-time cytosolic ATP levels in neuronal somata were measured using ATP biosensor PercevalHR in response to metabolic inhibitors. In healthy control neuronal somata, resting ATP production primarily relies on glycolysis. In contrast, *MAPT*^{P301L} and *MAPT*^{R317W} neurons exhibited reduced glycolytic reliance with a compensatory increase in oxidative phosphorylation–dependent ATP production. Healthy neurons acutely switched to other fuel source to compensate for ATP loss upon glycolytic inhibition, leading to our ongoing investigation of this metabolic flexibility changes in FTD neurons. Our findings demonstrate that impaired glycolytic activity reprograms FTD neuronal bioenergetics. We next tested whether promoting neuronal glycolysis could alter organismal outcomes in FTD models. In *Drosophila* FTD model expressing neuronal *MAPT*^{P301L}, both median lifespan and locomotor activity were significantly diminished. Overexpression of the glycolytic enzyme phosphofructokinase significantly extended survival in FTD flies, suggesting that neuronal glycolysis can be targeted to alleviate disease phenotypes. Altogether, our results provide new insights into metabolic dysfunction in FTD and suggest glycolysis restoration as a potential therapeutic strategy.

This work is supported by start-up fund from Rutgers and the NIH grant R01AG08137

Poster #17

Mapping morphine-induced changes of global protein phosphorylation in brain regions of a mouse tolerance model using tandem mass tag-based phosphoproteomics/proteomics.

Authors

Raymond Chien, Ayma F. Malik, Jin Xu, Guoan Zhang, Ying-Xian Pan

PI Name: Ying-Xian Pan

Mu opioids like morphine, fentanyl, and oxycodone remain central in managing moderate-to-severe pain despite side effects. Mu opioid tolerance can promote addiction and respiratory depression, significant factors in opioid use disorder and overdose deaths. Molecular mechanisms, especially posttranslational ones, are still unclear. In this study, we developed a long-term morphine tolerance model with twice daily administration (10 mg/kg, subcutaneously) for 6 weeks in C57BL/6J (B6) male mice. The results revealed that morphine tolerance increased linearly over the first 3 weeks and was sustained with a slight decrease after 3 weeks. This scenario reconciles the clinical observation that with extended dosing, morphine tolerance is progressively developed at the beginning and then stabilized for a relatively long time in cancer patients. Using tandem mass tag-based phosphoproteomics/proteomics, we unbiasedly mapped morphine-induced changes of global protein phosphorylation in five brain regions under this morphine tolerance paradigm. Our results showed that morphine can induce changes in many phosphoproteins, some of which were time-dependent or region-specific. Pathway analysis revealed shared and unique signaling pathways and identified several key regulators like kinases and ion channels, which likely are the causal factors for morphine tolerance. These results provide novel insights into the molecular mechanisms of morphine tolerance.

Supported by *funds from NIDA; Department of Anesthesiology, New Jersey Medical School; Brain Health Institute, Rutgers; The Mayday Foundation*

Poster #18

The latent-cause framework for understanding addiction: A focus on the orexin/hypocretin system.

Authors

David De Sa Nogueira and Gary Aston-Jones

PI Name: Gary Aston-Jones

Relapse after extinction is strongly influenced by contextual memory. The latent-cause inference framework proposes that during extinction, individuals must decide whether new information belongs to the same latent cause as prior learning or reflects a distinct one. When training, extinction, and testing occur in the same context (AAA), animals are more likely to infer a single latent cause, reducing renewal. In contrast, extinction in a different context (ABA) favors formation of a new latent cause, promoting renewal.

The orexin/hypocretin system modulates motivation and memory processes and projects strongly to the hippocampus, including CA3, an area crucial for contextual representation. We hypothesized that orexin signaling biases latent-cause inference during extinction learning.

Rats self-administered sucrose in context A, underwent extinction in either A (AAA) or B (ABA), and were tested for renewal in A. Pharmacological blockade of orexin receptor 1 (OxR1) with SB-334867 during extinction increased renewal selectively in AAA animals, without affecting locomotion. These results suggest that OxR1 signaling shapes how extinction memories are assigned to latent causes.

To test pathway specificity, we used chemogenetics (AAV-hM3Dq-mCherry) in orexin-Cre rats to selectively stimulate orexin terminals from the lateral hypothalamus to hippocampus CA3. Activation of LH→CA3 orexin projections decreased renewal in AAA animals, with no effect in ABA animals, indicating that orexin signaling supports updating of an existing latent cause representation.

These results reveal a circuit-specific mechanism through which orexin regulates latent-cause inference, offering a novel target for reducing relapse-like behavior.

Supported by P50MH-136296

Poster #19

Perceived Stress in Autistic vs Non-Autistic Adolescents

Authors

Tatyanna Landell, Mariella Vargas, Brinda Pusuloori, Elena Chauhan, Tejaswini Venkat, Xiangyu Tao, María Eugenia Contreras-Pérez, Casey Cragin, Anthony Spirito, Stephen Sheinkopf, Kristina Jackson

PI Name: Kristina Jackson

Autistic adolescents may be more vulnerable to stress than their peers because of neurological differences in coping. The Perceived Stress Scale (PSS) has been widely adopted in the general population, but it has not been validated among autistic individuals.

This poster aims to compare the factor structure of the PSS items between two separate samples: 111 autistic (Mage = 16.46, SD = 1.17) and 302 non-autistic adolescents (Mage = 16.21, SD = 0.767). All items were used in factor analyses to determine whether autistic and non-autistic adolescents responded similarly to the PSS items. A two-factor model was confirmed for both samples (configural model: CFI = .93, TLI = .91, RMSEA = .09), and the partial metric invariance was established after freeing one item ("*how often you have been able to control irritation in your life*"), which showed a substantially weaker loading among autistic adolescents (.34) compared with non-autistic adolescents (.71), $\Delta\chi^2(8) = 23.69$, $p = .003$.

The results suggest that perceived stress is relatively similar among both samples, but there is a notable difference in how they respond to irritability. Prior research has documented a positive correlation between irritability and sensory differences in autistic populations, suggesting that atypical sensory processing may contribute to this variability. Additionally, frontostriatal circuitry, implicated in irritability and ASD, may underlie these differences. Future research should examine the neural and sensory mechanisms associated with irritability to clarify its role in stress processing and identify potential intervention targets for autistic adolescents.

Supported by R01 AA029008-05

Poster #20

AAV-Mediated BDNF and GAS6 Muscle Delivery Delays Disease Onset in SOD1G93A ALS Mice

Authors

Yicong Le, Gongjie Liu, Shenzhe Wu, Marialaina Nissenbaum, Alexander W. Kusnecov, Philip Furmanski, Raymond B Birge, Renping Zhou

PI Name: Renping Zhou

Amyotrophic Lateral Sclerosis (ALS) is a fatal neurodegenerative disease, with limited treatments. Gene therapy offers an alternative strategy for treating a large portion of ALS patients, however, the disparate genetic alterations in ALS complicate the development of gene therapies. Tyrosine receptor kinase B (TRKB) and Tyro3 receptors are highly expressed in mouse spinal cord motor neurons, suggesting that their ligands, brain-derived neurotrophic factor (BDNF) and growth arrest-specific 6 (GAS6), respectively, are crucial for neuronal survival. In this study, we tested whether genetically induced and muscle tissue-specific expression of such survival-enhancing ligands would ameliorate symptom development in the $SOD1^{G93A}$ ALS mouse model. The therapeutic vectors (AAV-P_{mus}7-HuBDNF-teLuc or AAV-P_{mus}7-HuGAS6), or a control vector (AAV-P_{mus}7-teLuc) were injected intravenously via the retro-orbital route and intramuscularly into the hindlimb skeletal muscle of six-week-old mice. Treatment with the therapeutic vectors delayed disease onset and slowed progression in both male and female mice. Interestingly, a sex-specific response was observed, with female mice benefiting more from the treatments than males. Lumbar motor neuron survival was more sustained in the therapeutic vector-treated group compared to control vector group. No statistically significant extension of lifespan was observed in the treated groups.

Supported by Panathera. INC

Poster #21

Neural mechanisms underlying social behavior susceptibility to systemic inflammation.

Authors

Paula Diaz-Munoz, Hunter T. Lanovoi, Genesis Cedeno, Justin S. Riceberg, Ioana Carcea

PI Name: Ioana Carcea

Inflammation contributes to the onset and severity of many psychiatric disorders, especially those with a social dimension. In mammals, inflammation reshapes social choices based on pre-existing relationships, yet the underlying mechanisms remain unclear. Here, we tested whether hierarchical social relationships gate susceptibility to the immunogen lipopolysaccharide (LPS) and what interoceptive mechanisms mediate this effect. We ranked adult male and female C57BL/6N mice (8–10 weeks) via the tube test and treated them with LPS (0.5 mg/kg) or saline. Two hours later, we measured social novelty preference in a three-chamber assay. LPS caused dominant but not subordinate males to increase investigation of familiar cagemates ('dominant LPS' vs 'dominant saline', $p < 0.0001$; 'subordinate LPS' vs 'subordinate saline', $p = 0.415$; $N = 39$). We found no effect in females. In a separate cohort, immediate early gene mapping revealed LPS activation of the capsular nucleus of the central amygdala (CeC) in dominant ($p = 0.0085$, $N = 9$) but not subordinate males ($p = 0.104$, $N = 10$); ~40% of activated neurons expressed the oxytocin receptor (OTR). CeC-OTR$^+$ cells integrate visceral signals and social information, making them prime candidates for mediating rank-dependent shifts in social preference during inflammation. Using fiber photometry, we found that saline-treated dominant mice increased CeC-OTR$^+$ activity near novel mice, whereas LPS reversed this. Subordinates showed no consistent activity patterns. These data demonstrate that CeC-OTR$^+$ neurons encode rank-dependent social preference changes under systemic inflammation, identifying CeC oxytocin signaling as a key mediator of social susceptibility to inflammation.

Supported by NIH R01MH128688

Poster #22

Duel-edged Dosing: Dexamphetamine and Lisdexamfetamine Suppress Binge-like Eating but Disrupt Sleep in Female Rats.

