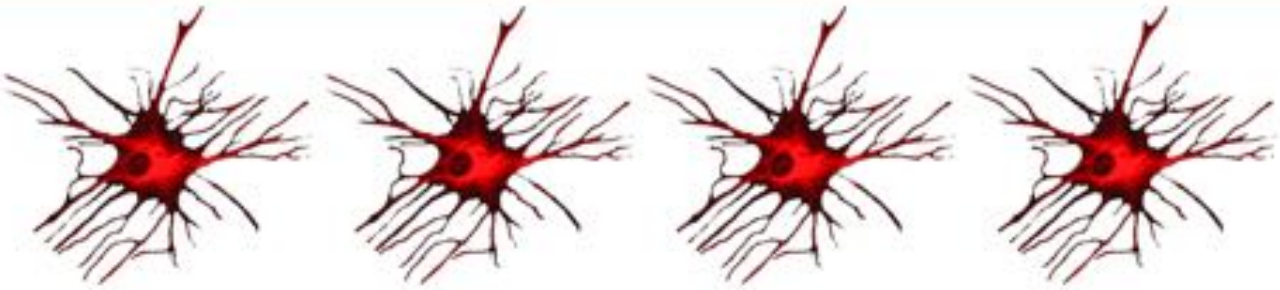
# Chicago Chapter of the Society for Neuroscience 2018 Scientific Meeting



## Friday, March 23rd, 2018

## Northwestern Memorial Hospital

# 2018 Annual Scientific Meeting

**Northwestern University - Memorial Hospital**
**March 23rd, 2018**

## MEETING OVERVIEW

**7:30-10:00 AM** **Registration/Continental Breakfast** — *3rd Floor*

**8:00-8:45 AM** **Mentoring Panel & Breakfast (with Conference Speakers)** — *Pritzker Aud.*
Moderators: Drs. Simon Kaja and Virgine Mansuy-Aubert

**8:00-4:00 PM** **Poster Viewing and Vendor Display** — *Atrium, 3rd Floor*

**9:10-10:30 AM** **PRESIDENTIAL SYMPOSIUM** — *Room A*

***New Developments in Neuroscience***
Chaired by Anna Lysakowski, Ph.D.

**Neuroinformatics and the Multiscale Brain: from genes to brain states and beyond**
**Sean Hill, Ph.D.** Krembil Centre for Neuroinformatics, Centre for Addiction and Mental Health, Toronto, Blue Brain Project, EPFL, Switzerland

**The Making of the Inner Ear**
**Doris Wu, Ph.D.** NIH-NIDCD

**10:30-11:30 AM** **KEYNOTE SPEAKER** — *Room A*

**The Neurobiology of Helping: Lessons from Order Rodentia**
**Peggy Mason, Ph.D.** Department of Neurobiology, University of Chicago

**11:30-2:00 PM** **POSTER COMPETITIONS AND LUNCH BREAK**

Graduate, Undergraduate and Postdoctoral Poster Competitions — *Atrium, 3rd Floor*

*"Diversity in Careers"* Lunch tables – Aaron Schirmer, organizer

**12:15-1:15 PM** Dr. Mason and Graduate Student Symposium participants lunch

**2:00- 3:30 PM** **GRADUATE STUDENT SYMPOSIUM** — *Room A*

Selected Graduate Student Talks from six of the Chicago area Ph.D. granting medical schools

**3:45- 5:40 PM** **PLENARY AFTERNOON SYMPOSIA** (*Concurrent Sessions*)

| | **Room A** | **Pritzker Auditorium** |
|---|---|---|
| **3:45-3:50 PM** | ***Progress in Movement Disorders***<br>Chaired by Joanne O'Keefe & Orly Lazarov | ***Biomedical Engineering Applications in Neuroscience***<br>Chaired by Kelly Langert & Kaiwen Kam |
| **3:50-4:15 PM** | ***Exercise and neuroprotection and clinical biomarkers in PD***<br>Daniel Corcos (NWU) | ***Biodegradable Nanoparticle Approach for the Treatment of MS and SCI***<br>Stephen Miller (NWU) |
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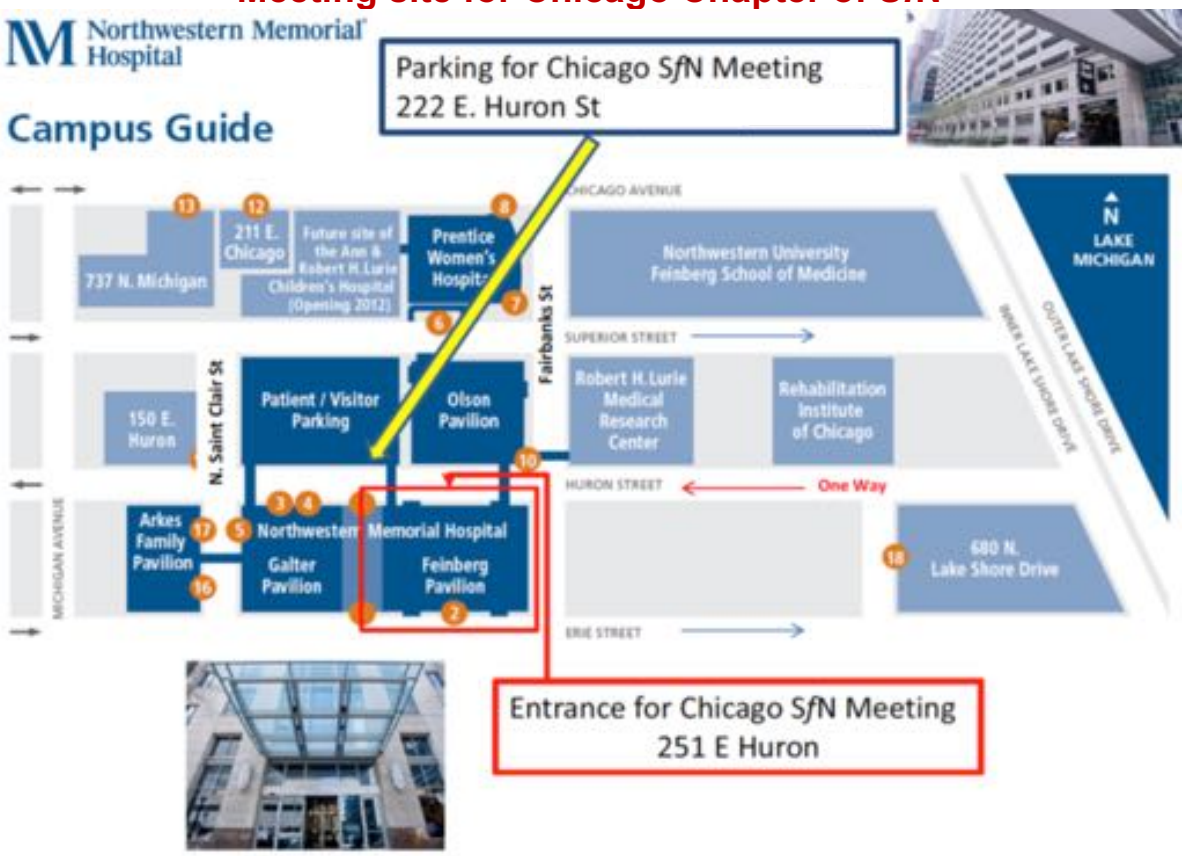
**5:45-7:00PM** **RECEPTION AND BUSINESS MEETING**
Social and announcement of awards, recognition, election results

Map of Northwestern University downtown campus and
Feinberg Pavilion, 3rd Floor Conference Center, 251 E. Huron St., Chicago, IL 60611

## Meeting site for Chicago Chapter of SfN




Atrium, 3rd floor

- Take the escalators or elevators to Conference Center on 3rd Floor.
- Please visit the corporate exhibitor tables in the Atrium on the 3rd floor.
- Posters should be removed by 4:00 PM today.

Vote for next year's Chicago Chapter SfN Officers and Councilors
https://goo.gl/forms/az1vAzGk7mju7Sy13

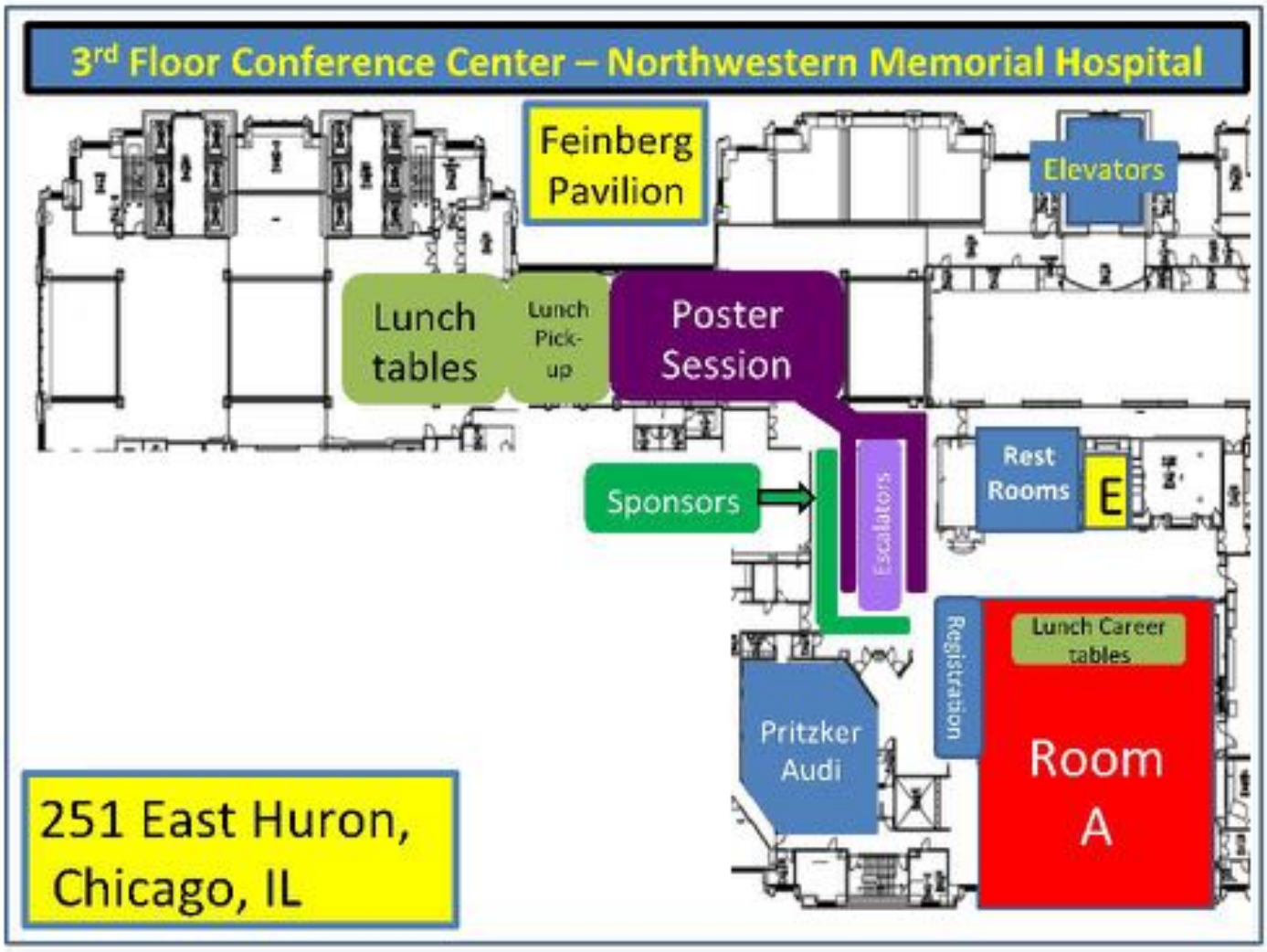
- Please give us your opinion by answering our online survey.
https://goo.gl/forms/1uwfY3PsICNPT1sJ2

- You will be included in a drawing for a $25 gift card. Your input is critical for a better meeting next year.

Parking

- When exiting Northwestern Hospital's parking garage, please show your validated parking voucher for a parking discount (up to 7 hours $11 and 8 hours or more $24).

**Cover Art:** Jennifer Schreiber, 4th Year Ph.D. Candidate from Loyola University Chicago. Ms. Schreiber is a member of Dr. Michael Collin's lab.

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"Having the chance to interact directly with faculty members, design projects with them, and conduct two years of solid research at Lake Forest College prepared me well for the future and helped to build my own career."

—Nijee Sharma Luthra '04

MD/PhD in Neuroscience
Loyola Stritch School of Medicine

Neurology Resident
University of California, Davis

Neurology Fellow
University of California, San Francisco

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Learn more about Lake Forest College's Neuroscience Program at
lakeforest.edu/neuroscience

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## Chicago Society for Neuroscience
## Career Achievement Award 2018

### Shubhik K. DebBurman, Ph.D.

Dr. DebBurman is the Disque D. and Carol Gram Deane Professor in the Department of Biological Sciences at Lake Forest College. He has dedicated his academic career to making outstanding contributions to neuroscience education, the advancement of neuroscience research, and public communication and outreach in the greater Chicago community and beyond. First, he has authored several scientific publications in the area of G-protein coupled receptors. Next, he has been an extremely active member of the Chicago chapter of Society for Neuroscience. His involvement has included serving as President (2013-15), initiating the undergraduate poster competition, and developing the Career Achievement Award. He has also organized (2016-present) the Chicago Brain Bee competition for area high school students. Finally, he has mentored over 240 Lake Forest College undergraduate students and served as chair on over 40 Senior Thesis Committees. Numerous students have gone on to earn advanced degrees in the Chicago Area, many of whom received prestigious grants and awards. In addition, he has a longstanding commitment to mentoring Chicago area graduate students and post-doctoral fellows in undergraduate teaching careers. Dr. DebBurman has been an asset to the neuroscience community in Chicago, and we are pleased to honor him with this award.

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Social and announcement of awards, recognition, election results

# MORNING PROGRAM

## Mentoring Panel

***Pritzker Auditorium***

8:00-8:45 AM With Keynote Speaker and Presidential Symposium Speakers
Moderated by: Drs. Simon Kaja and VIrgine Mansuy-Auber

## 2018 Chicago Career Achievement in Neuroscience Award

***Room A***

9:00-9:10 AM Recipient: Shubhik DebBurman, Ph.D. Lake Forest College
Presented by Dr. Doug Wallace and Dr. Anna Lysakowski

## PRESIDENTIAL SYMPOSIUM
## Sponsored by Takeda, Inc.

***Room A***

9:10-10:30 AM **"*New Developments in Neuroscience*"**
Chaired by Anna Lysakowski, Ph.D.

9:10-9:20 AM *Welcoming Remarks*

9:20-9:55 AM **Neuroinformatics and the Multiscale Brain: from genes to brain states and beyond** *(Abstract p. 23)*

**PSA** Sean Hill, Ph.D.
*Krembil Centre for Neuroinformatics, Centre for Addiction and Mental Health, Toronto, Blue Brain Project, EPFL, Switzerland*

9:55-10:30 AM **The Making of the Inner Ear** *(Abstract p. 23)*

**PSB** Doris Wu, Ph.D.
*NIH-NIDCD*

## KEYNOTE SPEAKER

***Room A***

10:30-11:30 AM *"***The Neurobiology of Helping: Lessons from Order Rodentia"** *(Abstract p. 22)*

Peggy Mason, Ph.D.
Department of Neurobiology, University of Chicago

# LUNCH BREAK

11:30-2:00 PM **Poster Viewing & Vendor Tables** ***Atrium, 3rd Floor***

12:00-12:15 PM **Poster Competitions**
12:00-12:45 PM Authors in group A present for competition
12:45-1:30 PM Authors in group B present for competition

**Post-doctoral Poster Competition**
Chaired by **Emma Childs, Ph.D. and Doug Wallace, Ph.D.**

**Graduate Student Poster Competition**
Chaired by **Latha Malaiyandi Ph.D. and Antonio Sanz-Clemente Ph.D.**

**Undergraduate Student Poster Competition**
Chaired by **Naomi Wentworth, Ph.D. and Bill Rochlin, Ph.D.**

***For poster titles and abstracts, go to pages 28 and 36, respectively.***

12:00-1:00 PM **Themed Lunch Tables (open to all Trainees)** ***Room A***

Chaired by Aaron Schirmer, Ph.D.

***"Diversity in Careers"***
***Know more about your professional options***

**Table 1** ***Research and Teaching in Academia***
Dr. Margaret Bell, Assistant Professor, Biological and Health Sciences Departments, DePaul University
Dr. Cindy Voisine, Assistant Professor, Department of Biology Northeastern Illinois University
Dr. Philip Hockberger, Associate Vice President for Research, Northwestern University

**Table 2** ***Corporate Careers***
Dr. Eileen Hartman, Director of Publication, Takeda US Medical Affairs, Takeda Pharmaceuticals USA, Inc.
Ms. Maggie McCue, Clinical Science Director, Takeda US Medical Affairs, Takeda Pharmaceuticals USA, Inc.

**Table 3** ***Corporate Careers***
Ms. Jill Erickson, Director of Medical Education, Takeda US Medical Affairs, Takeda Pharmaceuticals USA, Inc.
Dr. Eric Mohler, Senior Research Scientist III, Neuroscience Drug Discovery, AbbVie Inc.

**Table 4** ***Alternative Careers***

Dr. Angel Alvarez, Manager, Stem Cell Core
Northwestern University, Feinberg School of Medicine

Dr. Garry Cooper, Co-founder/CEO Rheaply, Inc.

Dr. Philip E Hockberger, Associate Vice President for Research
Northwestern University, Feinberg School of Medicine

Dr. Gabrielle B. Edgerton, Founder, Principal Consultant, Red Pen Scientific

**12:15-1:15 PM** **Dr. Mason and Graduate Student Symposium participants lunch** ***Room E***

# AFTERNOON PROGRAM

## GRADUATE STUDENT SYMPOSIUM *(For abstracts see p. 24)* ***Room A***

2:00- 3:35 PM Chaired by Joanne O'Keefe, Ph.D., Rush University

2:00-2:05 PM **Introduction**
Joanne O'Keefe, Ph.D., Rush University

2:05-2:20 PM **Peripheral immune challenge increases *in vivo* firing of basolateral amygdala neurons in adult male rats**
**GS1** Soumyabrata Munshi
*Departments of Cellular and Molecular Pharmacology and Neuroscience, Rosalind Franklin University of Medicine and Science*
**Advisor:** Amiel Rosenkranz

2:20-2:35 PM **Melanopsin phototransduction is repurposed by ipRGC subtypes to shape the function of distinct visual circuits**
**GS2** Takuma Sonoda
*Department of Neurobiology; Northwestern University Inter-departmental Neuroscience Program; Northwestern University*
**Advisor**: Tiffany Schmidt

2:35-2:50 PM **Increased miR-137 in the adult amygdala drives anxiety and alcohol intake after adolescent alcohol exposure**

**GS3** Evan J. Kyzar
*Center for Alcohol Research in Epigenetics, Departments of Psychiatry and Anatomy and Cell Biology; University of Illinois at Chicago*
**Advisor**: Subhash C. Pandey

2:50-3:05 PM **Unique gait, balance and tremor profiles distinguishes Fragile X- Associated Tremor/Ataxia Syndrome from Parkinson Disease and essential tremor.**
**GS4** Erin Robertson
*Departments of Cell & Molecular Medicine and Neurological Sciences, Rush University*
**Advisor**: Joan A. O'Keefe

3:05-3:20 PM **Feature encoding at the somatosensory periphery**
**GS5** Aneesha Suresh
*Department of Organismal Biology, University of Chicago*
**Advisor**: Sliman Bensmaia

3:20-3:35 PM **Risky teen drinking and generational consequences: how parental preconception alcohol exposure impacts offspring development**
**GS6** Anna Dorothea Asimes
*Department of Cell and Molecular Physiology, Loyola University Chicago*
**Advisor:** Toni Pak

## PLENARY AFTERNOON SYMPOSIUM-A *(for abstracts, see p. 23)* ***Room A***

3:45-5:30 PM ***"PROGRESS IN MOVEMENT DISORDERS"***
Chaired by **Drs. Joanne O'Keefe & Orly Lazarov**

3:45-3:50 PM ***INTRODUCTION***
Dr. Joanne O'Keefe

3:50-4:15 PM **Exercise and neuroprotection and clinical biomarkers in Parkinson's Disease**
**PASA1** Dr. Daniel Corcos
Physical Therapy and Human Movement Sciences, Northwestern University

4:15-4:40 PM **Niemann Pick Type C and scientific serendipity: Can we capitalize?**
**PASA2** Dr. Elizabeth Berry-Kravis
Department of Pediatrics, Rush University

4:40-5:05 PM **Probing the developmental roots of neurodegeneration**
**PASA3** Dr. Puneet Opal
Ken and Ruth Davee Department of Neurology and Department of Cell and Molecular Biology, Northwestern University

5:05-5:30 PM **New developments in deep brain stimulation**
**PASA4** Dr. Konstantin V. Slavin
Department of Neurosurgery, University of Illinois at Chicago

## PLENARY AFTERNOON SYMPOSIUM-B

***Pritzker Auditorium***

*(for abstracts, see p. 23)*

3:45-5:30 PM ***"BIOMEDICAL ENGINEERING APPLICATIONS IN NEUROSCIENCE"***
Chaired by **Drs. Kelly Langert & Kaiwen Kam**

3:45-3:50 PM **Introduction**
Dr. Kelly Langert

3:50-4:15 PM **Biodegradable nanoparticle approach for the treatment of multiple sclerosis and spinal cord injury**
**PASB1** Dr. Stephen Miller
Department of Microbiology-Immunology, Northwestern University

4:15-4:40 PM **Personalizing deep brain stimulation for treating epilepsy -- a neural engineering approach**
**PASB2** Dr. David Mogul
Department of Biomedical Engineering, Illinois Institute of Technology

4:40-5:05 PM **Cerebral blood flow and oxygen supply in the brain**
**PASB3** Dr. Andreas A. Linninger
Department of Bioengineering, University of Illinois at Chicago

5:05-5:30 PM **Deceptive technology for stroke rehabilitation**
**PASB4** Dr. James Patton
Department of Department of Bioengineering University of Illinois at Chicago

## EVENING PROGRAM

## AWARD CEREMONY, BUSINESS MEETING AND SOCIAL

***Atrium, Room A***

5:40-7:00 PM **Wine and Cheese Social ("EtOH Receptor Binding Study")**

**Election Results**
**Recognition of Councilors**

**Recognition of Chicago area students and student organizations for excellence in neuroscience education and outreach**

2018 Chicago Brain Bee winners
2018 Lake Forest College Neuroscience Student Organization SYNAPSE
2018 Northwestern University Brain Awareness Organization (NUBAO)

**Announcement of prize winners**

**Undergraduate Student Poster Competition**
Presented by Naomi Wentworth, Ph.D., Lake Forest College

**Graduate Student Poster Competition**
Presented by Antonio Sanz-Clemente, Ph.D., Northwestern University

**Post-doctoral Fellow Poster Competition**
Presented by Doug Wallace, Ph.D., Northern Illinois University

**Graduate Student Symposium**
Presented by Joanne O'Keefe, Ph.D., Rush University

# ABSTRACTS

## *KEYNOTE LECTURE*

### THE NEUROBIOLOGY OF HELPING: LESSONS FROM ORDER RODENTIA

Peggy Mason, Ph.D.

*Professor of Neurobiology, University of Chicago, Chicago, IL*

Among mammals, social interactions are critical for survival of the individual and the species. Moreover, by facilitating safety, food procurement, shelter and well-being, sociality also allows for longer, more rewarding lives. Pro-social behavior, or helping, that is provided between members of a group increases social cohesion of that group. Given the evolutionary advantages of pro-social behavior, it is not surprising that rats help other rats just as primates, including humans, do. Recently, my laboratory has demonstrated that rats deliberately liberate a conspecific trapped within a restrainer. Helping persists even when the free rat is unable to physically interact with the liberated rat. Moreover, helping is socially selective, occurring only for rats of a familiar type but independent of individual familiarity.

A key question is the motivation that drives rats to help another in distress. We have demonstrated that some degree of affective distress within the helper is required for motivating helping. Yet, to our surprise, helper rats help even when the victim rat shows no distress, evidence that cognitive empathy can motivate a rat witness to action. An additional motivating factor for helping is a rat's assessment of other rats' assessments of the trapped rat. Reminiscent of the human bystander effect, rats are more likely to help in the presence of naïve rats and less likely to help in the presence of non-helper (confederate) rats. In sum, the conservation of complex motivated helping behavior across divergent mammals raises the exciting possibility that we can learn about the social influences on human sociality from rats.