Authors

Joel S. Raymond, Avi D. Desai, Theresa S. Joseph, Saanvi S. Narava, Abanoub J. Armanious, and Morgan H. James

PI Name: Morgan H. James

Binge eating disorder (BED), characterized by recurring episodes of excessive, rapid-paced food consumption, is often comorbid with insomnia. The only FDA-approved BED medication, lisdexamfetamine (LDX), converts to dexamphetamine, suppressing binge eating but potentially worsening sleep if taken in the evening—when most binge eating occurs. No prior studies quantified sleep disruption at binge suppressing doses. Adult female Long Evans rats were implanted with wireless telemetry probes to record electroencephalographic and electromyographic data. Polysomnographic (PSG) recordings were scored for wake, NREM, and REM sleep. Binge-like eating was measured using an intermittent access model, where rats with *ad libitum* chow received a sweetened fat mixture (8.6 kcal/g) for 30 min twice weekly. Dexamphetamine hemisulfate (0-1.5 mg/kg) and LDX mesylate (0-3.371 mg/kg) were administered intraperitoneally. For sleep assessment, 19-h PSG recordings were conducted from ZT23 to ZT18, with drug administration at ZT23. For binge-like eating suppression, drugs were administered 30 min before testing (ZT20-22). Dexamphetamine and equivalent LDX doses dose-dependently suppressed binge-like eating and disrupted sleep. At the lowest effective doses, both drugs markedly delayed sleep and REMS onset, increased wake time, and suppressed NREM and REM sleep during the first 3 hours of the light phase, with minimal to no rebound sleep recovery. Our findings confirm clinical reports of significant iatrogenic sleep disturbances induced by LDX and its active metabolite when used to suppress binge eating. These results highlight the need for novel treatments that curb binge eating without disrupting sleep.

Supported by NIDA (R01 061303 and R00 045765 – MHJ/GAJ); NIEHS (R21 035838 – MHJ); New Jersey Health Foundation Inc.; American Australian Association

Poster #23

Genetic Liability for Substance Use Explains Variations in Suicidal Ideation and Behaviors: Findings from the Adolescent Brain Cognitive Development Study.

Authors

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PI Name: Jill A. Rabinowitz

Suicide ideation (SI) and attempt (SA) remain a public health crisis and are associated with substance use, particularly among adolescents. However, less is known about potential links between genetic liability for substance use disorders (SUDs) and suicidal thoughts and behaviors (STBs) during adolescence. We examined whether genetic liability for SUDs, indexed via polygenic risk scores (PGS), were associated with passive SI, active SI, and SA using data drawn from the Adolescent Brain Cognitive Development Study. Participants (N= 4,797, *M*age at baseline=9.47; 47% female) were children genetically similar to reference panels of European ancestry. Structural equation modeling was used to incorporate data from child- and parent-reported suicidality. The outcome was a latent variable capturing all variance in the child report and shared variance in the parent report. PGS for SUD were derived from a large genome-wide association study of common liability to SUD. Lifetime STBs were assessed via child and parent report at baseline and two annual follow-up assessments, which were collapsed across time. The SUD PGS was significantly positively associated with active SI (β=0.10, 95%CI [0.04,0.16]), but not with passive SI (β=0.02, 95%CI [-0.03,0.07]) or SA (β=0.08, 95%CI [-0.02,0.18]). Findings indicate that greater genetic liability for SUDs is associated with increased risk for active SI specifically, underscoring the importance of shared genetic propensities for SUDs as a risk factor for this suicidal phenotype as early as childhood. Future research would benefit from expanding the analysis across different ancestral groups to determine whether the generalizability of study findings to other populations.

Supported by NIDA (R01DA057552) to Drs. Rabinowitz, Maher, and Felton, and Rutgers Brain Health Institute

Poster #24

Neuropilin-2 Receptor Promotes Dorsal Spinal Cord Progenitor Survival by Maintaining Apical Cell Adhesion and Basal Lamina Stability.

Authors

Adefemi C. Baderinwa, Victor Danelon, Shifa Zahir Hussain, Valerie Chuquino, Michael Bailey, Tracy S. Tran

PI Name: Tracy S. Tran

Neuropilin-2 (Nrp2) is a receptor for class 3 secreted semaphorins (Sema3s), known for their role in axon guidance. While studies have implicated Nrp2 in neuronal development, its role in progenitor development and maintenance remains unexplored. Here, we identified a novel role for Nrp2 in regulating the survival of a subset of spinal cord neural progenitors expressing the transcription factor Neurogenin1 (Ngn1). These progenitors belong to the dorsal progenitor 2 (dP2) domain, which gives rise to dorsal interneuron 2 (dI2), a commissural neuron population essential for motor coordination. Using the *Ngn1:tau-mCherry* reporter mouse line, we assessed dP2 number and survival in the *Nrp2* knockout (*KO*) embryo at E9.5 and E10.5. We observed a significant reduction in dP2 number in *Nrp2KO* embryos at E9.5, when these cells first emerge**.** The reduction persists and is accompanied by increased apoptosis at E10.5. At E10.5, progenitor apical endfeet at the ventricular surface is thinner compared to the controls, suggesting disrupted cell-cell adhesion. Consistently, N-cadherin and β-catenin levels are reduced, with decreased colocalization at dP2 apical endfeet, indicating adherens junction (AJ) impairment. Additionally, the fibronectin network at progenitor basal endfeet is reduced and disorganized, indicating basal lamina instability. Lastly, *Nrp2KO* embryos exhibit elevated matrix metalloproteinase-7 (MMP7) levels at both apical and basal surfaces. Known to remodel extracellular matrix (ECM) and degrade N-cadherin, MMP7 may contribute to the observed adhesion and ECM defects. Our findings suggest that Nrp2 supports commissural dI2 neuron development by maintaining their progenitor pool through the preservation of cell adhesion and ECM stability.

Supported by NSF (1556968, 203486), NJCSCR (CSCR16IIRG013), NSF STEM (1905142) and NIH 5T32GM140951-02

Poster #25

Neural progenitor proliferation and oligodendrogenesis mediated by the p75 neurotrophin receptor in the adult rat subventricular zone.

Authors

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PI Name: Wilma J. Friedman

The timing of postnatal neuro- and gliogenesis in the developing brain is a highly regulated process through which the correct balance between proliferation and differentiation is crucial to proper neural development and maintenance through adulthood. Most postnatal oligodendrocytes are generated from neural progenitors residing in the subventricular zone (SVZ), the germinal niche surrounding the lateral ventricles. Here, we demonstrate that the p75 neurotrophin receptor (p75NTR) is expressed by oligodendrocyte-lineage cells in the rat SVZ postnatally and throughout adulthood where its expression corresponds to proliferating intermediate neural progenitors. Whereas the p75NTR is known to initiate apoptotic signaling after brain injury, its expression in proliferating neural progenitor cells in the adult SVZ suggests that it may serve a different role in their maintenance and availability throughout life. We have previously found that rats lacking p75NTR show dysregulated oligodendrocyte maturation throughout early postnatal development. Additionally, we observe that p75NTR is expressed in immature oligodendrocyte-lineage cells within demyelinated lesions of the adult rat. We are investigating the long-term consequences of deleting p75NTR on oligodendrocyte maintenance, and how this affects the cellular composition of the SVZ and the capacity for remyelination throughout adulthood.

Supported by NIH/NINDS 1R21NS137258

Poster #26

Anatomical Patterns Constrain Functional Dynamics in Corticothalamic Systems.

Amber Howell, Carrisa Cocuzza, Avram Holmes, & Linden Parkes

PI Name: Linden Parkes/Avram Holmes

The thalamus is a critical nexus for information transmission, yet the link between its architecture and corticothalamic system dynamics is not fully understood. In humans, association cortical areas project more diffusely within the thalamus relative to sensorimotor areas. Additionally, animal models also suggest that cortical areas dynamically recruit thalamic subpopulations. These findings suggest a critical relationship between the topography of corticothalamic projections and spatial variability in the time-varying properties of human brain function.

We used diffusion and functional MRI data from healthy adults (n=820) to test if thalamic anatomical connectivity patterns relate to more variable and complex functional dynamics at rest. To test this, we used tractography to identify thalamic voxels with the strongest anatomical connections to each cortical area. We then calculated the Euclidean Distance (ED) between these voxels to quantify how spread out they are. Additionally, we leveraged measures of functional variability, entropy, and timescale to quantify the functional dynamic properties of cortical areas and thalamic voxels. We then compared each cortical area's ED values to its own functional properties and the functional properties of its targeted thalamic voxels.

We found that cortical areas with more diffuse connections showed more variable and complex functional dynamics at rest. Furthermore, these areas exhibited more homogenous functional connectivity across the thalamus and preferentially projected to thalamic voxels with more variable intrinsic dynamics and longer timescales. These results demonstrate that the spatial patterning of corticothalamic anatomy fundamentally constrains the dynamic nature and flexibility of their functional interactions.

Poster #27

Purkinje cell development, survival, and adaptive motor behavior in mice require the redundant function of the small GTPases Rab11a and Rab11b.

Authors

Jack DeLucia, Edward Martinez, Haniya Naveed, Michael W. Shiflett, Tracy S. Tran

PI Name: Tracy S. Tran

Cerebellar Purkinje cells (PCs) form the core of cerebellar circuitry, both as the sole outputs of the cerebellar cortex and the chief organizers of cerebellar development. A critical function necessary in development is the trafficking of signaling proteins between membrane compartments. Rab11, a member of the Rab family of small GTPases, resides within the recycling endosome and is responsible for trafficking signaling proteins to and from the plasma membrane in addition to its many other cellular functions. Mutations in *RAB11A* and *RAB11B* produce severe neurodevelopmental disorders in humans, including ataxia, learning disabilities, and cerebellar hypoplasia. However, the role Rab11 plays in the development and function of cerebellar PCs is unknown. We show that specific loss of *Rab11a* and *Rab11b* in PCs (*Rab11-PdKO*) causes drastic loss of these cells at P21, which increases in severity by adulthood. *Rab11-PdKO* cerebella have a reduced whole cerebellar area, internal granule layer area, and molecular layer area. Surviving PCs in the *Rab11-PdKO* cerebella are often highly deformed with misoriented dendritic arbors. The direct inputs onto PCs from Climbing fibers shown by the immunolabeling of vesicular glutamate transporter 2 positive (vGluT2+) puncta are reduced in all lobules. Foliation defects, including additional or absent lobules, are also seen in some *Rab11-PdKO* cerebella. Additionally, *Rab11-PdKO* have dramatically impaired motor coordination and learning as measured by the accelerating rotarod test. Together, our data demonstrate the critical role of Rab11a and Rab11b in the development, survival, and function of cerebellar PCs and the behavioral output of the animal.