# PRESIDENTIAL SYMPOSIUM

## NEW DEVELOPMENTS IN NEUROSCIENCE

### PSA
### NEUROINFORMATICS AND THE MULTISCALE BRAIN: FROM GENES TO BRAIN STATES AND BEYOND

Sean Hill, Ph.D.

Krembil Centre for Neuroinformatics, Centre for Addiction and Mental Health, Toronto. Blue Brain Project, EPFL, Switzerland

Understanding the brain requires an integrated view of different scales of organization spanning genes, channels, cells, microcircuits, brain regions and their roles in behaviour from perception to action and in different states including wakefulness and sleep. Neuroinformatics is a key tool in organizing, analyzing and modeling the brain across scales using data-driven approaches. This talk will start with data-driven approaches to reconstructing brain circuitry and describe the neuroinformatics necessary to enable large-scale computational modeling in basic and clinical research.

### PSB
### THE MAKING OF THE INNER EAR

Doris Wu, Ph.D.

National Institute on Deafness and Other Communicative Disorders, National Institutes of Health, Bethesda, MD

The inner ear is a structurally complex organ that is responsible for detecting sound and maintaining balance. These modalities are detected by sensory hair cells within various sensory organs of the inner ear. On top of each hair cell is a stereociliary bundle, also known as the hair bundle, which is comprised of a kinocilium (the true cilium) and a number of specialized microvilli, organized in a stair-case fashion. The deflection of a hair bundle in response to sound or head movements opens the mechanosensory transduction channels on the tip of the stereocilia, which allow positive ions to enter and activate the sensory hair cell. However, only deflection of the hair bundle towards a specific direction will open the transduction channels. Therefore, the orientation of the hair bundle on the apical surface determines the directional selectivity of the hair cell. Based on this property of the hair bundle, each sensory organ of the inner ear exhibits a defined hair bundle orientation pattern that is tailored to its function. Notably, in the two vestibular organs responsible for detecting linear head movements, the maculae of the utricle and saccule, an imaginary line of polarity reversal (LPR) can be drawn which divides each organ into two regions of opposite hair bundle orientation. This presentation will focus on the role of a transcription factor, Emx2, in establishing the LPR in the macular organs.

## PLENARY AFTERNOON SYMPOSIUM A

## PROGRESS IN MOVEMENT DISORDERS

### PASA1
### EXERCISE AS MEDICINE FOR PARKINSON'S DISEASE

D.M. Corcos

[1]Department of Physical Therapy and Human Movement Sciences, Northwestern University, Chicago IL

Parkinson's disease is a debilitating progressive, neurodegenerative movement disorder caused by damage to dopaminergic neurons. The early stages of the disease are characterized by motor symptoms but non-motor symptoms play a larger role in reducing quality of life over time. Medication and deep brain stimulation are very effective in treating the motor symptoms of the disease but are much less effective at treating the non-motor symptoms. In addition, they can be associated with side effects such as dyskinesias, in the case of medicine, and impaired cognition in the case of deep brain stimulation. There is now mounting evidence that exercise is therapeutically beneficial for Parkinson's disease and benefits both the motor and the non-motor symptoms of the disease. Progressive resistance exercise reduces the symptoms of the disease, increases muscle strength, movement speed, facilitates muscle activation and improves measures of cognitive abilities including attention and short term-memory. High intensity endurance exercise improves oxygen consumption, as measured by $VO_2$ max, and delays the rate at which Parkinson's disease progresses. Finally, tai chi improves balance. Although the mechanisms by which exercise positively affect the disease are not known, potential candidates include: improved cortical vascularity, increased cortical thickness, increased brain connectivity, increased dopamine metabolism, angiogenesis, neurogenesis, neuroplasticity, anti-inflammatory effects, improved mitochondrial function and oxidative stress, and increased levels of neurotrophic factors such brain derived neurotrophic factor. Collectivity, a multimodal exercise program reduces the symptoms of the disease, delays the rate at which the disease progresses and improves balance. The exercise program should include progressive resistance exercises two times per week, endurance exercise three times per week, and balance two times per week. If exercise was a pill, there would be no doubt that people would take it.

*These studies were funded by research awards from the National Institute of Health (NS074343 and NS28127).*

**PASA2**

**NIEMANN-PICK TYPE C AND SCIENTIFIC SERENDIPITY: CAN WE CAPITALIZE?**

Elizabeth Berry-Kravis

Elizabeth Berry-Kravis MD PhD,[1] Jamie Chin BS,[2] Anne Hoffmann PhD,[3] Amy Winston AuD,[4] Robin Stoner AuD,[4] Lisa LaGorio PhD MPH,[4] Katherine Friedmann RN,[2] Mariana Hernandez MD,[2] Daniel S. Ory MD,[5] Forbes D Porter MD PhD,[6] Joan A. O'Keefe PhD[7]

*[1]Departments of Pediatrics, Neurological Sciences, Biochemistry, Rush University Medical Center, Chicago, IL [2]Department of Pediatrics, Rush University Medical Center, Chicago, IL [3]Departments of Pediatrics and Communication Disorders and Sciences, Rush University Medical Center, Chicago, IL [4]Department of Communication Disorders and Sciences, Rush University Medical Center, Chicago, IL [5]Diabetic Cardiovascular Disease Center, Washington University School of Medicine, St. Louis, MO [6]Program in Developmental Endocrinology and Genetics, Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH, Bethesda, MD [7]Departments of Cell and Molecular Medicine and Neurological Sciences, Rush University Medical Center, Chicago, IL*

Neimann-Pick type C disease (NPC) is a highly variable but inexorably progressive neurodegenerative disorder caused by defects in the NPC1 or NPC2 genes, and associated with defective cholesterol transport and lysosomal storage of cholesterol. This talk will summarize the discovery of 2-hydroxypropyl-beta-cyclodextrin (HP-B-CD) as a potential treatment to stabilize or slow disease progression based on work in animal models and will describe the translation effort to achieve approval of this treatment in humans with NPC1. Challenges of translating findings in animal models to patients in rare neurodegenerative diseases will be described as well as progress to date in the drug development process for HP-B-CD in NPC1.

*This work was supported by the Hope for Hayley Fund, Samantha's Search for the Cure Fund, Vtesse, Inc., Sucampo, K01 HD088762-01 (JOK), NS 081985 (DSO), and a Rush Schweppe Translational Science Consortium grant (JOK). FDP is supported by the Intramural Research Program of the Eunice Kennedy Shriver National Institute of Child Health and Human Development.*

***PASA3***

**PROBING THE DEVELOPMENTAL ROOTS OF NEURO-DEGENERATION**

Puneet Opal

Chandrakanth Reddy Edamakanti [1], Jeehaeh Do [2], Alessandro Didonna [3], Marco Martina [2] and Puneet Opal[1,4]

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Spinocerebellar ataxia type 1 (SCA1) is an adult-onset neurodegenerative disease caused by a polyglutamine expansion in the protein ATXN1, which is involved in transcriptional regulation. Although symptoms appear relatively late in life, primarily from cerebellar dysfunction, pathogenesis begins early, with brain-wide transcriptional changes detectable as early as a week after birth in SCA1 knock-in mice. Given the importance of this postnatal period for cerebellar development, we asked whether this region might be developmentally altered by mutant ATXN1. We found that expanded ATXN1 stimulates the proliferation of postnatal cerebellar stem cells in SCA1 mice. These hyper-proliferating stem cells tended to differentiate into GABAergic inhibitory interneurons rather than astrocytes; this significantly increased the GABAergic inhibitory interneuron synaptic connections, disrupting cerebellar Purkinje cell function in a non-cell autonomous manner. We confirmed the increased basket cell-Purkinje cell connectivity in human SCA1 patients. Mutant ATXN1 thus alters the neural circuitry of the developing cerebellum, setting the stage for the later vulnerability of Purkinje cells to SCA1. We propose that other late-onset degenerative diseases may also be rooted in subtle developmental derailments.

**PASA4**
**NEW DEVELOPMENTS IN DEEP BRAIN STIMULATION**

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Over the last several decades, deep brain stimulation (DBS) has become a commonly used approach in treatment of multitude of neurological and psychiatric conditions. Currently, it is considered a standard of surgical care for patients with movement disorders, such as Parkinson disease (PD) and essential tremor (ET), and is used on an investigational device exemption basis for dystonia and obsessive-compulsive disorder.

Despite its widespread worldwide acceptance and recognized safety and efficacy, multiple issues remain unsolved. To address them, we are now exploring various technological innovations aimed at optimization of the surgical procedure and the way stimulation is delivered to the brain. The presentation will cover several problems related to variability of targeted structures, stimulation-related side effects and development of tolerance, and their theoretical and practical solutions.

Recent advances in DBS technology include introduction of intraoperative visualization and asleep surgery, recent approval of directional stimulation, modeling of electrophysiological signals, and development of real-time feedback that allows one to modify stimulation parameters based on the brain activity, chemical indicators or surface EMG data.

Technological developments in the field of DBS are expected to streamline surgical intervention, improve side effect profile, optimize stimulation parameters and individualize settings based on the closed loop mechanism.

## *PLENARY AFTERNOON SYMPOSIUM B*

**BIOMEDICAL ENGINEERING**
**APPLICATIONS IN NEUROSCIENCE**

**PASB1**

**A BIODEGRADABLE NANOPARTICLE APPROACH FOR THE TREATMENT OF MULTIPLE SCLEROSIS AND SPINAL CORD INJURY (SCI)**

Stephen Miller

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Ag-specific tolerance is the desired therapy for immune-mediated diseases. Our recent phase I clinical trial showed that infusion of myelin peptide-coupled autologous apoptotic PBMCs induces dose-dependent regulation of myelin-specific T cell responses in MS patients. Experiments in EAE and T1D models showed that antigen-coupled apoptotic leukocytes accumulate in the splenic marginal zone (MZ) and are engulfed by $F4/80^+$ MZ macrophages and $CD8^+$ DCs inducing upregulation of PD-L1 in an IL-10-dependent manner. Tolerance results from the combined effects of PD-L1/PD-1-dependent T cell anergy and activation of Tregs recapitulating how tolerance is normally maintained in the hematopoietic compartment in response to uptake of senescing blood cells.

To further advance clinical translation of tolerogenic therapies, we have shown that long-lasting tolerance is inducible by i.v. administration of (auto)antigens covalently linked to 500nm carboxylated poly(lactide-*co*-glycolide) (PLG) nanoparticles (Ag-NP) abrogating development of Th1/Th17-mediated autoimmune disease (EAE and T1D) and Th2-mediated allergic airway disease when used prophylactically and ameliorated progression of established disease when administered therapeutically. Ag-NP-induced tolerance is mediated by the combined effects of cell-intrinsic anergy and regulatory T cell (Treg) activation and is dependent on route of administration, particle size and charge, uptake by MZ macrophages via the MARCO scavenger receptor, and can be induced either by NP covalently coupled with or encapsulating the (auto)antigen. Additionally, we have shown that i.v. infusion of 'naked' carboxylated PLG NP targets inflammatory monocytes/macrophages in a MARCO-dependent fashion leading to their sequestration in the spleen and eventual apoptosis and is a potent therapy for ameliorating acute inflammatory diseases including spinal cord injury (SCI). These findings demonstrate the utility of Ag-NP as a novel, safe and cost-effective means for inducing antigen-specific tolerance for therapy of MS and other (auto)immune-mediated diseases using an FDA-approved biomaterial easily manufactured under GMP conditions.

*Supported by grants from NIH, NMSS, JDRF and Cour Pharmaceutical Development Company.*

**PASB2**
**PERSONALIZING DEEP BRAIN STIMULATION FOR TREATING EPILEPSY -- A NEURAL ENGINEERING APPROACH**

David Mogul (IIT)

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Deep brain electrical stimulation (DBS) is a treatment modality being explored for many neurological diseases and is a potentially potent means for disrupting the aberrant rhythms that arise during the epileptic seizures that afflict over 1% of the population. However, current DBS protocols typically employed are formulated *a priori* and do not reflect the electrophysiological dynamics within the brain as seizures arise which may underlie their limited efficacy. Furthermore, most stimulation paradigms in therapeutic devices seek to reduce the frequency of seizure onset but are not specifically tailored to terminate a seizure once ictal activity has initiated simply because past efforts at this goal have not yet shown strong efficacy. Our lab has been investigating how the efficacy of DBS to terminate seizures could be

improved using endogenous dynamics to inform stimulation protocols. Multi-site brain dynamics within the circuit of Papez were calculated in a chronic rat limbic epilepsy model induced via LiCl/pilocaprine i.p. injections. Stimulation/recording electrodes were placed in the CA3 region of left and right hippocampi and the anteromedial nucleus of left thalamus. Deconvolution of local field potentials using empirical mode decomposition (EMD) and phase synchrony analysis revealed multisite coherence as seizures approached natural termination that could not be detected with Fourier analysis. Multisite stimulation used charge-neutral biphasic square waves at frequencies observed during natural seizure termination. Synchronization of electrical activity across sites occurred as both spontaneous and evoked seizures naturally terminated in freely-moving rats. Further, the location and frequency (7Hz to 300Hz) of the synchrony varied between subjects but was stable in time within each animal. DBS efficacy was significantly more effective at rapidly stopping seizures when the frequency and location of multi-site stimulation reflected the endogenous synchrony dynamics observed in each subject as seizures naturally terminated. Furthermore, applying the same analytical techniques to intracranial recordings from human epilepsy patients revealed similar elevated coherence at natural seizure termination suggesting that this may also be how the brain naturally arrests epileptic seizures in humans. These results support the approach of personalizing DBS protocols to individual endogenous rhythms that mimic how brains naturally resolve epileptic seizures. This methodology may significantly improve the overall efficacy of this potentially important therapy for seizure termination and may also show relevance to seizure prevention.

*These studies were funded by a research award from the National Institutes of Health (NINDS R01 NS092760) to*

**PASB3**
**CEREBRAL BLOOD FLOW AND OXYGEN SUPPLY IN THE BRAIN**

Andreas Linninger

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Intracranial dynamics concerns the physical and chemical interactions of cerebral vasculature, cerebrospinal fluid (CSF) and the brain tissue. Despite ever more accurate and detailed imaging modalities, fluid and momentum exchange between blood and CSF is poorly understood. To answer questions about key metabolic functions of the brain, we harness predictive mathematical models informed by three-dimensional dynamic imaging data. This lecture will survey current knowledge of cerebral blood flow and CSF dynamics including natural CSF flow patterns and mixing phenomena of solute in CSF. A theory of CSF flow will be shown to explain the causes of brain disease and point to strategies for effective drug delivery to the central nervous system. In addition, novel hypotheses for microcirculatory water exchange will be presented. These new insights on cerebral fluid clearance and perivascular fluid transport may hold the key to understanding and treating several pathologies including brain edema, hydrocephalus and Alzheimer's disease.

**PASB4**
**DECEPTIVE TECHNOLOGY FOR STROKE REHABILITATION**

James Patton

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It has been shown to be powerful to leverage what we know (i.e., existing models) about neural adaptation in neurorehabilitation. These models stem from how people learn through mistakes, and several techniques on using deception and distortion for enhanced training conditions. Through smart mechanical designs and programs, new techniques for motor teaching in therapy (and beyond) exist. Moreover, clinical trials in this field have not proven to be very effective because of few samples and restrictive inclusion criteria. I will show how such "small data" problems lend themselves well to scrutiny and interrogation from modern predictive modeling and validation techniques. This suggests a more "organic" approaches for gathering data.

## *GRADUATE STUDENT SYMPOSIUM*

**GS1**

**PERIPHERAL IMMUNE CHALLENGE INCREASES *IN VIVO* FIRING OF BASOLATERAL AMYGDALA NEURONS IN ADULT MALE RATS**

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Peripheral inflammation is often associated with changes in mood and emergence of depressive behavior, and is characterized by a group of physical manifestations including lethargy, malaise, anhedonia, listlessness, decreased appetite and fever. These changes are mediated at the cellular level by pro-inflammatory cytokines like interleukin (IL)-1β, IL-6 and TNF-α. The basolateral amygdala (BLA) is a key brain region involved in mood and may mediate some of the behavioral effects of inflammation. However, it is unknown whether peripheral inflammatory state affects the activity of BLA neurons. Therefore, we tested the effects of inflammation on BLA physiology in adult male Sprague-Dawley rats. In the first set of experiments, rats were treated acutely with IL-1β (1 μg, single dose, i.p.). Behavioral studies showed reduced open-field locomotion and home-cage mobility consistent with features of sickness-like behavior. Using *in vivo* single-unit extracellular electrophysiological recordings, we found that IL-1β treatment acutely (<30 min) increased the spontaneous BLA neuronal firing followed by a return to the baseline level (≥30 min). The effects were significantly more prominent in the basal nucleus of the BLA complex. Increase in BLA firing rate was also seen after treatment with another longer-lasting inflammagen, lipopolysaccharide (250 μg / kg, single dose, i.p.). The findings demonstrate a rapid effect of acute peripheral inflammation on BLA activity. In a second set of experiments, chronic low-grade subthreshold inflammation was induced with low-dose of IL-1β (0.25 μg, i.p.) administered twice / day for five consecutive days. On the sixth day, we observed anxiety-like behavior and significantly increased spontaneous BLA neuronal firing rate. These results indicate that chronic subthreshold inflammation can induce anxiety behavior and amygdala hyperactivity even in the absence of overt sickness. Taken together, our findings suggest a link between BLA neuronal firing and triggering of behavioral consequences of both acute and chronic-subthreshold peripheral inflammation.

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**GS2**

**MELANOPSIN PHOTOTRANSDUCTION IS REPURPOSED BY INTRINSICALLY PHOTOSENSITIVE RETINAL GANGLION CELL SUBTYPES TO SHAPE THE FUNCTION OF DISTINCT VISUAL CIRCUITS**

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The mammalian retina contains three classes of photoreceptors: rods, cones and intrinsically photosensitive retinal ganglion cells (ipRGCs). ipRGCs express the photopigment melanopsin and were initially thought to be a homogeneous population of cells that only drive subconscious visual behaviors such as circadian photoentrainment and pupil constriction. However, recent evidence has demonstrated that ipRGCs are in fact a heterogeneous population consisting 5 subtypes (M1-M5), with some subtypes also mediating conscious visual perception (image-forming vision). A major unanswered question is how the same photopigment, melanopsin, influences such vastly different visual functions. Our data show that melanopsin is required for normal functioning in an ipRGC subtype that mediates image-forming vision (M4 ipRGCs). Melanopsin achieves this influence by increasing the excitability of M4 ipRGCs via closure of leak potassium channels. In contrast, melanopsin phototransduction in M1 ipRGCs targets canonical transient receptor potential (TRPC) channels to influence subconscious visual functions. Thus, melanopsin signaling is repurposed by ipRGC subtypes to shape distinct visual behaviors.

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**GS3**

**INCREASED MIR-137 IN THE ADULT AMYGDALA DRIVES ANXIETY AND ALCOHOL INTAKE AFTER ADOLESCENT ALCOHOL EXPOSURE**

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Adolescent binge drinking is a risk factor for alcohol use disorder (AUD) and comorbid anxiety in adulthood. As microRNAs (miRNAs) are involved in neurodevelopment, we investigated the role of miRNAs in the amygdala in adolescent intermittent ethanol (AIE) exposure-induced epigenetic reprogramming and adult psychopathology. Rats were exposed to 2g/kg ethanol (2 days on/off; AIE) or intermittent n-saline (AIS) during postnatal days (PND) 28-41 and allowed to grow to adulthood for analysis of behavior, miRNA profiling, and related epigenetic measures in the amygdala. AIE adult rats show anxiety-like behaviors and differential expression of miRNAs by microarray. miR-137, a crucial neurodevelopmental miRNA, was significantly increased and its target genes lysine-specific demethylase 1 (*Lsd1*) and *Lsd1+8a* were decreased in the AIE adult amygdala. Infusion of an antagomir specific to miR-137 directly into the central nucleus of the amygdala

(CeA) rescues alcohol drinking and anxiety-like behaviors due to normalization of decreased *Lsd1* expression, decreased LSD1 occupancy, and an associated increase in repressive H3K9me2 at the *Bdnf IV* promoter in AIE adult rats. We measured miRNA expression in human postmortem amygdala and noticed a significant increase in miR-137 in tissue from patients diagnosed with AUD compared to controls. Taken together, AIE causes an enduring increase in amygdala miR-137 leading to epigenetic reprogramming, anxiety, and higher alcohol intake in adulthood. Our translational results highlight miR-137 as a potential therapeutic target for anxiety and AUD susceptibility in both humans and rodents.

*This work was supported by the National Institute on Alcohol Abuse and Alcoholism at the National Institutes of Health UO1AA-019971 (NADIA project) & P50AA022538 grants to SCP and F30AA024948 to EJK.*

**GS4**

**FRAGILE X-ASSOCIATED TREMOR/ATAXIA SYNDROME, PARKINSON DISEASE, AND ESSENTIAL TREMOR PATIENTS DEMONSTRATE DISTINCT GAIT, BALANCE AND TREMOR DEFICITS UNDER NORMAL, ENVIRONMENTALLY CHALLENGING, AND DUAL-TASK CONDITIONS**

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Fragile X-associated tremor/ataxia syndrome (FXTAS), a neurodegenerative disease affecting carriers of a 55-200 CGG repeat in the *fragile X mental retardation 1* gene, may be initially diagnosed as Parkinson disease (PD) or essential tremor (ET) due to overlapping motor symptoms. Challenging and dual-task (DT) cognitive-motor interference paradigms can reveal subtle gait and balance impairments, and tremorography correlates with clinical tremor rating scales. Therefore, we sought to compare FXTAS, PD, ET and controls using quantitative measures of gait, balance, and tremor. Subjects with FXTAS (n = 22), PD (n = 23), ET (n = 20) and controls (n= 20) underwent gait and balance testing with an inertial sensor system (APDM™; Oregon). Instrumented Timed Up and Go (i-TUG) and 2-minute walk (2MWT) tests were used to test gait and the i-SWAY to test balance, with a verbal fluency DT. Subjects underwent tremorography using the ETSense™ sensor with the Kinesia ™ system. Cognitive tests were also administered to explore the impact of cognition on motor performance. PD had reduced stride length and greater DT costs for stride length and velocity compared to FXTAS at preferred speed and DT 2MWT. On the i-TUG, PD had reduced sit to stand peak velocity compared to FXTAS. Reduced verbal fluency and information processing speed in FXTAS associated with worse performance on i-TUG movement transitions. Stride length distinguished FXTAS from ET in the regression model. On the i-SWAY, FXTAS had greater sway variability compared to PD during DT with vision removed. When base of support was reduced they had greater jerk, total sway area and sway variability compared to PD and greater sway variability compared to ET. Unique correlations were found between cognitive and balance measures in all groups. In the regression model, greater sway variability distinguished FXTAS from PD and ET during DT and greater total sway area distinguished FXTAS from PD. On tremorography, FXTAS had greater kinetic tremor than PD and increased bradykinesia than ET. PD had worse bradykinesia compared to FXTAS. In FXTAS, PD and ET, slower processing speed and verbal fluency associated with worse dysrhythmia and bradykinesia. Finger tap speed distinguished FXTAS from ET and rapid alternating movements amplitude distinguished FXTAS from PD in the regression model. This pilot data demonstrates that FXTAS, PD, and ET exhibit distinct deficits in gait, balance and tremor profiles under normal, challenging and DT conditions, suggesting that these quantitative measures may be sensitive to distinguish FXTAS from PD and ET and may have clinical utility in the appropriate differential diagnosis of these disorders.

*This study was funded by a National Fragile X Foundation Summer Fellowship Award to ER and a NIH grant (K01 HD088762) to JO.*

**GS5**

**CODING OF EDGE ORIENTATION IN AFFERENT RESPONSES OF MACAQUES**

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One of the tactile features extracted by the somatosensory system during object manipulation is the orientation of object edges on the skin. We examined edge orientation processing in the glabrous skin of non-human primates by presenting both scanned and static indented bar stimuli to the receptive field area. First, we show that monkeys exhibit complex receptive field structure, consisting of multiple hot spots, similar to the structure found in humans. Second, we confirm that spike trains elicited in response to stimulating these complex receptive fields can classify edge orientation. Third, we find that orientation signals in tactile afferents are often not robust to changes in the stimulus unrelated to its orientation, for example amplitude modulation. In contrast, cortical neurons generalize orientation selectivity across different stimulation conditions. Overall, our results suggest that this peripheral signal might be more important during development to guide the refinement of orientation tuning of cortical neurons than it is in the mature cortex.