Supported by NSF/IOS (1556968, 2034864), the NJCSCR (CSCR16IIRG013), NJ Governor's Council for Medical Research and Treatment of Autism (CAUT17BSP022) , NJ Governor's Council for Medical Research and Treatment of Autism Graduate Student Research Fellowship (CAUT22AFP008)

Poster #28

Modulation of Intersectin Condensate Material Properties in Live Cells.

Authors

Yuzhou Xia, Huan Wang, Jinying Wang, Zheng Shi

PI Name: Zheng Shi

Intersectin-1 (Itsn1), a multi-SH3-domain scaffolding protein critical for endocytosis and vesicle recycling, has recently been linked to Parkinson's disease (PD). Lower expressions or defective forms of Itsn1 can make PD symptoms worse and cause the disease appear earlier. Itsn1 form liquid condensates that colocalize with various neuronal proteins. However, the material properties of Itsn1 condensates and how they are regulated in cells remain unclear. Here, we use micropipette aspiration and whole-cell patch-clamp (MAPAC) and fluorescence microscopy to quantify the viscoelasticity of Itsn1 condensates in HEK293T cells. We found that condensates of Itsn1-(SH3)AE domain are significantly more solid than those of full length Itsn1. Interestingly, the presence of neuronal proteins α-synuclein, synapsin-1, and tau all fluidize Itsn1-(SH3) AE condensates, while exhibiting varying levels of condensate partitioning.

Our findings suggest that synaptic proteins can tune Itsn1 condensate mechanics through specific domain–domain interactions, providing a physical mechanism by which condensates contribute to synaptic regulation and PD pathology.

Supported by National Institute of General Medical Sciences of the National Institutes of Health grant R35GM147027

Poster #29

Spatial metabolomics and quantitative proteomics of brain and other samples using Synapt XS and timsUltra AIP at the Center for Advanced Metabolomics & Proteomics Research (CAMPR)

Authors

Ariful Islam, Tong Liu, Hong Li

PI Name: Hong Li

Explore molecular complexity of your challenging samples with the integrated 'omics services at the Center for Advanced Metabolomics & Proteomics Research (CAMPR). We are providing services for spatial metabolomics and advanced proteomics to provide information required for cutting-edge discovery, particularly in complex, heterogeneous tissues like the brain. CAMPR offers a powerful, multi-platform approach designed to overcome limitations of traditional bulk analysis.

We have Waters Synapt XS mass spectrometer with DESI imaging (DESI-MSI) to provide spatial metabolomics service. DESI-MSI is non-destructive, and can generate high-resolution molecular maps, revealing the in situ distribution of hundreds of lipids, amino acids, TCA cycle intermediates and other metabolites directly within your sample tissue sections.

We also have Bruker timsUltra AIP for ultra-sensitive quantitative proteomics. With trapped Ion Mobility Spectrometry (tims) and the innovative Athena Ion Processor (AIP), this instrument delivers unparalleled proteome depth and high-throughput quantification from the most sample-limited materials, such as single-cell fractions or micro-biopsies.

With the state-of-the-art MS instrument, CAMPR is offering advanced metabolomics and proteomics research, enabling accelerated biomarker discovery and providing crucial, spatially-resolved insights into disease mechanisms and cellular communication networks. Contact us to discuss your project's analytical needs.

Poster #30

Reducing neuronal nitric oxide synthase (nNOS) expression in the ventromedial hypothalamus (VMH) increases body weight and blood glucose levels while decreasing body temperature in male mice.

Authors

Tyler Kays, James Muldowney, Gwyndolin Vail, Rumi Oyama, Pallabi Sarkar, Vanessa Routh

PI Name: Vanessa Routh

The ventromedial hypothalamus (VMH) raises blood glucose during hypoglycemia and exercise, while restraining glucose levels at rest. VMH glucose-inhibited (GI) neurons are critical for restoring euglycemia following insulin-induced hypoglycemia. VMH neuronal nitric oxide synthase (nNOS) is required to activate VMH GI neurons in low glucose and to raise glucose after hypoglycemia. Thus, we originally hypothesized that VMH nNOS neurons comprise the circuit that raises blood glucose and that reducing VMH nNOS expression would lower blood glucose at rest. Surprisingly, reducing VMH nNOS in female mice using nNOS shRNA increased body weight, adiposity, and blood glucose while decreasing locomotor activity. These data suggest that VMH nNOS neurons may play a role in both raising blood glucose during metabolic need and restraining glucose levels at rest. Next, *we hypothesized the same effect would be seen in males*. As in females, VMH nNOS shRNA increased body weight (23%) and adiposity (47%) in males, with an associated increase in blood glucose. However, in contrast to shRNA injected females, locomotor activity was unchanged in shRNA injected males. Instead, body temperature decreased by 0.44^{o}C. In conclusion, reducing VMH nNOS expression in both males and females increases body weight, adiposity and blood glucose. However, the underlying mechanism appears to be sex specific. That is, in females decreased activity may drive the increased body weight and blood glucose, whereas in males these effects may be mediated by decreased thermogenesis. These data are important to understand the sex differences in energy homeostasis.

Supported by NIH R01 DK135857

Poster #31

Perinatal IL-6 elevation changes hippocampal plasticity and memory via postsynaptic mechanisms.

Authors

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PI Name: *Ozlem Gunal*

Autism spectrum disorder is characterized by social deficits with underlying hippocampal dysfunction. Studies have demonstrated that maternal infections stimulate interleukin-6 (IL-6) production, which can cross the placenta and fetal blood brain barrier. We have injected male and female mice with PBS or with 75 ng IL-6 twice daily, from postnatal days 3 - 6 to model increased IL-6 at the end of the second trimester of human development. Here we show that when tested at 7 weeks of age, the IL-6 treated males spent more time exploring an object at a new location compared to the controls, while the IL-6 treated females showed no preference. The paired associative learning task showed that IL-6 treated male mice learned and remembered the spatial location of three different visual stimuli at the same pace as the controls. After inducing long-term potentiation (LTP) in the CA1, only the IL-6 injected male mice showed an increase in the acquisition of LTP, with no sustained change in the LTP maintenance. Male, but not female mice, showed a larger and sustained depression of the field EPSP after long-term depression induction. In addition, the input/output function of IL-6 injected male mice was decreased compared to control indicating that the increased synaptic plasticity is caused by post-synaptic and not pre-synaptic changes. Total apical dendritic spines were reduced in the CA1 region of the IL-6 treated males with an increase in thin spines. Altogether, these data show that a short increase in IL-6 causes lasting changes in hippocampal synaptic plasticity and function.

Supported by HD113311

Poster #32

Retinal Regulation of Locus Coeruleus: A Chemogenetic Approach to Treat Neurodegenerative Disorders.

Authors

Sarah.I. Delcourte, G Crozier, G. Aston-Jones

PI Name: Gary Aston-Jones

Alzheimer's Disease (AD) is the most common dementia, with early pathology linked to tau protein accumulation in the locus coeruleus (LC). While direct LC activation rescues cognitive deficits in animal models [1], its deep brainstem location limits clinical applications. The suprachiasmatic nucleus (SCN) indirectly influences LC via the dorsomedial hypothalamus (DMH) [2], integrating light information through the Photic Regulation of Arousal and Mood (PRAM) pathway (retina → SCN → DMH → LC) [3]. We tested whether PRAM activation could mitigate cognitive deficits in Tg-F344 AD rats.

6 mo Tg-F344 and WT rats received intravitreal AAV injections encoding excitatory Gq DREADD or control virus. Learning and memory were assessed three months later using Morris Water Maze. DREADD activation via clozapine-N-oxide (2 mg/kg, i.p.) was administered 30 minutes before acquisition, referral, and/or reversal sessions. Electrophysiological and Fos analyses confirmed increased activity in retinal ganglion cells, SCN, DMH, and LC. Only female Tg-F344-AD rats exhibited learning deficits at 9 months, which improved with six days of retinal DREADD stimulation. PRAM activation also enhanced memory recall during referral.

PRAM activation during acquisition influenced reversal learning. Rats showed impaired reversal performance, due to enhanced memory of the original platform location, interfering with new learning. Structural equation modeling confirmed this effect. When adjusting for acquisition effects, analysis revealed beneficial PRAM effects during reversal trials, indicating PRAM stimulation improves cognitive flexibility but can be masked by prior acquisition-phase stimulation.

These findings suggest PRAM-induced LC activation attenuates AD-related learning deficits, offering a minimally invasive therapeutic avenue for AD.

Supported by PHS grant R21-MH121723-S1

Poster #33

Sex specific pathological mechanisms of motor dysfunction in Familial Danish Dementia.