**GS6**

**MECHANISMS AND CONSEQUENCES OF EPIGENETIC INHERITANCE FOLLOWING PARENTAL PRECONCEPTION ALCOHOL EXPOSURE**

AnnaDorothea Asimes, Chun K Kim, Toni R Pak

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Recent advances in genomics research have revealed that preconception behaviors and experiences of mothers and fathers, including diet, environmental toxicants, and drug abuse, can impact future offspring through epigenetic mechanisms. This means that the risky behaviors of young people, such as the extremely popular practice of binge drinking, have potentially far-reaching consequences for generations to come. While there has been considerable research into fetal alcohol exposure and parental alcoholism, there has yet to be sufficient investigation into the mechanism of epigenetic inheritance or the functional consequences of parental preconception binge pattern alcohol abuse. We tested the hypothesis that parental preconception alcohol exposure can impact offspring through epigenetic inheritance of DNA methylation patterns in the hypothalamus, leading to impaired hypothalamic function during development. This hypothesis was tested using an established rodent model of repeated adolescent binge alcohol (EtOH) exposure. Wistar rats received 3g/kg of 20% (v/v) EtOH via oral gavage once daily for 3 days, then 2 days vehicle, and another 3 days EtOH at both early and late puberty (PND37, 67). Animals were paired for mating 24h after the last EtOH dose. Offspring were birthed normally and examined at different ages of development, both before and after weaning. Offspring which were treated with EtOH were given the same paradigm described above for their parents at PND37. This work reveals that alcohol-naïve juvenile male offspring of both maternal and paternal preconception alcohol exposure have genome-wide changes in methylation patterns in the hypothalamus, and that offspring have altered hypothalamic function resulting in modest phenotypic and behavioral changes lasting through pubertal development. Additionally, we show that parental preconception alcohol exposure does not confer advantages to offspring for improved EtOH metabolism or reduced stress response. These results suggest that parental preconception EtOH exposure during adolescence confers maladaptive traits to first generation offspring through epigenetic mechanisms, suggesting multigenerational transmission of the effects of binge EtOH exposure on the brain.

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**G- GRADUATE STUDENT COMPETETION**
**PD- POSTODOCTORAL STUDENT COMPETITION**
**UG- UNDERGRADUATE STUDENT COMPETITION**
**A/B- JUDGING GROUP A OR JUDGING GROUP B**

## POSTER ABSTRACT TITLES

### *THEME A. COGNITION AND BEHAVIOR*

**A1 UG-A**
**HOW DO ENERGY DRINKS MAKE YOU FEEL? EFFECTS OF ENERGY DRINK COMPONENTS ON ANXIETY BEHAVIOR AND CORTISOL RESPONSES IN ZEBRAFISH (*DANIO RERIO*)**
A.O. Alia, S.P. Pattison, H.L. Townsend, and M.L. Petrunich-Rutherford
*Department of Psychology, Indiana University Northwest*

**A2 UG-B**
**LIKE AN (UNSTRESSED) FISH OUT OF WATER: LACK OF EFFICACY OF A 30-SECOND NET STRESS ON ANXIETY BEHAVIOR AND CORTISOL RESPONSES IN YOUNG ADULT ZEBRAFISH (DANIO RERIO)**
A. Aponte and M. L. Petrunich-Rutherford
*Department of Psychology, Indiana University Northwest*

**A3**
**IDENTIFYING SEX DIFFERENCES IN THE BASOLATERAL NUCLEUS TO CENTRAL NUCLEUS CIRCUIT OF THE AMYDALA THROUGH ANTIDROMIC IN VIVO STIMULATION**
B. Avonts, J. Vantrease, A. Rosenkranz
*Department of Cellular and Molecular Pharmacology, Rosalind Franklin University of Science and Medicine*

**A4 UG-A**
**STRINGING TOGETHER SPECIES: COMPARING KINEMATIC AND TOPOGRAPHIC CHARACTERISTICS OF BIMANUAL STRING-PULLING BEHAVIOR IN HUMANS (HOMO SAPIENS) AND RATS (RATTUS NORVEGICUS)**
Joey G. Petersen, Brandi D. Schell, Mark T. Banovetz, Ashley A. Blackwell, Douglas G. Wallace
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**A5 UG-B**
**BEHAVIORALLY INHIBITED INDIVIDUALS EXHIBIT MORE FREQUENT AROUSALS DURING SLEEP COMPARED TO NON-BEHAVIORALLY INHIBITED INDIVIDUALS**
Buchholz K.[1], Cook-Snyder D.R.[1], and Miller J.R.[2]
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**A6 PD-A**
**CHILDHOOD SOCIOECONOMIC STATUS PREDICTS TONG TERM COGNITIVE OUTCOMES FOLLOWING TRAUMATIC BRAIN INJURY**
S. Cohen-Zimerman[1], Z. R. Kachian[1], F. Krueger[2,3], B. Gordon[4,5], J. Grafman[1,6]
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**A7 UG-A**
**PARTIALLY REINFORCED SIGNALED LEVER PRESS CONDITIONING REVEALS DIFFERENCES IN THE EXPECTATION VERSUS THE PRESENCE OF SHOCK IN BEHAVIORALLY INHIBITED WISTAR-KYOTO RATS COMPARED TO SPRAGUE DAWLEY RATS**
L. Krenzke, H. Latham, T. Perlmutter, D.R. Cook-Snyder, D.Miller
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**A8 UG-B**
**IMAGE-BASED CLASSIFICATION OF BODY LANGUAGE POSES TO EMOTIONS USING CONVOLUTIONAL NEURAL NETWORKS (CNN)**
A. Tanveer, S. Banerji
*Department of Mathematics and Computer Science, Lake Forest College, Lake Forest, IL 60045*

**A9 UG-A**
**ATTENTION PATTERNS PREDICT MENTAL ARITHMETIC PERFORMANCE**
K. Meuli, R. Gyorfi, N. Wentworth
*Department of Psychology and Neuroscience Program, Lake Forest College, IL 60045*

**A10 UG-B**
**HEMISPHERIC PROCESSING OF EMOTION WORDS: THE EFFECTS OF SEX AND VALENCE**
H. Nabulsi, B. Tomkins, S. Virtue
*Department of Psychology, College of Science and Health, DePaul University*

**A11 UG-A**
**SAVINGS MEMORY IS ACCOMPANIED BY TRANSCRIPTIONAL CHANGES THAT PERSIST BEYOND THE DECAY OF RECALL**
Leticia Perez, Ushma Patel, Athira Jacob, Steven Farrell, Derek Steck, Irina Calin-Jageman, Robert Calin-Jageman
*Neuroscience Program, Dominican University*

**A12**
**FREQUENT BINGE DRINKERS EXHIBIT BLUNTED REWARDING EFFECTS OF METHAMPHETAMINE**
Yang, Hyerim; Bremmer, Michael; Lutz, Joseph; Childs, Emma
*Human Addiction Psychopharmacology Laboratory, Department of Psychiatry, University of Illinois at Chicago, Chicago, IL*

**A13 UG-A**
**STEP FATHERING BEHAVIOR OF NON-REPRODUCTIVE AMPHIPRION OCELLARIS AFTER REMOVAL OF THE BREEDING PAIR FROM THE GROUP**
Elizabeth Phillips, Alia Kirsch, Lotanna Ezenekwe, Justin S. Rhodes
*University of Illinois at Urbana-Champaign, Beckman Institute for Advanced Science and Technology, Department of Psychology*

**A14**
**DIVERGING ROLES OF SOMATOSENSORY CORTICAL SUB-REGIONS IN A WHISKER-SIGNALED ASSOCIATIVE LEARNING TASK**
Leah T. Vinson; Craig Weiss; John F. Disterhoft
*Department of Physiology and Northwestern University Interdepartmental Neuroscience*

**A15 G-A**
**HEMISPHERIC PROCESSING OF STRONGLY-CONSTRAINED AND WEAKLY-CONSTRAINED PREDICTIVE INFERENCES: BEHAVIORAL AND ELECTROPHYSIOLOGICAL EFFECTS**
Blaine Tomkins Sandra Virtue
*Department of Psychology, College of Science and Health, DePaul University*

**A16 PD-B**
**AGE-DEPENDENT DISRUPTION OF PREFRONTAL LOCAL FIELD POTENTIAL RESPONSES BY REPEATED CANNABINOID EXPOSURE DURING ADOLESCENCE IN FEMALE RATS**
Thomases D.R.[1], Molla H.M.[1,2], Cass D.K.[1], and Tseng K.Y.[1]
*[1]Department of Anatomy & Cell Biology, University of Illinois at Chicago – College of Medicine, Chicago, IL; 2Department of Cellular & Molecular Pharmacology, Rosalind Franklin University, North Chicago, IL*

**A17 UG-B**
**EFFECTS OF ACUTE AEROBIC EXERCISE ON BEHAVIORAL AND PREFRONTAL HEMODYNAMIC RESPONSES TO A FATIGUING COGNITIVE TASK**
Caryn Ausenhus[1], Gunnar Goebel[1], Kathryn Halldin[1], Derek Monroe[2], Brian Hunt[1], and Nathaniel Thom[3]
*[1]Department of Applied Health Science, Wheaton College, [2]Department of Neurology, University of California-Irvine, [3]Department of Biology, Wheaton College*

## *THEME B. DEVELOPMENT*

**B1 G-B**
**OTOLITH DYSFUNCTION DISRUPTS EXPLORATORY MOVEMENT ORGANIZATION ACROSS TWO DEVELOPMENTAL TIME POINTS**
Donaldson, T.N.[1], Miller, M.K.[1], Eash J.L[2], Yoder, R.M.[2], Wallace, D.G[1]
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**B2 G-A**
**DEVELOPMENTAL REGULATION OF PREFRONTAL PLASTICITY BY ENDOCANNABINOID SIGNALING IN VIVO**
Hanna M. Molla[1,2], Daniel R. Thomases[2], Kuei Y. Tseng[2]
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## *THEME C. DISORDERS OF THE NERVOUS SYSTEM*

**C1 G-B**

**Discovering a Novel Mechanism Underlying Sporadic Alzheimer's Disease.**

Jacqueline A. Bonds[1], Zhenlong Chen[2], Marcelo G. Bonini[3], Leon Tai, Jacob Haus[5], Richard D. Minshall[2,4], Orly Lazarov[1]
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**C2 PD-B**

**LEVERAGING LRRK2 BIOLOGY TO DEVELOP NOVEL THERAPEUTIC OPPORTUNITIES FOR PARKINSON'S DISEASE**

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**C3**

**TRANSCRIPTIONAL CHANGES IN FOREBRAIN AND CEREBELLUM OF A CLN2 NEURONAL CEROID LIPOFUSCINOSES MOUSE-MODEL**

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**C4**

**CHARACTERIZING CEREBRAL ENDOTHELIAL EXTRACELLULAR MATRIX PROTEINS IN THE PRESENCE OF HTRA1 DEFICIENT ASTROCYTES**

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**C5 PD-A**

**DIFFERENTIAL GENOME-WIDE METHYLATION IN ALCOHOL USE DISORDER SUBJECTS: FOCUS ON THE CORTICO-LIMBIC GLUCOCORTICOID RECEPTORS (NR3C1)**

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**C6 G-A**

**HYPERBARIC PRESSURE-INDUCED REACTIVE ASTROCYTOSIS RESULTS IN ELEVATED LEVELS OF OXIDATIVE STRESS IN PRIMARY OPTIC NERVE HEAD ASTROCYTES**

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**C7**

**AMPA RECEPTOR TRAFFICKING IN THE LIMBIC SYSTEMS OF RATS TREATED WITH PRAMIPEXOLE**

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**C8 G-B**

**DEVELOPMENTAL SYNAPSE MATURATION IS IMPAIRED IN INFANTILE NEURONAL CEROID LIPOFUSCINOSIS**

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**C9 PD-B**

**ACTIVATION OF PPAR-ALPHA BY THE ENDOCANNABINOID N-PALMITOYLETHANOLAMINE (PEA) IMPROVES PTSD-LIKE BEHAVIORAL PHENOTYPE BY NORMALIZING CORTICOLIMBIC ALLOPREGNANOLONE LEVELS**

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**C10 UG-A**

**ASSESSMENT OF DRUG THERAPIES ON A DROSOPHILA PARKINSON'S MODEL AND THEIR EFFECTS ON MOTOR FUNCTION AND AGGREGATE FORMATION**

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**C11 G-A**

**MECHANISM OF IRL 1620 INDUCING NEUROGENESIS FOLLOWING SPINAL CORD INJURY IN MICE EX VIVO**

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**C12 G-B**

**VIRAL VECTOR-MEDIATED A-SYNUCLEIN OVEREXPRESSION RAT MODELS OF PARKINSONIAN AND CEREBELLAR VARIANTS OF MULTIPLE SYSTEM ATROPHY**

David J. Marmion[1], Ronald J. Mandel[2], Deniz Kirik[3], Yaping Chu[1], Thomas J. McCown[4,5], Steven J. Gray[6,7,8], Jeffrey H. Kordower[1,9]
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**C13 PD-A**

**ELUCIDATING THE SYNAPTIC DEFECTS IN HEREDITARY SPASTIC PARAPLEGIAS USING IPSC CO-CULTURE MODELS**

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**C14 G-A**

**ENVIRONMENTAL AND GENETIC CONTRIBUTIONS IN AN ALS RAT MODEL: FAILED RECOVERY AND ENHANCED VENTRAL HORN INFLAMMATION AFTER PERIPHERAL NERVE INJURY**

S. Schram, D. Chuang, G. Schmidt, H. Piponov, C. Helder , J. Kerns, M. Gonzalez, F. Song, & J.A. Loeb
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**C15 UG-B**

**LOW-DOSE ASPIRIN STIMULATES DOPAMINE PRODUCTION: IMPLICATIONS FOR PARKINSON'S DISEASE**

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**C16**

**TRANSECTION RAPIDLY REORGANIZES THE CORTICAL ACTIN CYTOSKELETON OF THE NEURITE IN VITRO**

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**C17 PD-B**

**MITOCHONDRIAL OXIDATATIVE STRESS REGULATES TGF-β2 DEPENDENT REMODELING OF THE EXTRACELLULAR MATRIX IN HUMAN TRABECULAR MESHWORK CELLS**

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**C18 G-B**

**SINGLE NUCLEOTIDE POLYMORPHISMS OF GCH1 ASSOCIATES WITH SICKLE CELL DISEASE PAIN IN AFRICAN AMERICANS**

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**C19 G-A**

**LY6K PROMOTES GLIOBLASTOMA TUMORIGENESIS BY ENHANCEMENT OF ERK SIGNAL TRANSDUCTION**

Namratha Sastry, Tianzhi Huang, Angel A. Alvarez, Rajendra P. Pangeni, Xiao Song, Xuechao Wan, John Kessler, Cameron W. Brenann, Erik P. Sulman, Ichiro Nakano, Bo Hu, and Shi-Yuan Cheng
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**C20**

**KNOCKDOWN OF CORTICOTROPIN RELEASING HORMONE RECEPTOR 1 (CRF1) IN THE ROSTRAL PERICELLULAR REGION OF THE LOCUS COERULEUS (LC) NORMALIZES ANXIETY-LIKE BEHAVIOR IN FEMALE RATS SUBJECTED TO CORONARY ISCHEMIA AND REPERFUSION INJURY (IR)**

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**C21**

**EXAMINING THE STRIATAL PROTEOME IN DOPAMINE DEFICIENT MOUSE MODELS: IMPLICATIONS IN PARKINSON'S DISEASE**

S. Smith, J.F. Poulin., Y.Z. Wang, J. Savas, R. Awatramani
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**C22 UG-A**

**INSIGHT INTO PARKINSON'S DISEASE FROM YEASTS: GROWING EVIDENCE FOR SUMOYLATION AS A PROTECTIVE FACTOR AGAINST A-SYNUCLEIN TOXICITY**

Rosemary Thomas, Alexandra Roman, Morgan Marshall, Yoan Ganev, Galina Lipkin, and Shubhik K. DebBurman
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**C23 PD-A**

**REPURPOSING AN FDA APPROVED CARDIAC GLYCOSIDE FOR MYELIN REPAIR THERAPY IN COMBINATION WITH IMMUNE TOLERANCE IN MULTIPLE SCLEROSIS.**

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**C24 G-B**

**ACTIVATED MESENCHYMAL STEM CELLS INCREASE LONG-TERM RECOVERY FOLLOWING ISCHEMIC STROKE VIA REDUCTION OF MICROGLIA ACTIVATION AND INDUCTION OF OLIGODENDROGENESIS**

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**C25 UG-B**

**DIFFERENTIAL REGIONAL BUILDUP OF DISTINCT AB OLIGOMER SPECIES IN THE 5XFAD MOUSE MODEL OF ALZHEIMER'S DISEASE**

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**C26 G-A**

**CLOSED-HEAD INJURY MODEL OF REPEAT SUBCONCUSSION IN THE ADULT RAT**

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## *THEME E. HOMEOSTATIC AND NEUROENDOCRINE SYSTEMS*

**E1 UG-B**

**ACTIVATION OF HYPOTHALAMIC OXYTOCIN NEURONS IN RESPONSE TO FEAR CONDITIONING**

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**E2 UG-A**

**CONTINUOUS SOCIAL ISOLATION IMPAIRS FEAR EXTINCTION LEARNING: POSSIBLE ROLE OF THE OXYTOCIN RECEPTOR**

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**E3 UG-B**

**DO BEHAVIORALLY INHIBITED INDIVIDUALS EXHIBIT GREATER MINUTE VENTILATION AND BREATHING FREQUENCY THAN NON-INHIBITED INDIVIDUALS WHEN BREATHING ENHANCED CO2?**

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**E4 G-B**

**MODULATION OF BASOLATERAL AMYGDALA INPUTS INTO NUCLEUS ACCUMBENS BY GSK3B INHIBITION AND ANTERIOR CINGULATE CORTEX STIMULATION TRAINS**

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**E5 UG-A**

**A CIRCADIAN OUTPUT CENTER CONTROLLING FEEDING RHYTHMS IN DROSOPHILA**

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**E6**

**C2-DOMAIN PHOSPHORYLATED PROTEIN (C2CD5) INVOLVED IN HYPOTHALAMIC PROTEIN TRAFFICKING AND ENERGY BALANCE**

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**E7 UG-A**

**IDENTIFICATION OF CIRCADIAN OUTPUT GENES THAT AFFECT REST:ACTIVITY RHYTHMS IN DROSOPHILA**

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**E8 UG-B**

**EFFECTS OF PERINATAL POLYCHLORINATED BIPHENYLS (PCBS) ON DOPAMINERGIC AND INFLAMMATORY GENE EXPRESSION IN THE ADOLESCENT HYPOTHALAMUS AND PREFRONTAL CORTEX IN RATS.**

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## *THEME F. NEURONAL EXCITABILITY, SYNAPSES AND GLIA*

**F1 PD-B**

**GENOME-WIDE TRANSCRIPTIONAL CHANGES IN THE RAT HIPPOCAMPUS DURING WITHDRAWAL FROM CHRONIC ALCOHOL DRINKING**

Wei-Yang Chen, Hu Chen, Ying Chen, Huaibo Zhang, Harish R. Krishnan, Chunyu Liu, Dennis R. Grayson, Subhash C. Pandey, and Amy W. Lasek
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**F2 G-A**

**REGULATION OF NMDAR TRAFFICKING BY PROTEIN PHOSPHATASE 1**

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**F3 PD-A**

**UPTAKE AND ANTI-INFLAMMATORY EFFECTS OF MOLECULAR FORMS OF DOCOSAHEXAENOIC ACID IN MICROGLIA**

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**F4 UG-A**

**ATP-INDUCED EXTRACELLULAR ACIDIFICATION FROM RETINAL MÜLLER GLIA IS SENSITIVE TO EXTRACELLULAR CONCENTRATIONS OF SODIUM AND POTASSIUM.**

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**F5 PD-B**

**INPUT-SPECIFIC MODULATION OF PREFRONTAL AFFERENT DRIVE BY α7nAChR SIGNALING IN VIVO**

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**F6**

**METAPLASTICITY IN THE MOUSE MODEL OF FRAGILE X SYNDROME**

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**F7 G-B**

**DEFECTS IN LYSOSOME-AUTOPHAGOSOME REGULATION AND SYNAPTIC VESICLES IN ALZHEIMER'S DISEASE MOUSE MODELS AND HUMAN INDUCED NEURONS**

Sarah Mustaly[1], Megan Garstka, Nicolas Kapecki Kenneth D. Beaman[2], Alice Gilman-Sachs[2], Sean Schrank[1], Robert Marr[1], John McDaid[1], Grace Stutzmann[1]
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**F8 G-A**

**A COMPUTATIONAL MODEL OF TWO-PHOTON CALCIUM IMAGING IN SPINES AND DENDRITES OF CA1 PYRAMIDAL NEURONS USING A GENETICALLY ENCODED CALCIUM INDICATOR**

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**F9 G-B**

**NEURINFLAMMATORY CONTRIBUTION OF ASTROGLIAL CELLS IN A BINGE ETHANOL IN VITRO MODEL**

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**F10 G-A**

**MODELING THE DEVELOPMENT AND BINOCULAR MATCHING OF ORIENTATION SELECTIVITY IN THE MOUSE VISUAL CORTEX**

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**F11 PD-A**

**RAPID THREE-DIMENSIONAL SUPER-RESOLUTION IMAGING OF DENDRITIC SPINULE DYNAMICS IN CORTICAL MOUSE NEURONS**

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## *THEME G. NOVEL METHODS AND TECHNOLOGY DEVELOPMENT*

**G1**

**MUSCLE-NERVE-NERVE GRAFTING IMPROVES FACIAL REANIMATION IN RATS FOLLOWING FACIAL NERVE INJURY**

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**G2**

**PRE-IMPLANT MODELING FOR DEPTH LEAD PLACEMENT IN WHITE MATTER FOR MAXIMIZING DIRECT NEUROSTIMULATION THERAPY**

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**G3 G-B**

**PROTEASOME-TARGETED NANOBODIES ALLEVIATE PATHOLOGY IN A SYNUCLEIN-BASED PARKINSON'S DISEASE MODEL**

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**G4**

**PARAMETRIC SUBTRACTED POST-ICTAL DIFFUSION TENSOR IMAGING FOR GUIDING DIRECT NEUROSTIMULATION**

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**G5 UG-B**

**OPTIMIZING PREDICTION MODEL FOR A NONINVASIVE BRAIN COMPUTER INTERFACE PLATFORM USING CHANNEL SELECTION, CLASSIFICATION AND REGRESSION**

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**G6 G-A**

**HUMAN INDUCED NEURONS MODEL HALLMARKS OF ALZHEIMER'S DISEASE**

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**G7 UG-A**

**BINOCULAR INNATE VISUAL LEARNING THROUGH SPONTANEOUS ACTIVITY PATTERNS**

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**G8 G-B**

**IDENTIFYING SMALL MOLECULE INHIBITORS OF THE RNA EDITING ENZYME, ADAR2.**

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**G9**

**DEVELOPMENT OF MOUSE DERIVED DRG EXPLANT TO INVESTIGATE HSV-1 PATHOGENESIS**

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**G10**

**INTRACELLULAR UPTAKE OF CARBON NANOTUBES AUGMENTS NEURAL CONDUCTIVITY AND THE EXTENT OF ACTIVATION DURING DIRECT BRAIN STIMULATION THERAPY**

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## *THEME H. SENSORY AND MOTOR SYSTEMS*

**H1**

**ENERGY OUTPUT OF MITOCHONDRIA LOCATED NEAR SYNAPTIC RIBBONS IN INNER EAR HAIR CELLS**

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**H2 G-A**

**UNDERLYING FACTORS CONTRIBUTING TO REACHING FUNCTION IN CHRONIC STROKE: PRELIMINARY RESULTS OF A PREDICTIVE MODEL**

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**H3 G-B**

**ORIGIN OF RECURRENT MOTOR CIRCUITS IN ZEBRAFISH SPINAL CORD**

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**H4 UG-A**

**THERMOSENSORY EFFECTS ON DROSOPHILA CIRCADIAN RHYTHMS**

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**H5 PD-B**
**INACTIVATING Kv CURRENTS IN TYPE II HAIR CELLS OF THE MOUSE UTRICLE**
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**H6**
**GENE EXPRESSION ANALYSIS AS A TOOL TO MEASURE RECOVERY OF THE NASOPHARYNGEAL REFLEX**
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**H7 UG-B**
**MITOCHONDRIA IN VESTIBULAR EFFERENT BOUTONS**
P. Mozaffari[1], A. Kambalyal[1], S. Sobkiv[2], K. Arias[2], F. Padron[2], J. Lesus[4], B. Desai[2], R. Bahari[2], G. Perkins[3], A. Lysakowski[4]
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**H8 G-A**
**A RETINAL GANGLION CELL USES INTRINSIC PROPERTIES TO CONTROL ITS FEATURE SELECTIVITY**
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**H9 G-B**
**FUNCTIONAL DEPENDENCE ON CONTRALESIONAL HEMISPHERE AND ITS RELATIONSHIP WITH CORTICO-RETICULOSPINAL STRUCTURE AND HAND/ARM IMPAIRMENT IN MODERATE TO SEVERE CHRONIC STROKE**
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**H10 G-A**
**GENETICALLY MODIFIED MICE TO STUDY SCHWANN CELL-SPECIFIC PATHWAYS IN VIVO IN A MODEL FOR PAINFUL OBESITY-INDUCED NEUROPATHY**
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# POSTER ABSTRACTS