Authors

Arnab Choudhury, Teesta Naskar, Eric Aronowitz, Tao Yin, Ruben Vidal, Luciano D'Adamio, Jonathan Dyke, Hyung Jin Ahn

PI Name: Hyung Jin Ahn

Familial Danish Dementia (FDD) is a rare autosomal dominant neurodegenerative disorder caused by a mutation in the integral membrane protein 2B (ITM2b) gene. Clinically, FDD presents cerebral amyloid angiopathy (CAA), cerebellar ataxia, and dementia, sharing neuropathological features with Alzheimer's disease (AD), including CAA, neuroinflammation, and neurofibrillary tangles. In this study, we investigated the mechanisms linking CAA, cerebellar white matter damage, and motor dysfunction using a knock-in (FDD-KI) rat model carrying the Danish Itm2b mutation along with a humanized App gene sequence. FDD-KI rats exhibited age-dependent vascular Danish amyloid (ADan-CAA) and Aβ (Aβ-CAA) deposits in cerebellar vessels, mirroring human FDD pathology. Bulk RNA-seq analysis of 13-month-old FDD-KI rat cerebella revealed sex-specific gene expression signatures, with male rats showing dysregulation of pathways related to Rho-GTPase signaling, ribosomal quality control, rRNA processing, protein translation, and serotonin receptor signaling. Upstream regulator analysis identified inhibited β-estradiol, estrogen, and ESR1 signaling, suggesting hormonal modulation of the downregulated genes found in male cerebellum. Neuropathological analysis revealed greater ADan deposition and elevated CD11b-positive microglial activation in male than female cerebellum. behavioral analysis revealed male-specific motor dysfunction and abnormal gait compared to female FDDKI rats, indicating sex specific deficit. Correspondingly, MRI analysis showed increased T2-weighted signals in the genu of the corpus callosum of male FDD-KI rats, consistent with enhanced microglial activation and demyelination, indicating myelin damage. Collectively, these findings suggest that *Itm2b* gene mutation drives sex-specific alterations in gene expression, amyloid deposition, neuroinflammation, and myelin integrity, contributing to motor and gait deficits in FDD.

Supported by NIH RF1AG078245, R01NS104386, BHI travel grant 2025

Poster #34

Autism- and Schizophrenia-Associated 3q29 CNV Deletion Drives Transient Hyperproliferation of Neural Precursor Cells During Cortical Neurogenesis.

Authors

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PI Name: Emanuel DiCicco-Bloom

The 3q29 deletion is a rare copy number variant that increases risk up to 36-fold for autism spectrum disorder and ~40-fold for schizophrenia. Affected individuals often present with delayed development, intellectual disability, and reduced head circumference. To model this, we generated a CRISPR-engineered 3q29 deletion (3q29del) mouse, which exhibited a 9% reduction in forebrain weight by postnatal day 7, suggesting that this reduction may originate prenatally. We investigated cortical neural precursor cell (NPC) proliferation from E13.5–E16.5 using EdU incorporation and immunohistochemistry for Pax6, Ki67, PH3, and Caspase-3. The number of EdU+ cells was increased in 3q29del embryos at E14.5 and E15.5 (E14.5: 18%, $p=0.005$; E15.5: 37%, $p<0.003$), with no differences at E13.5 or E16.5, suggesting the presence of a transient window of hyperproliferation. Spatial analysis of cell distribution revealed EdU+ enrichment in bins 2 to 4 - ventricular/subventricular zones (VZ/SVZ), which supports an expansion of the proliferative compartment. These regions also showed increased thickness, marked by Pax6 and Ki67 expression. Elevated levels of PH3+ mitotic cells further support enhanced proliferation. In contrast, caspase-3 staining at E13.5, E15.5, and E17.5 revealed no genotype differences, indicating that apoptosis is unlikely to account for the observed phenotype. Together, these findings identify a narrow mid-gestational window of NPC dysregulation in the 3q29del mouse cortex, which may contribute to altered cortical development associated with this deletion. This proliferative surge may alter cortical neuron identity, potentially increasing upper-layer production. Ongoing work examines laminar fate using layer-specific markers to assess long-term effects.

This work was supported by BHI NJACE SEED Grant Travel Award

Poster #35

Phosphodiesterase 2A Modulates Aβ-induced Oxidative Stress & Neuroinflammation in HT22 cells

Authors

Jason Sherman, Baomin Dou, Olivia Brzostek, Mathew Ayoub, Fu Xu, Hailiang Li, Zhutao Sheng, Ying Xu

PI Name: Ying Xu

Introduction: Alzheimer's disease (AD) is a progressive neurodegenerative disorder and is characterized by neuronal atrophy and loss. Its pathology is primarily driven by extracellular amyloid plaques and intracellular tau tangles, leading to synaptic and neuronal dysfunction. Our study aimed to identify molecular pathways driving AD progression, focusing on phosphodiesterase 2A (PDE2A), an enzyme that hydrolyzed both cAMP and cGMP into inactive forms. Since cAMP and cGMP exert neuroprotective effects, PDE2A activity may exacerbate oxidative stress and neuroinflammation. Methods: We investigated whether inhibiting PDE2A with Bay 60-7550 could mitigate neurodegenerative effects in cell models of AD. The hippocampus derived neuronal cells (HT-22 cells) were transfected with adeno-associated virus (AAV) to overexpress PDE2A or treatment of cells with beta amyloid to model AD pathology. Cell viability and apoptosis were quantified using CCK-8 and Annexin V-FITC/PI, respectively. Oxidative stress was assessed via reactive oxygen species (ROS), malondialdehyde (MDA), and superoxide dismutase (SOD) activity, while neuroinflammation was measured through TNF-α and IL-1β. Results :Both PDE2A overexpression and beta amyloid treatment decreased cell viability and increased apoptosis, ROS, MDA, TNF-α, and IL-1β, while reducing SOD activity. Treatment with Bay 60-7550 reversed these effects, as evidenced by significantly improving viability and reducing oxidative stress, neuroinflammation, and apoptosis. Conclusion : These findings suggest a pivotal role of PDE2A in AD pathogenesis through oxidative stress and neuroinflammation. Inhibition of PDE2A with Bay 60-7550 may represent a promising therapeutic to counteract neurodegeneration in Alzheimer's Disease.

Supported by NIH R01AG070873-01A1 and CBIR23IRG008

Poster #36

The maternal inflammation associated cytokine Interleukin-6 alters gene expression in neural progenitors.

Authors

Rebecca Zaritsky, Fernando Janczur Velloso, and Steven W. Levison

PI Name: Steven Levison

The incidence of Autism Spectrum Disorder (ASD) is rising, highlighting the importance of studying contributing environmental factors. Maternal immune activation, particularly elevated levels of the cytokine IL-6, which crosses into the fetal brain, has been consistently linked to ASD risk in offspring. Our group has developed a novel mouse model to show that transiently increasing early postnatal levels of IL-6 leads to behavioral abnormalities associated with ASD, including impaired sociability, altered communication, and increased repetitive behavior. Notably, these effects occur without neuroinflammation, suggesting direct effects of IL-6 on neurodevelopment. We have hypothesized that IL-6 disrupts neurogenesis and gliogenesis by altering gene expression of neural progenitors (NPs) in the subventricular zone (SVZ). These NPs will later produce interneurons, astrocytes, and oligodendrocytes that populate the neocortex. We have previously identified 8 sub-populations of NPs using antigenic profiles and have demonstrated heterogeneity in responses to IL-6, with one subpopulation proliferating more while others proliferate less. In this study, we have built on our previous characterization of NPs by using a novel combination of Parse Biosciences Split-Seq and CITE-Seq to analyze the impact of IL-6 administration on gene expression in defined NPs. Using this method, we have identified 21 unique cell populations in the periventricular region. We have identified Cdk8, a key regulator of stem cells, as being significantly upregulated in mouse SVZ cells following IL-6 administration and have found that IL-6 leads to increases in genes related to transcriptional regulation and decreases in genes related to mitochondrial energy synthesis.

This study was supported by CAUT26GFP004

Poster #37

The Interaction Between Physical and Mental Fatigue.

Authors

Aishwarya Dodda, Glenn Wylie

PI Name: Glenn Wylie

Physical and mental fatigue are common, but since physical fatigue relies on motor processes, and mental fatigue relies on cognitive processes, it is unclear if they are independent or interact with one another. We hypothesized that physical and mental fatigue rely, at least in part, on a common mechanism and that they will therefore interact with one another. To test this, 12 healthy individuals participated on 3 separate days. On Day 1, their maximum physical capacity (pedaling on an exercise bike) was established. On Days 2 and 3, physical and mental fatigue were induced in a counterbalanced order. Physical fatigue was induced by pedaling at 80% capacity until they were unable to maintain their cadence; mental fatigue induced with a moderately difficult cognitive task and was assessed using the Visual Analog Scale of Fatigue at intervals during the task. Results showed that as mental fatigue increased, subjects had longer response times on the cognitive task, but only when they were physically fatigued. Also, as mental fatigue increased, accuracy decreased. The order of physical and cognitive tasks affected physical performance such that subjects became physically fatigued faster if they were already mentally fatigued. Signal detection theory analysis of the cognitive task showed that sensitivity decreased as mental fatigue increased, and response bias was higher when participants were physically fatigued. These results show that physical and mental fatigue are not independent: physical fatigue affects mental fatigue and vice versa. This suggests that physical and mental fatigue share a common, central mechanism.

Supported by New Jersey Commission for Brain Injury Research (CBIR23IRG006)

Poster #38

Lipid droplets promote the aberrant liquid-liquid phase separation of alpha-synuclein in Parkinson's disease leading to impaired energy homeostasis.

Authors

Jose Cevallos, Elena Eubanks, Sunghoo Jung, Yiming Huang, Elyse Guadagno, Neeharika Rao Suvvari, Aryan Doshi, Nitya Ravinutala, Alejandro Mosera, Tim Bartels, Ulf Dettmer, Eleanna Kara

PI Name: Eleanna Kara

Alpha-synuclein (αSyn) is an intrinsically disordered protein that misfolds and aggregates within neurons in Parkinson's disease (PD), forming inclusions known as Lewy bodies. Recent evidence suggests that aberrant liquid-liquid phase separation might be involved in the early stages of αSyn dysregulation in PD. We investigated how aberrant phase separation is involved in the early stages of αSyn dysregulation. We modeled αSyn inclusions using human neuroblastoma M17D cell lines engineered to overexpress YFP-tagged αSyn under doxycycline induction. We had two separate cell lines for each of the following versions of αSyn: 1) wild type αSyn, 2) a PD autosomal dominant E46K mutant ("1K"). Wild type and 1K αSyn did not spontaneously form inclusions, but did so after treatment with oleic acid, which induces the formation of lipid droplets. The resulting inclusions entrapped lipid droplets. Fluorescence recovery after photobleaching (FRAP) experiments showed that they were liquid, consistent with biomolecular condensates. They were readily dissolvable through treatment with 1,6-hexanediol, which is an alcohol that breaks apart hydrophobic interactions, further supporting their liquid status. A pulse chase assay showed that lipid droplets entrapped within αSyn condensates had a reduced turnover rate in comparison to lipid droplets in the cytoplasm, suggesting that αSyn condensates could impair energy homeostasis in the cell. Finally, mitochondria around αSyn condensates were depolarized, which indicates that αSyn condensates could be toxic species affecting mitochondrial function. We conclude that αSyn forms biomolecular condensates in lipid droplet-rich environments, which impair lipid droplet turnover and can contribute to energy failure.

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Poster #39

Dopamine transporter dysfunction in the high-risk schizophrenia 3q29 deletion mouse model

Authors

Sindhu Sriramoji-Virdi and Miriam E. Bocarsly

PI Name: Miriam E. Bocarsly

In humans, the 3q29 microdeletion presents a >40-fold increased risk of schizophrenia, a debilitating mental disorder affecting behavior and cognition. Dopamine dysregulation has long been associated with schizophrenia symptomology but remains unexplored in 3q29 deletion patients. This current study utilizes the CRISPR CAS9 mouse model of the 3q29 deletion (3q29del) to explore dopaminergic functioning.

Employing fast-scan cyclic voltammetry (FSCV), we observed elevated dopamine levels in the dorsal striatum of 3q29del mice compared to littermate controls. To understand what is underlying this finding, we explored dopamine synthesis, reuptake, and degradation in 3q29 deletion mice. While no deficits were seen in indicators of dopamine synthesis and degradation, dopamine clearance capabilities of the dopamine transporter (DAT) were seen. Despite no differences in DAT protein levels, DAT ligand binding showed a 15% reduction in membrane DAT available in 3q29del mice compared to littermate controls. This suggests that DAT is present, but not functionally available at the membrane.

To determine what could be affecting the functionality of DAT, we turned our attention to PKCβ II, a protein kinase that regulates DAT trafficking to regulate dopaminergic signaling. Our studies suggest conflicting levels of PKCβ II activity between the brain and the periphery.

This research can be used to pioneer new therapies with novel targets for 3q29 deletion syndrome related symptom management.

Poster #40

Effort to Engage, Effort to Adapt: The Brain Near Criticality.

Authors

Li Xin Lim, and Andrew Westbrook

PI Name: Andrew Westbrook

Criticality – a balanced state of excitation versus inhibition which implies flexibility in how the brain responds to cognitive demands – is marked by scale-invariant activity. Prior work has shown divergence from criticality during demanding cognitive tasks, suggesting that divergence may track both effortful engagement and the loss of flexibility. We applied d_2, a metric quantifying deviation from criticality in terms of scale invariance (Sooter et al, 2024), to EEG data collected during a task-switching paradigm. We find that baseline-corrected d_2 is lower during easier Regular trials than during harder non-Regular (forced-switch, distractor, or voluntary-switch) trials, indicating greater deviation from criticality with greater demand. Performance metrics revealed region-specific patterns. During Regular trials, d_2 and accuracy negatively correlate in both midline and posterior electrodes, indicating that operating closer to criticality reflects preparedness and higher performance. During non-Regular trials, both regions exhibit an inverted-U relationship, suggesting an optimal level of divergence from criticality during preparation that maximizes accuracy. d_2 peaks at the end of a trial and peak midline d_2 increases with accuracy suggesting that the peak reflects active task engagement and effort exertion. Lower d_2 in posterior regions during the peak window was associated with higher performance in non-Regular trials, suggesting a role load-dependent processing. Furthermore, lower baseline posterior d_2 reflected increased voluntary switching, implying flexibility. Together, these results suggest that the d_2 metric offers a scale-sensitive marker of how neural dynamics adaptively shift with cognitive control demands for both effortful engagement and flexibility.

Supported by NIMH R00MH125021

Poster #41

Repurposing FDA-approved Drugs to Mimic Exercise Benefits and Improve Cognitive Health in Cancer Survivors

Authors

Sang Hoon Kim, Euiyeon Lee, Woohyun Jo, Sang Hoon Lee, Amelia Moon, Jade Lee, Mohammad A. Rashid, Bo Qin, Yoon-Seong Kim, Mi-Hyeon Jang

PI Name: Mi-Hyeon Jang

Chemotherapy-induced cognitive impairment (CICI), also known as chemobrain, occurs during or after cancer treatment, varying in onset, severity, and duration. This condition significantly impacts patients' quality of life due to a decline in cognitive functioning. Despite a 34% decrease in the overall cancer mortality rate from 1991 to 2022, many chemotherapy patients still experience memory, attention, processing speed, and executive function difficulties, highlighting the urgent need for new therapeutic strategies to mitigate chemobrain. Exercise and physical rehabilitation have consistently shown neuroprotective and restorative benefits for cancer patients, alleviating several negative effects of chemotherapy. However, regular exercise often proves challenging due to treatment-related fatigue, neuropathic pain, or mobility limitations. To overcome this, this study aims to identify and pharmacologically replicate the molecular mechanisms activated by exercise that protect against chemobrain. In preclinical models, cisplatin and doxorubicin treatments have been shown to induce characteristic symptoms of CICI, including sleep disturbances, weight loss, memory deficits, and anxiety-like behaviors. Bulk and single-cell RNA sequencing of these models revealed significant molecular changes underlying these symptoms. Through an AI-assisted bioinformatic workflow developed in this study, we identified exercise-responsive molecular candidates. A screening of FDA-approved drugs then uncovered compounds that mirrored the molecular signatures induced by exercise, underscoring their potential as pharmacological exercise mimetics. Overall, this research aims to uncover druggable neuroprotective pathways that connect exercise to cognitive resilience and repurposing safe, clinically available medications to treat chemobrain. This innovative approach holds great promise for improving cognitive outcomes and enhancing the quality of life for cancer survivors.

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Poster #42

Synaptojanin1 and VPS35 Cooperate at Presynaptic Endosomes to Regulate Dopamine Release.

Authors

Nirmal Kumar, Elnaz Khezerlou, Justin Cai, Hanna Caiola, Ulrik Gether, Huaye Zhang, Ping-Yue Pan

PI Name: Ping-Yue Pan

Synaptic dysfunction is a hallmark of early Parkinson's disease (PD), but molecular mechanisms underlying dopaminergic synaptic maintenance remain poorly understood. Here, we identify a functional interaction between two PD genes, Synaptojanin1 and VPS35, in regulating endosomal sorting of the dopamine D2 short (D2S) autoreceptor, thereby modulating dopamine signaling. We show that Synaptojanin1 deficiency results in reduced behavioral responsiveness to a D2-like agonist and impaired gating of dopamine release due to intracellular retention of the D2S. VPS35 is recruited to D2S-containing and Rab7a-positive endosomes in a Synj1-dependent manner, and VPS35 overexpression overcomes Synj1 deficiency-associated impairment in D2S surface delivery in axons. Moreover, Synj1 regulates VPS35 expression and localization in dopaminergic axons, indicating their broader roles in presynaptic cargo sorting. These findings reveal a novel cooperation between a synaptic vesicle endocytic regulator and a core component of the retromer complex, providing new mechanistic insights into presynaptic trafficking and its disruption in PD.

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Poster #43

The role of Tex15 in stochastic olfactory receptor choice.

Authors

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PI Name: Kevin Monahan

Mammals smell by using odorant receptor (OR) proteins to detect chemicals present in the environment. Each olfactory sensory neuron in the mouse main olfactory epithelium (MOE) expresses only one type of OR. Many developmental mechanisms contributing to singular OR choice in mature ONs have been identified, including de novo heterochromatin deposition, establishment of zonal organization of the MOE, co-expression of multiple ORs etc. But the factors guiding the process of OR choice remain poorly understood. These key processes occur in the immediate neuronal progenitors and immature OSN cell stages. Here, we show that Testes expressed gene 15 (Tex15) is transiently expressed in OSNs during this critical developmental window. The absence of Tex15 causes a vast skewing of OR choice towards ORs that are normally expressed during early differentiation and then repressed. Thus, it results in a deterministic patterning of the MOE where only a few ORs are highly expressed. Tex15 KO mice fail to repress these ORs and generally exhibit increased levels of OR expression in immature cells. There is a dramatic breakdown of spatial patterning of OR gene choice. These findings reveal a novel mechanism that represses the expression of the earliest expressed OR genes, allowing OR choice to sample the full repertoire of OR genes. Thus, at the individual OSN level, OR choice is stochastic, at the whole population level, OSNs chose their OR in a probabilistic manner allowing for the full diverse set of ORs to be expressed and allowing mice to smell many odorant chemicals.

Supported by 5F31DC021641, 5R35GM146901, Rita Allen Foundation Grant

Poster #44

OCNDS-associated CK2a mutations disrupt stress granule homeostasis to cause neurodevelopmental defects.

Authors

Manasi Agrawal, Shruti Ghumra, Meghal Desai, Yashashree Bhorkar, Brandon Vaglio, Bonnie Firestein, Pabitra Sahoo

PI Name: Pabitra Sahoo

Okur-Chung neurodevelopmental syndrome (OCNDS) is caused by mutations in *CSNK2A1*, a gene that encodes Casein Kinase 2α (CK2α) protein. It is a disorder characterized by intellectual disability and developmental delay. CK2α is a constitutively active serine/threonine kinase with multiple substrates and is implicated in diverse signaling pathways. While the OCNDS-associated CK2a mutants show varying degrees of reduced kinase activity, the molecular mechanisms linking CK2α dysfunction to neurodevelopmental defects remain unclear. Previously, we demonstrated that in peripheral nerves, CK2α promotes axon regeneration by phosphorylating G3BP1, a key stress granule (SG) protein that inhibits axonal protein synthesis. Here, we investigate whether OCNDS-linked CK2α mutants cause neurodevelopmental defects via G3BP1 granules. Using primary rat cortical neurons, COS7, and HEK cells as model systems, we find that expression of OCNDS-associated CK2α mutants decreases G3BP1 phosphorylation and forms defective SG cores, leading to aberrant SG assembly. Additionally, these mutations lead to reduced protein synthesis and defects in axonal and dendritic growth. RNA-immunoprecipitation analyses reveal reduced G3BP1 and its target mRNA interactions, suggesting broad defects in post-transcriptional regulation. Together, these findings reveal a signaling cascade in which CK2α-dependent phosphorylation of G3BP1 regulates SG dynamics, resulting in impaired neuronal structure and function.