## *THEME A. COGNITION AND BEHAVIOR*

**A1 UG-A**
**HOW DO ENERGY DRINKS MAKE YOU FEEL? EFFECTS OF ENERGY DRINK COMPONENTS ON ANXIETY BEHAVIOR AND CORTISOL RESPONSES IN ZEBRAFISH (DANIO RERIO)**

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This study investigated the independent and combined effects of caffeine and taurine on anxiety behavior and neuroendocrine responses in the adult zebrafish (*Danio rerio)*. Caffeine (1,3,7-trimethylpurine-2,6-dione), the world's most commonly used psychoactive drug, acts as an adenosine receptor blocker and a mild central nervous system stimulant. However, excessive usage of caffeine is associated with anxiety. Taurine (2-aminoethanesulfonic acid), a semi-essential amino acid synthesized within the human brain, has been hypothesized to play a role in anxiolytic behavior. Caffeine and taurine are two common additives in energy drinks, and are often found in high concentrations in these beverages. This raises the question regarding the actions of these two chemicals on anxiety when consumed together. A suitable vertebrate to examine anxiety-like behavior is the zebrafish, which has shown promise due to the substantial physiological and genetic overlap with humans. Anxiety behavior in zebrafish can be determined by analyzing habituation to novelty when fish are placed into a novel tank. Neuroendocrine responses can be measured by analyzing whole-body cortisol levels. The goal of this study was to determine if exposure to caffeine, taurine, or a combination of the two compounds altered anxiety-like behavior measured by the novel tank test and whole-body cortisol levels in zebrafish. Zebrafish were individually exposed to either caffeine (100 mg/L), taurine (400 mg/ L), or both for fifteen minutes. Zebrafish in the control group were handled in the same manner but were kept only in system tank water. After treatment, fish were transferred to the novel tank test. Behavior was tracked for six minutes after introduction into the novel tank. Fifteen minutes after introduction to the novel tank, fish were euthanized for

the analysis of whole-body cortisol levels. The results suggest that caffeine treatment decreased the amount of swimming and exploration in the top of the tank, which supports the established anxiogenic effect of caffeine. Taurine alone did not alter basal levels of behavioral responses; however, taurine did appear to ameliorate the anxiogenic effects of caffeine on behavior when the two compounds were administered concurrently. The current results of this study suggest that taurine may work to mitigate the anxiety-producing effects of caffeine in energy drinks, although similar studies in humans are needed to confirm.

*This study was supported by the IU Northwest Faculty Grant in-aid of Research and the Faculty for Undergraduate Neuroscience (FUN) Equipment Loan program.*

**A2 UG-B**

**LIKE AN (UNSTRESSED) FISH OUT OF WATER: LACK OF EFFICACY OF A 30-SECOND NET STRESS ON ANXIETY BEHAVIOR AND CORTISOL RESPONSES IN YOUNG ADULT ZEBRAFISH (DANIO RERIO)**

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In recent years, the zebrafish (*Danio rerio*) has become a popular model to study the mechanisms of physiological and behavioral effects of stress, due to the similarity in neural structures and biochemical pathways between zebrafish and mammals. Previous research in this animal model has demonstrated an increase in whole-body cortisol resulting from an acute (30-second) net handling stress, but it remains unclear whether such a stressor will concomitantly increase anxiety-like behavior in zebrafish. In the current study, young adult zebrafish (approximately 90 days post-fertilization) were briefly exposed to a net handling stressor and were subsequently subjected to either the novel tank test or the light/dark preference test. The novel tank test was used to measure exploration and habituation in response to a novel environment, and the light/dark preference test was used to measure locomotor activity and scototaxis behavior. Additionally, we sought to replicate whether this acute (30-second) net stressor was sufficient to increase whole-body levels of cortisol. Contrary to expectations, there was no effect of acute net handling on cortisol levels or anxiety behavior as measured by the novel test tank. Similarly, acute net handling did not significantly induce anxiety-like behavior during the light/dark preference test. Our findings demonstrate that there are possible developmental differences in response to an acute handling stress, as this paradigm is not sufficient to elicit hormonal or behavioral changes in young adult zebrafish. These results suggest the need for a different or more intense acute stressor in order further explore neuroendocrine mechanisms and anxiety-like behavior at this developmental stage in this animal model.

*This study was supported by the IU Northwest Faculty Grant-in-aid of Research, the IU Northwest Minority Opportunity for Research Experience (MORE) program, and the Faculty for Undergraduate Neuroscience (FUN) Equipment Loan program.*

**A3**

**IDENTIFYING SEX DIFFERENCES IN THE BASOLATERAL NUCLEUS TO CENTRAL NUCLEUS CIRCUIT OF THE AMYDALA THROUGH ANTIDROMIC IN VIVO STIMULATION**

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Anxiety disorders are the most common type of psychiatric disorders with the prevalence two times higher among females than males. The amygdala is hyperactive in patients with anxiety disorders and has sex differences in activation in response to emotional provoking stimuli. The basolateral amygdala (BLA) is known to play a role in fear and anxiety and our lab has previously shown that BLA activity in naïve female rats is increased compared to naïve male rats. The BLA has many downstream projections, so we specifically wanted to look at the projection to the central amygdala (CeA), which is necessary for cued fear conditioning and has connections controlling the motor expression associated with fear behaviors, such as freezing. Sex differences have also been observed in behavioral responses to cued fear, with females freezing more than males in response to a conditioned tone. Since the CeA is required for cue conditioned freezing, we hypothesized that there will be a stronger output. in females. In order to test this hypothesis, we used single unit in vivo electrophysiology and stimulated the CeA to antidromically identify BLA neurons that project to the CeA. Understanding the specific outputs of the BLA could lead to novel target therapies.

*This study is funded by grants from the National Institutes of Health (RO1MH084970) and (RO1MH100536)*

**A4 UG-A**

**STRINGING TOGETHER SPECIES: COMPARING KINEMATIC AND TOPOGRAPHIC CHARACTERISTICS OF BIMANUAL STRING-PULLING BEHAVIOR IN HUMANS (HOMO SAPIENS) AND RATS (RATTUS NORVEGICUS)**

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Manipulatory scale space constrains many aspects of fine motor movement and has likely influenced the evolution of neural structures that mediate this behavior. Recent work has demonstrated that spontaneous string-pulling behavior in rodents is a highly organized sequence of movements. The current study used motion capture software to characterize movement organization during string-pulling behaviors in humans (*Homo sapiens*) and rodents (Rattus norvegicus). The first experiment examined topographic and

Kinematic aspects of movement when both species spontaneously engaged in string-puling behavior. Both species were observed to organize their movements into a sequence of reach and withdraw phases that alternated between hands. The second experiment examined the effects of increasing string weight on the organization of string-pulling behavior. Both species exhibited systematic changes in manipulatory scale movement associated with increasing weight in the string-pulling task. This comparative work establishes that humans and rats, with limited training, exhibit similar spontaneous and dynamic range of string-pulling behavior. These characteristics make string-pulling an ideal fine motor task to investigate rodent models of neuropathology and may provide a useful assessment of the efficacy of novel therapeutic interventions.

*This research was funded by Northern Illinois University.*

**A5 UG-B**

**BEHAVIORALLY INHIBITED INDIVIDUALS EXHIBIT MORE FREQUENT AROUSALS DURING SLEEP COMPARED TO NON-BEHAVIORALLY INHIBITED INDIVIDUALS**

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Behavioral inhibition (BI) is a trait characteristic that is identified by extreme social withdrawal and reserved behavior in response to unfamiliar stimuli. Behaviorally inhibited individuals have been shown to be at an increased risk for later onset of anxiety disorders, such as Posttraumatic Stress Disorder (PTSD). Recent studies have evaluated the sleep architectures of PTSD patients and have revealed distinct disruptions throughout their sleep cycle. PTSD patients experience longer sleep latencies, reduced REM sleep periods, and increased arousals from sleep, specifically while in REM sleep. REM sleep deprivation has also been shown to decrease emotional stability, prevent fear consolidation, and further progress anxiety disorders. This serves as a critical issue, due to the already altered expressions of anxiety and startle response in PTSD patients. There are conflicting hypotheses as to whether the alterations in sleep architectures are involved in either the etiology or symptomology of PTSD. Thusly, the goal of this study to determine if similar sleep disturbances are present in individuals who are at a higher risk for onset of PTSD. To determine if BI individuals experience similar sleep architectural disturbances seen in PTSD patients, sleep patterns will be examined through use of an electroencephalogram, electrooculogram, temperature transducer and respiratory effort transducer over a 120-minute sleep period. While no REM-sleep was reached during the sleep period, it was determined that BI individuals show trends for shorter sleep latencies and more frequent arousals from sleep when compared to non-behaviorally inhibited individuals. Future studies will be directed towards increasing sample size, as well as increasing sleep period times in order to obtain REM sleep.

**A6 PD-A**

**CHILDHOOD SOCIOECONOMIC STATUS PREDICTS TONG TERM COGNITIVE OUTCOMES FOLLOWING TRAUMATIC BRAIN INJURY**

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Higher childhood socioeconomic status (SES) is associated with higher intelligence scores as well as better cognitive recovery following pediatric traumatic brain injury (TBI). Less is known about the effects of childhood SES on long-term cognitive outcome following TBI acquired in adulthood. We examined the association between childhood SES and general intelligence throughout adulthood in a sample of 240 Vietnam veterans from diverse socioeconomic backgrounds. 186 of the study participants sustained a penetrating brain injury (pTBI) in their 20s, providing the largest study testing childhood SES effects on recovery of cognitive function following non-pediatric TBI. General intelligence was measured pre-injury (upon enlistment in the military), and then again 15, 35, and 42 years post-injury. For both participants with and without pTBI, childhood SES was a significant predictor of intelligence scores at age 65, however it was not associated with the rate of cognitive change. After controlling for brain lesion volume and education, SES remained a significant predictor of intelligence scores pre-injury and 15 years post injury, but not 35+ years post injury. Lastly, intelligence scores of the Low-SES control group were not statistically different from either the Low or High SES pTBI groups in all post-injury phases. These findings provide the first evidence indicating the persistent effects of childhood SES on cognitive functioning later in adulthood following a TBI.

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**A7 UG-A**

**PARTIALLY REINFORCED SIGNALED LEVER PRESS CONDITIONING REVEALS DIFFERENCES IN THE EXPECTATION VERSUS THE PRESENCE OF SHOCK IN BEHAVIORALLY INHIBITED WISTAR-KYOTO RATS COMPARED TO SPRAGUE DAWLEY RATS**

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People with a behaviorally inhibited temperament (reticence to act within the environment) are 33% more likely to be diagnosed with a stress or anxiety disorder (Gladstone and Parker, 2005). Avoidance preservation, or the maintenance of a learned action to circumvent an aversive stimulus, is a key characteristic of behaviorally inhibited individuals (Jiao et al., 2015). The Wistar-Kyoto (WKY) rat strain has been studied extensively as a model for anxiety disorders, as they show faster

lever-press avoidance and slower extinction rates as compared to the non-behaviorally inhibited Sprague-Dawley (SD) rats (Servatius et al, 2008). In this study, we are examining the differences in response between female WKY rats and female SD rats in open field testing, a lever-press avoidance paradigm and immunohistochemistry analyses. In the lever-press avoidance paradigm, we are specifically examining the differences in avoidance acquisition in both 100% reinforcement of a paired tone-shock trials and 50% partial reinforcement with some tone-only trials between both rat strains. The results of the open field testing showed significantly lower response latency times and number of sites visited in the female WKY rats as compared to the female SD rats, indicating behavioral inhibition. In the lever-press paradigm, it was found that both the 100% and 50% reinforced WKY groups had higher avoidance acquisition than both 100% and 50% reinforced SD groups. The behaviorally inhibited rats were extremely influenced by the tone-shock paradigm and this enhanced ability to learn to evade aversive stimuli could be a major reason why behaviorally inhibited individuals are more likely to acquire anxiety and stress disorders. Lastly, preliminary immunohistochemistry analyses using zif as an immediate early gene marker to detect active brain regions, indicate some differentially activated circuits in avoidance learning between strains. Future steps will quantify this data to conclusively determine the active brain regions involved in this process in both SD and WKY rat strains.