Supported by Merkin Peripheral Neuropathy and Nerve Regeneration Center.

Poster #45

Storage and translational regulation of axonal *App* mRNA by G3BP1 granules.

Authors

Shruti Ghumra, Manasi Agrawal, Meghal Desai, Mansi Pandya, Wilma Friedman, Pabitra Sahoo

PI Name: Pabitra Sahoo

Stress granules (SGs) are molecular condensates of ribonucleoprotein (RNP) - based cellular compartments that are formed upon exposure to various environmental stressors. Their primary function is temporarily storing and protecting mRNA from being degraded, halting translation until normal conditions are restored. SGs comprise many proteins, including G3BP1, a core nucleating factor. The amyloid precursor protein (APP) is well-known for its role in promoting cell growth, synaptic plasticity, and neural health, with its expression significantly upregulated in neurodegenerative diseases like Alzheimer's disease (AD). There is a possibility that SGs, particularly G3BP1 granules, may play a role in regulating local *App* mRNA translation, potentially linking them to axonal degeneration. How dysregulation of local APP synthesis contributes to neurodegeneration, especially the local translation of *App* mRNA at axons which may regulate synaptic functions, remains unclear. Understanding how local APP synthesis contributes to axonal degeneration even in non-pathological conditions, is crucial to comprehending how normal synapses might become vulnerable under stress or in disease contexts. ProNGF induces retrograde axonal degeneration in basal forebrain cholinergic neurons (BFCNs) through p75 neurotrophin receptor (p75NTR) activation, which triggers the local synthesis of APP in axons. Mass spectrometry analysis has revealed that APP acts as a key upstream regulator of axon degeneration pathways following proNGF stimulation, with APP synthesis specifically occurring in axons. Blocking local APP synthesis using siRNA protects against proNGF-induced axonal degeneration, highlighting the critical role of locally synthesized APP in neurodegeneration. Our objective is to determine whether *App* mRNA is stored in axonal stress granules in BFCNs and how its release is regulated upon neurotoxic stimulation.

Supported by BHI Travel award 2025

Poster #46

Loss of function mutations in neurodevelopmental and psychiatric risk genes alter neuronal morphology and synaptic transmission in human neurons.

Authors

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PI Name: Zhiping Pang

Neurodevelopmental and psychiatric disorders (NPDs), including autism, schizophrenia, bipolar disorder, and major depressive disorder, are complex conditions with high heritability and significant genetic contributions. Over the past decade, increases in genome-wide association studies (GWAS), whole-exome sequencing (WES), and study cohort sizes have enabled the detection of rare genetic variants associated with various NPDs. However, the effects of these mutations on neuronal function and synaptic transmission have not been assayed on a large scale. To address this gap, we developed a pipeline for clonal loss-of-function (LoF) mutagenesis in human induced pluripotent stem cells (hiPSCs) using cytosine base editing to introduce premature stop codons. Using this pipeline we have generated LoF mutants for >60 NPD risk genes across 4 different hiPSC lines, which were verified for pluripotency, genomic integrity, and gene LoF through transcript and protein-level analyses. These edited lines were differentiated into 2D cocultures of excitatory and inhibitory neurons to assess the impact of individual gene disruptions on neuronal morphology and function. We used high-content imaging to assay morphological differences in neuronal cultures and identified mutations that decrease neurite branching and synaptic puncta density. To evaluate synaptic transmission, we used the optical calcium sensor jGCaMP8f to measure spontaneous neuronal activity at single-cell and network levels. Our analysis revealed that specific LoF mutations result in altered calcium dynamics, suggesting changes in baseline synaptic activity and excitability. By combining these complementary assays, we aim to uncover shared functional signatures and identify common cellular pathways to deepen our understanding of the biology of NPD.

Supported by NIH-NIMH grant RM1MH133065, NJGCA postdoctoral associate grant CAUT26DAP007

Poster #47

Forebrain Traumatic Brain Injury Causes Brainstem Adenosine Surging During Respiratory Arrest: Photometry-Based Insights from a Mouse Model.

Authors

Benton Purnell, Elise Schicker, Anna Schicker, Jayant Bhasin, Detlev Boison

PI Name: Detlev Boison

Traumatic brain injury (TBI) is the leading cause of death in individuals under 45. Acute TBI can cause life-threatening respiratory arrest. The cause of this phenomenon is enigmatic as it can occur without substantive blood loss or damage to brainstem respiratory control centers. Adenosine is neuroprotective and TBI increases adenosine signaling near the injury. Excessive adenosine in the brainstem causes respiratory arrest, but whether forebrain TBI increases adenosine signaling in the brainstem was unknown. The goal of this investigation was to determine whether TBI precipitates adenosine surging in the brainstem during respiratory arrest. Our approach was to induce cortical lateral fluid percussion injury while monitoring extracellular adenosine and intracellular calcium in the brainstem dorsal raphe nucleus using dual-color fiber photometry in mice (adenosine, AAV9-hSyn-Ado1.1m; calcium, AAV9-Syn-jRGECO1a). Breathing was assessed retrospectively by video analysis. TBI consistently and rapidly increased extracellular adenosine and intracellular calcium (adenosine, $p < 0.001$; calcium, $p = 0.028$; $n = 8$-11). Adenosine and calcium florescence peaked simultaneously, within seconds of the injury ($p = 0.808$). However, intracellular calcium fell faster than extracellular adenosine ($p = 0.045$). This data suggests that there is a rapid surge in extracellular adenosine in the brainstem during TBI that may contribute to respiratory arrest. However, the intracellular calcium surge may be indicative of spreading depolarization, which can also cause respiratory arrest. Improving our understanding of TBI-induced respiratory arrest may provide translatable insights for the management of acute TBI in humans and best practices in livestock euthanasia.

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Poster #48

Effect of neurodevelopmental disorder associated G3BP1 mutations on stress granule assembly and function.

Authors

Meghal Desai, Manasi Agrawal, Shruti Ghumra, Robert Palmere, Vlad Kholodovych, Bonnie Firestein, Pabitra K. Sahoo

PI Name: Pabitra Sahoo

Neurodevelopment is a meticulously organized process involving cell migration, proliferation, and differentiation, collectively regulating nervous system development. Any disruption in these processes can lead to neurodevelopmental disorders (NDDs), resulting in impairments in personal, social, academic, or occupational functioning. During neuronal development, local protein synthesis plays key roles in processes such as neurite growth, establishing dendrite-axon polarity, and the formation of functional synapses. Multiple pieces of evidence show that specific mRNAs are stored in dendrites and axons in a translationally suppressed state, only to be translated when specific stimuli trigger this process. Stress granules (SGs) are dynamic ribonuclear protein complexes formed that store and protect mRNAs. We have shown that SG-like structures are present in the axons of both the central nervous system (CNS) and peripheral nervous system (PNS) neurons under physiological conditions, providing a mechanism to regulate localized protein synthesis. Ras GTPase-activating protein-binding protein 1 (G3BP1) is a core protein that has been shown to nucleate SG assembly. Recent studies have identified de novo mutations in G3BP1 in individuals with neurodevelopmental disorders, though the underlying mechanism remains unknown. We identified G3BP1 granules within synapses, where they may contribute to synapse development and function regulate local protein synthesis. We also observed NDD associated G3BP1 mutations altered granule disassembly. NDD associated G3BP1 expression alters G3BP1 granules formation in neurons, which in turn causes development defects. We next aim to characterize transcriptomic changes in G3BP1 granules carrying NDD-associated mutations, which may underlie defects in neuronal development.

Supported by BHI trainee travel award (BHI Neurodevelopmental FAWG)

Poster #49

Endothelial Ahnak deletion mitigates neuroinflammation and amyloid-β pathology by preserving blood-brain barrier integrity.

Authors

Yu Young Jeong, Chi Chang Sung, Bruno Carabell, Gaetan Barbet, Yong Kim

PI Name: Yong Kim

Vascular dysfunction and blood-brain barrier (BBB) breakdown are early and critical events contributing to abnormal neuroinflammation and Alzheimer's disease (AD) pathogenesis. Despite their importance, the molecular mechanisms by which BBB integrity is preserved and neuroinflammation is mitigated in AD remain poorly understood. Recent genomics and proteomics studies have identified Ahnak as a novel hub molecule. However, Ahnak function in AD pathology has not been fully elucidated. Ahnak is highly enriched in cerebrovascular endothelial cells (ECs) in both human and rodent brains. Using endothelial cell-specific translating ribosome affinity purification (EC-TRAP) followed by RNA sequencing and biochemical validation of vessel-enriched fractions, we show that Ahnak deletion reprograms endothelial gene expression toward an immune-suppressive state in mice. This shift is characterized by the upregulation of barrier-stabilizing and immune-regulatory molecules, including tight junction proteins and glycocalyx components such as endomucin. Furthermore, siRNA-mediated Ahnak knockdown in human umbilical vein endothelial cells (HUVECs) increases Claudin-5 expression in an AMPK-dependent manner, an in-cell effect of Ahnak deletion on maintaining junctional integrity. In APP/PS1 transgenic AD mice, endothelial-specific Ahnak deletion restores Claudin-5 and Endomucin expression, while reducing microgliosis and amyloid-β deposition. Collectively, these findings identify Ahnak as a key regulator of BBB integrity and neuroinflammation in AD. Targeting the Ahnak pathway may represent a promising therapeutic strategy to enhance vascular resilience and slow AD progression.

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Poster #50

In vivo discovery of blood–brain barrier modulators for improved brain drug delivery.

Authors

Thomas Potts, Erin McDonnell, Lewis Gall, Will Capello, Neha Gururaj, Allie Vindigni, Manan Gosai, Natasha O'Brown

PI Name: Natasha O'Brown

The blood–brain barrier (BBB) refers to the uniquely restrictive properties of the brain's blood vessels that maintain neural homeostasis while preventing entry of toxins and pathogens. BBB disruption is linked to numerous neurodegenerative diseases, including Alzheimer's, Parkinson's, and multiple sclerosis, yet its selectivity also blocks over 98% of small molecules from entering the brain. Therefore, there is a critical need for strategies to modulate BBB properties to improve treatment of neurological disorders. Zebrafish provide a powerful model to study BBB function because they are optically transparent, produce hundreds of offspring, develop a functional BBB by 5 days post-fertilization, and rapidly absorb small molecules from water. However, standard tracer leakage assays rely on cardiac injections and confocal imaging, limiting throughput. To overcome this, we developed a novel activity-based assay that enables efficient testing of BBB modulators in 96-well format. Using this platform, we screened 2,320 FDA-approved small molecules for their ability to increase BBB permeability and identified 17 hits spanning diverse molecular pathways. Follow-up tracer leakage assays refined this list to 8 compounds that increased BBB permeability across both assays, including two in the vitamin D pathway. We are now validating these hits in mouse and human iPSC-derived neurovascular models to assess conservation of their effects. These findings highlight the power of zebrafish for rapid in vivo drug discovery and reveal new molecular pathways that can be harnessed to modulate BBB permeability, paving the way for improved strategies to enhance therapeutic delivery to the brain.

Supported by Damon Runyon Timmerman Traverse-Rachleff Innovator Award

Poster #51

Ethanol induces neuroimmune dysregulation and soluble TREM2 generation in a human iPSC neuron, astrocyte, microglia triculture model.

Authors

Andrew J. Boreland; Yara Abbo; Xindi Li; Alessandro C. Stillitano; Siwei Zhang; Jubao Duan; Zhiping P. Pang; Ronald Hart

PI Name: Ronald Hart

Alcohol use disorders (AUDs) affect substantial populations worldwide and increase the risk of developing cognitive impairments and alcohol-associated dementia. While chronic inflammatory signaling likely plays an important role in alcohol-associated neurological sequalae, the precise mechanisms underlying alcohol-associated neuropathology remain enigmatic. We hypothesize that alcohol leads to neuroimmune dysregulation among neurons, astrocytes, and microglia; and is perpetuated by innate immune signaling pathways involving cell-cell signaling. To investigate how alcohol dysregulates neuroimmune interactions in a human context, we constructed a triculture model comprising neurons, astrocytes, and microglia derived from human induced pluripotent stem cells. After exposure to ethanol, we observed significant differential gene expression relating to innate immune pathways, inflammation, and microglial activation. Microglial activation was confirmed with morphological analysis and expression of CD68, a lysosomal-associated membrane protein and marker for phagocytic microglial activation. A striking finding in our study was the elevation of TREM2 expression and, specifically, TREM2 alternatively spliced isoforms that are predicted to give rise to soluble TREM2. TREM2 loss-of-function variants have been reported to be a risk factor for Alzheimer's disease. These results suggest that ethanol exposure in the brain may lead to increased microglial activation and production of soluble isoform named TREM2219 through alternate splicing. Deciphering the molecular and cellular mechanisms underpinning ethanol-related neuroimmune dysregulation within a human context promises to shed light on the etiology of AUD-related disorders, potentially contributing to the development of effective therapeutic strategies.

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Poster #52

TET3-mediated regulation of hippocampal neuronal morphology.

Authors

James M. Kina and Wilma J. Friedman

PI Name: Wilma J. Friedman

The regulation of neuronal morphology during development is critical for proper hippocampal circuit formation. Several factors are involved in regulating neuronal morphology, including neurotrophins such as brain-derived neurotrophic factor (BDNF). However, the spectrum of genes that are targeted by neurotrophin signaling to regulate neuronal morphology is likely different than those involved in other processes, such as maintaining survival. Epigenetic processes alter the conformation of chromatin to change how accessible genes are to transcription factors. Ten-eleven translocation (TET) enzymes play a key role in DNA demethylation and changing the expression status of methylated genes from being silenced to actively expressed. Of the three distinct TET enzymes, TET3 is the most abundant in the brain and hippocampus. Therefore, we hypothesized that TET3 activity is important in regulating hippocampal neuronal morphology. Here, we demonstrate that TET3 knockdown in E18 hippocampal neurons leads to extensive dendritic growth and complexity. Interestingly, inhibition of Trk signaling greatly reduces the TET3 knockdown-induced growth, with a similar effect also observed by inhibiting the Akt or Erk1/2 pathways. Further, we investigated the spectrum of genes that are influenced after knocking down TET3. Many differentially expressed genes that were identified play crucial roles in regulating cytoskeletal changes.

Poster #53

Expression Level Impact of Developmental *Cc2d1a* Reduction as a Sex-Specific Mouse Model of ASD/ID and growth deficits.

Authors

Abigail Heller, Shanzeh Rauf, Aniket Bhattacharya Emma Suzuki, and M. Chiara Manzini

PI Name: M. Chiara Manzini

Intellectual disability (ID) and Autism Spectrum Disorder (ASD) exhibit notable male diagnostic bias (4:1 for ASD, 2:1 for ID) with complex genetic heterogeneity. Previous research identified *CC2D1A* loss of function (LOF) as a cause of 100% penetrant ID, and highly penetrant ASD. While existing *Cc2d1a* conditional knockout (cKO) mouse models avoid early postnatal lethality of global gene removal, their conditional limitations inadequately represent patients with developmental *CC2D1A LOF*. In generating mice where CC2D1A is tagged with V5-HA epitope tags we found that this modification results in protein degradation and a hypomorph allele. Homozygous V5-HA mice (*Cc2d1a*$^{VH/VH}$) survive despite ~86% CC2D1A reduction but do not show ASD/ID-like behaviors. To explore phenotypic penetrance thresholds, we crossed this line with a null allele, creating a more severe hypomorph (*Cc2d1a*$^{-/VH}$) with ~90% protein reduction. These mice are viable and fertile without major sensory-motor deficits. *Cc2d1a*$^{-/VH}$ females show developmental stunting at the onset of puberty but normal behavior. While *Cc2d1a*$^{-/VH}$ males exhibit deficits in sociability, social novelty preference, and spatial memory acquisition as we had found in forebrain-specific cKOs. But unlike forebrain specific cKOs these mice do not recapitulate CREB activation deficits. Instead, they show a male specific cortical CaMKII activation deficit. This severe developmental hypomorph model reveals unique neuronal mechanisms driving sex-specific ASD/ID-like phenotypes within *Cc2d1a* reduction, offering novel insights into what factors may be at play within the ASD and ID diagnostic sex bias.

Supported by Eagles Autism Foundation R21MH140028

Poster #54

Advanced Biomaterial Delivery of Hypoxia-Conditioned Extracellular Vesicles (EVs) as a Therapeutic Platform for Traumatic Brain Injury.

Authors

Joshua B. Stein, Songzi Zhang, Eun Ji Roh, Jeffrey Luo, Meizi Chen, Hyunjun Jang, Li Ling Goldston, Brandon Conklin, Inbo Han, Ki-Bum Lee

PI Name: Ki-Bum Lee

Traumatic Brain Injury (TBI) is a common and debilitating injury, causing long-lasting neurological deficits. Current therapeies for recovery remain inadequate, undersing the urgent need for innovative interventions. In this study, a novel therapeutic approach is introduced that delivers extracellular vesicles (EVs) derived from human-induced pluripotent stem cell-derived neural progenitor cells (hiPSC-NPCs) with a gelatin-based injectable bioorthogonal hydrogel (BIOGEL). The hiPSC-NPCs are conditioned with deferoxamine (DFO) to simulate hypoxia, resulting in EVs enriched with neurotrophic and angiogenic factors critical for neural repair. The biomimetic mechanical properties of BIOGEL, similar to those of native brain tissue, contribute to sustained EV delivery and promote neural regeneration. BIOGEL with hypoxia-conditioned EVs showed significant tissue regeneration in vivo using a rat model of TBI. Our nanomaterial platform reduced cortical lesions, improved neurological and motor recovery, enhanced hippocampal neurogenesis and myelination, and reduced neuroinflammation, demonstrating strong therapeutic potential for neural repair. In summary, this study demonstrated proof-of-concept for a multifaceted therapeutic platform that simultaneously targets key pathological features of TBI, providing a scalable and clinically translatable approach to effective neural tissue regeneration. The synergistic combination of hypoxia-conditioned EVs and biomaterial delivery offers a promising strategy for advancing regenerative medicine techniques for neural repair.

Supported by partial financial support from the NJCSCR (CSCR16ERG019; CSCR24IRG005), 1R01NS130836-01A1, RM1 NS133003-01, R21 NS132556-01, NJCBIR (CBIR25IRG005; CBIR25IRG015), SAS-Grossman Innovation Prize, AARG-NTF-21-847862 and CDMRP (OCRP, OC220235P1, and NETP, PD210065). HR16C0002, NRF grant (No. RS-2025-00515818). MSIT, T32EB005583. NSF GRFP (DGE-2233066).

Poster #55
Characterization of new knock-in rat models for cerebral amyloid angiopathy in Alzheimer's Disease.

Authors
Asma Rizvi, Vishal Singh, Hyung Jin Ahn

PI Name: Hyung Jin Ahn

Cerebral amyloid angiopathy (CAA), caused by amyloid-β (Aβ) deposition around cerebral blood vessels, is a key contributor to vascular dysfunction in Alzheimer's disease (AD) and occurs in most AD patients. CAA-associated pathologies include smooth muscle cell loss, microhemorrhages, microinfarcts, neuroinflammation, and motor impairments. However, the mechanisms by which CAA contributes to vascular dysfunction in AD remain unclear, partly due to the lack of an ideal rodent model. Existing transgenic rodent models, which overexpress the human amyloid precursor protein (APP) gene under artificial promoters, disrupt the natural gene-protein network and therefore have limitations. To address this, we generated two APP knock-in rat models using CRISPR-Cas9 technology: (1) the SIA-KI rat, carrying three APP mutations known to enhance CAA and amyloid pathology with a humanized Aβ sequence, and (2) the Iowa-KI rat, carrying a CAA associated mutation with a humanized Aβ sequence. We examined CAA pathology, neuroinflammation, and motor dysfunction in both models. The SIA-KI rats showed significant Aβ plaque deposition and CAA in both capillaries (type 1) and larger vessels (type 2) within the olfactory bulb, cortex, and thalamus, with an age-dependent increase. These rats also exhibited pronounced neuroinflammation and progressive motor deficits, including gait abnormalities and impaired coordination. In contrast, Iowa-KI rats showed no evidence of CAA pathology, neuroinflammation, and gait abnormalities, likely due to less Ab deposition. Overall, the SIA-KI rat represents a promising model for studying the mechanisms and pathophysiological consequences of CAA in Alzheimer's disease.

Supported by New Jersey Health Foundation PC114-25

Poster #56
Chimeric Brain Models to Study Human Glial-Neuronal and Macroglial-Microglial Interactions.

Authors
Mengmeng Jin, Ziyuan Ma, Haiwei Zhang, Rui Dang, Ava V. Papetti, Alessandro C. Stillitano, Lisa Zou, Steven A. Goldman, Peng Jiang

PI Name: Peng Jiang

Chimeric brain models, generated by transplanting human pluripotent stem cell (hPSC)- derived neural cells, are valuable for studying the development and function of human neural cells in vivo. Understanding glial-glial and glial-neuronal interactions is essential for unraveling the complexities of brain function and developing treatments for neurological disorders. To explore these interactions between human neural cells in vivo, we co-engrafted hPSC-derived neural progenitor cells together with primitive macrophage progenitors into the neonatal mouse brain. This approach creates chimeric brains containing human microglia, macroglia (astroglia and oligodendroglia), and neurons. Using super-resolution imaging and 3D reconstruction techniques, we examine the dynamics between human neurons and glia, and observe human microglia pruning synapses of human neurons and engulfing neurons themselves. Single-cell RNA sequencing analysis of the chimeric brain uncovers a close recapitulation of the human glial progenitor cell population, along with a dynamic stage in astroglial development that mirrors the processes found in the human brain. Furthermore, our cell–cell communication analysis reveals significant human neural cell interactions, with a notable emphasis on neurexin–neuroligin adhesion molecule interactions between neurons and astrocytes. Interestingly, neuroligin-3 (NLGN3) was the only neuroligin family member identified in neurexin-mediated interactions, appearing exclusively as a receptor on human astrocytes. We also found interactions between microglia and astroglia mediated by SPP1, crucial for promoting microglia growth and astrogliosis, and the PTN-MK pathways, instrumental in homeostatic maintenance and development in macroglial progenitors. This innovative co-transplantation model opens new avenues for exploring the complex pathophysiological mechanisms underlying human neurological diseases. It holds promise for studying disorders where glial-neuronal interactions and non-cell-autonomous effects play crucial roles.

Poster #57

Cut Off, Tuned Up: Adolescent Social Isolation Amplifies Dopamine Signaling in the Dorsomedial Striatum.

Authors

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PI Name: Elizabeth Holly

Adolescence represents a critical period of cognitive, neural, and social maturation. During this stage, social play and exploration increase in parallel with corticostriatal and dopaminergic system development. Social experience has a profound influence on the development of neural circuitry, so social deprivation during adolescence can induce long-lasting behavioral and physiological alterations. As dopaminergic circuitry continues to develop through adolescence, we investigated whether adolescent social isolation (ASI) disrupts dopaminergic signaling and dopamine-dependent learning and decision behaviors. To model ASI, mice were housed either with a littermate or in isolation at weaning. In adulthood, mice were trained in an operant value-based decision-making task in which lever contingencies were reversed following stable performance, requiring behavioral adaptation. Because dopamine circuits continue to develop across adolescence, we hypothesized that ASI disrupts this normal development, manifested as augmented dopamine release both in slice and in vivo. To test this, we recorded ex vivo dopamine release using fast scan cyclic voltammetry and found that ASI induces pronounced alterations in dopamine kinetics. Isolated mice exhibited significantly greater and more prolonged dopamine transients relative to group-housed controls, with exaggerated signaling upon reward delivery followed by a suppression phase. We next recorded the GRAB-DA dopamine sensor via fiber photometry as mice completed an operant probabilistic reversal task that requires flexible behavioral adaptation and learning. We observed amplified DA signaling during rewarded, but not unrewarded, trials. Ongoing work continues to explore in vivo dopamine signaling during value-based choice, as well as the causality of these changes.

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Poster #58

Loss of BCKDK impairs neuromuscular function in male mice.

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Branched-chain amino acids (BCAAs) support protein synthesis and neurotransmitter metabolism, and their oxidation is restrained by branched-chain α-keto acid dehydrogenase kinase (BCKDK). We previously observed that in the brains of Bckdk knockout (KO) mice, BCAA levels fall while glutamate is preserved and other amino acids rise. We asked why this profile emerges and how it affects motor and nutrient-stress responses. Whole-body Bckdk KO and wild-type (WT) mice were fed control or low-protein diets. Motor behavior, nutrient signaling in skeletal muscle and brain, and nitrogen delivery from BCAAs were assessed using constant-rate [13C/15N]-BCAA infusions, LC-MS of serum [15N]-BCAAs, and MALDI-MS imaging of brain [15N]-glutamate. KO mice maintained grip strength but showed poorer beam traversal and reduced voluntary running after protein restriction. In skeletal muscle, fasting suppressed protein synthesis and mTORC1 similarly in both genotypes and recovered with leucine or protein, without activating the integrated-stress response (ISR). In brain, KO mice exhibited fasting-induced eIF2α phosphorylation and up-regulation of the Slc7a5 mRNA that only partly relieved with refeeding. Tracer studies revealed lower serum [15N]-BCAA enrichment and diminished brain [15N]-glutamate labelling in KOs, despite unchanged total glutamate. Thus, constitutive BCKDH activation accelerates peripheral BCAA oxidation, limits BCAA-derived nitrogen delivery to brain, and triggers Slc7a5-mediated substitution by non-BCAA amino acids. This mechanism explains the motor deficit in Bckdk KOs and aligns with human BCKDK or LAT1 mutations linked to autism spectrum disorder.

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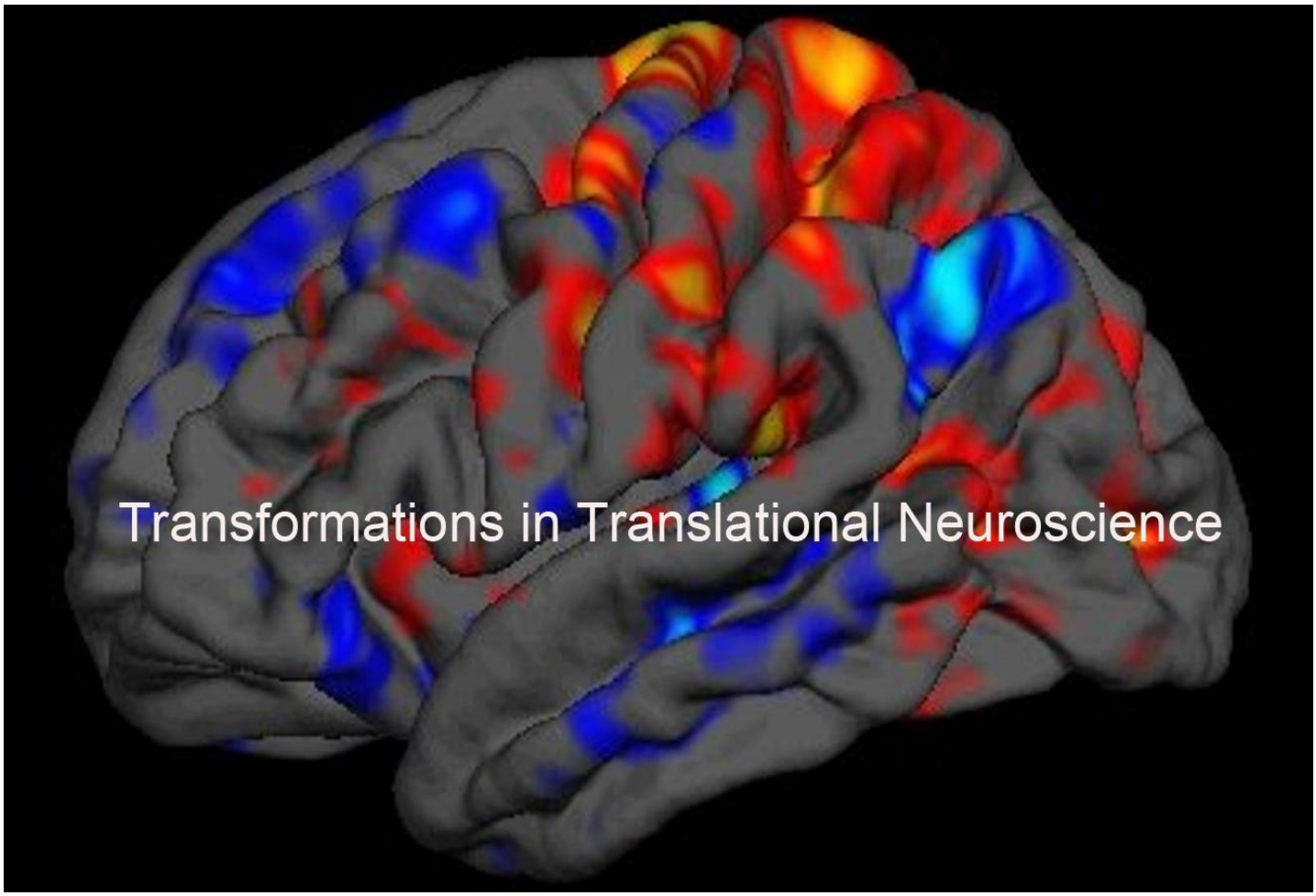

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