**A8 UG-B**
**IMAGE-BASED CLASSIFICATION OF BODY LANGUAGE POSES TO EMOTIONS USING CONVOLUTIONAL NEURAL NETWORKS (CNN)**

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Recognizing human emotions based on a person's body language is a complex neurological process that humans often take for granted. The visual pathway that propagates from the eye to the occipital lobe, breaking down images from basic features and building upon the previous layer, can be replicated in a computer using artificially intelligent algorithms in the field of computer vision using convolutional neural networks (CNN). The neural network, just like in the brain, models the interaction between neurons, with each neuron being represented by a mathematical function called a perceptron. Real-world images of humans in varying environments displaying emotion through variations in expression, body language, colorings, and features are manually labeled and used to train the CNN. This allows the algorithm to pick out features that occur in pictures of each emotion that will intelligently aid in relating body language, expressions and poses to an emotion, even if this relation is not defined previously. The accuracy of the classification is then analyzed by reviewing what features were extracted from the images and the network is retrained accordingly to output even more accurate results.
*This study was funded by Lake Forest College.*

**A9 UG-A**
**ATTENTION PATTERNS PREDICT MENTAL ARITHMETIC PERFORMANCE**

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Despite the crucial role that attention plays in basic functions and in Attention Deficit/ Hyperactivity Disorder (ADHD), it remains poorly understood. Research has shown that fluctuations in attention can be measured by changes in pupil dilation (i.e., pupillometry), which is an autonomic arousal response. The mechanism underlying the correlation between attention patterns and pupil dilation has been found to result from cognitive regulation of pupil dilation. Greater increases in pupil dilation have been associated with engagement in more difficult tasks, while smaller increases in pupil dilation have been related to inattention during attention tasks. Studies on attention have frequently administered mental arithmetic tasks because solving mental arithmetic engages attention and performance can be clearly measured. This study used pupillometry to investigate if changes in attention patterns can predict performance on mental arithmetic problems. A mental arithmetic task was developed based on tasks used in previous studies and fourth grade education standards to ensure the 12 college students recruited would be able to solve all problems. The time-constrained task appeared on a computer and contained 40 basic addition problems, each involving the addition of three 2-digit numbers. Problems were designed with varying levels of difficulty, in which more difficult problems involved carrying and easier problems did not. The mental arithmetic task was administered while recording pupil dilation at 60 Hz with an ISCAN eye tracker and recording response latency. Between each problem, a baseline pupil dilation was recorded. Patterns of pupil dilation fluctuations from the baseline were compared based on problem difficulty and problem performance.
*This study was funded by Lake Forest College.*

**A10 UG-B**
**HEMISPHERIC PROCESSING OF EMOTION WORDS: THE EFFECTS OF SEX AND VALENCE**

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Previous research suggests that females tend to perform better than males on tasks that require the recognition of an emotion in nonverbal tasks (Hall, 1984; McClure, 2000), however, other research suggests that females tend to perform better than males only for negatively-valenced (e.g., *anger, disgust*) but not positively-valenced (e.g., *love, happiness)* emotions (Thompson & Voyer, 2014). In addition, research has shown that males and females have different degrees of hemispheric lateralization when processing visual (i.e., picture) stimuli (Wager et al., 2003). Currently, it is unclear how these hemispheric differences between males and females shown for emotion processing in nonverbal tasks extend to language-based tasks. In the present study,

undergraduate students (58 = female, 17 = male) performed a lexical decision task for positively-valenced and negatively-valenced words presented to either the left visual field-right hemisphere or the right visual field-left hemisphere in a divided visual-field paradigm. Both males and females showed a left hemisphere advantage during this word recognition task, regardless of valence. However, males showed faster response times for positive words relative to negative words. In addition, males showed faster response times than females for positive words. Females were also more accurate when responding to positive words compared to negative words. These results suggest that males might have an advantage when processing positively-valenced words, and that the female advantage previously shown for processing emotional information may not extend to language-based tasks. The results also suggest that sex differences during word recognition for emotion words depends on the valence of the target word.

**A11 UG-A**
**SAVINGS MEMORY IS ACCOMPANIED BY TRANSCRIPTIONAL CHANGES THAT PERSIST BEYOND THE DECAY OF RECALL**

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Most long-term memories are forgotten, becoming progressively less likely to be recalled. Still, some memory fragments may persist beyond forgetting, as savings memory (easier relearning) can persist long after recall has become impossible. What happens to a memory trace during forgetting that makes it inaccessible for recall and yet still effective to spark easier re-learning? We are addressing this question by tracking the transcriptional changes that accompany learning and then forgetting of a long-term sensitization memory in the tail-elicited siphon withdrawal reflex of *Aplysia californica*. First, we tracked savings memory. We found that even though recall of sensitization fades completely within 1 week of training, savings memory is still robustly expressed at 1 week and 2 weeks post training. Next, we used microarray to identify transcriptional changes that persist beyond the decay of recall; we identified 11 transcripts strongly regulated 1 week after training that validated with qPCR in an independent set of samples. Finally, we tracked the time-course of regulation of these 11 'savings-related' transcripts at 1 hour, 1 day, 5 days, and 2 weeks after the induction of sensitization. Some are regulated rapidly after induction and then persist for up to 1 week; others show a delayed but persistent regulation. Remarkably, 2 transcripts still show strong regulation of expression 2 weeks after training. Our results provide the first evidence of transcriptional fragments of a learning experience that persist far beyond the decay of recall.

**A12**
**FREQUENT BINGE DRINKERS EXHIBIT BLUNTED REWARDING EFFECTS OF METHAMPHETAMINE**

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Epidemiological evidence shows high co-abuse of alcohol and stimulant drugs i.e., amphetamines, cocaine. In addition, preclinical and clinical studies show that a history of alcohol use influences behavioral responses to stimulant drugs. Together the evidence suggests specific behavioral or neuropharmacological mechanisms may underlie co-use of alcohol and psychostimulants. In this study, we compared subjective responses to methamphetamine (MA) between frequent binge drinkers (N=26, > 5 binges per month) and non-binge drinkers (N=21, no binges). Healthy men and women completed separate experimental sessions with double-blind administration of 0 and 20mg MA (in randomized order). They reported subjective mood and drug effects before and at repeated times after drug administration. The groups differed in age (frequent binge drinkers were younger) but did not differ on current or previous history of drugs other than alcohol. In addition, the groups did not differ on personality traits or behavioral impulsivity measures. In comparison to non-binge drinkers, frequent binge drinkers reported significantly lower positive rewarding effects of MA (Stimulation $p<0.05$, Elation $p<0.05$). Our findings suggest that frequent binge drinking may cause tolerance to the stimulant effects of MA.
*This research was supported by a grant from the National Institute on Drug Abuse (DA033488).*

**A13 UG-A**
**STEP FATHERING BEHAVIOR OF NON-REPRODUCTIVE AMPHIPRION OCELLARIS AFTER REMOVAL OF THE BREEDING PAIR FROM THE GROUP**

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*Amphiprion ocellaris* anemonefish are a useful model species for studying the evolution of complex social behavior because of their unique life history. *A. ocellaris* live in small social groups consisting of one breeding pair and a varying number of non-related, non-reproductive subordinates. Being unrelated to either member of the breeding pair, evolutionary theory predicts that non-reproductive individuals should eat any eggs left unattended by the breeding pair to maximize their fitness. Observations have shown instead that when the male is removed from the area, non-reproductive individuals will begin to exhibit parental care behaviors towards the eggs. It has been suggested that this behavior represents an evolutionary adaptation because it increases the odds of the female spawning with the non-reproductive subordinate next. The purpose of this study was to quantify these behaviors and determine if non-reproductive individuals

would continue to provide parental care in the absence of both members of the breeding pair. Groups consisting of 3 individuals were videotaped for 4 days after eggs were first seen in the tank and manipulations were applied (removal of male, removal of male and female, or no removals). Parental behaviors were counted for each video and compared across tanks and conditions. Pictures of eggs in the nest were also captured and counted before individuals were removed from the nest and after they were returned. When the male was removed, both the non-reproductive individual and female tended to show increased parental care behavior. When both the male and the female were removed from the tank, the non-reproductive individual tended to show higher levels of parental care behavior compared to both the control and only male removal conditions. Higher rates of egg loss were also observed when individuals were removed from the tank. Preliminary results suggest that though non-reproductive individuals do eat a higher proportion of eggs than males, they will still somewhat care for unrelated eggs when left unattended. Step fathering represents a substantial effort on the part of the non-reproductive subordinate, and therefore caring for unrelated eggs seems to be maladaptive for individual fitness. Further research is needed to develop a coherent evolutionary hypothesis for the origin of this type of seemingly maladaptive step fathering behavior.

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**A14**

**DIVERGING ROLES OF SOMATOSENSORY CORTICAL SUB-REGIONS IN A WHISKER-SIGNALED ASSOCIATIVE LEARNING TASK**

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Learning and memory deficits are a prominent and severely debilitating symptom of neurodegenerative disorders and even normal aging. The somatosensory cortex, which is responsible for encoding and processing tactile stimuli, is commonly used to investigate mechanisms of learning and memory. Research conducted in rabbits and rodents has demonstrated that a sub-region of the somatosensory cortex, the primary somatosensory cortex (SI), is necessary for acquisition, but not retention of memory demanding tasks that involve whisker stimulation. What remains unknown is where consolidated memories are stored in the neocortex. **Therefore, the objective of this study is to determine if another sub-region of the somatosensory cortex, the secondary somatosensory cortex (SII), is an essential site for whisker-evoked memories.** Using trace eyeblink conditioning (tEBC), mice are trained to associate vibration of the whiskers (a neutral conditioning stimulus (CS)) with an airpuff directed at the eye (an aversive unconditioned stimulus (US)) to elicit a blink. After repeated pairings of the CS and US, the CS alone elicits a blink or conditioned response (CR) in mice that learned to associate the two stimuli. The consolidated memory is robust and can be demonstrated after a one-month training hiatus. Reversible inactivation of virally infected neurons that express Designer Receptors Exclusively Activated by Designer Drugs (DREADDs) was used in an attempt to inhibit activity of SI or SII during acquisition experiments to 1) determine the necessity of the primary somatosensory cortex and 2) determine the necessity of the secondary somatosensory cortex in tEBC acquisition in mice. Multi-unit neuronal recordings were used to verify inactivation of the targeted regions. Immunohistochemistry was used to check location and spread of inactivated regions. Preliminary experiments have demonstrated that inhibition of SI during tEBC acquisition experiments impairs learning, and interestingly, inhibition of SII during tEBC acquisition does not impact learning. Ultimately, understanding *where* and subsequently how memories are stored will put us in a much better position to precisely target therapeutic measures to combat the symptoms and underlying causes of learning and memory disorders.

*This study is funded by a research award from the NIH Postbaccalaureate Research Education Program (R25 GM121231).*

**A15 G-A**

**HEMISPHERIC PROCESSING OF STRONGLY-CONSTRAINED AND WEAKLY-CONSTRAINED PREDICTIVE INFERENCES: BEHAVIORAL AND ELECTROPHYSIOLOGICAL EFFECTS**

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Previous research has shown hemispheric asymmetry for the processing of strongly-constrained (SC) and weakly-constrained inferences (WC) during reading (Virtue, van den Broek & Linderholm, 2006). Specifically, the right hemisphere (RH) shows high levels of inference-related facilitation in lexical decision tasks for both SC and WC predictive inferences, whereas the left hemisphere (LH) shows high levels of inference-related facilitation only for SC predictive inferences. These results are consistent with the fine-coarse semantic coding theory (Jung-Beeman, 2005), which proposes that the LH activates closely related semantic associates (e.g., scissors) of words (e.g., cut), whereas the RH activates distantly related semantic associates (e.g., foot, cry, glass) of words. In addition, physiological research shows differences in event-related potentials (ERPs) depending on the type of inference readers generate during reading. Specifically, N400 amplitudes vary depending on if readers generate a bridging or a predictive inference (St. George, Mannes & Hoffman, 1997). Currently, it is not clear how these N400 amplitudes differ in the cerebral hemispheres depending on the level of textual constraint. In the current study, participants read a brief passage and then performed a divided visual-field lexical decision task for inference-related target words presented either to the LH or the RH. In Experiment 1, targets were strongly-constrained toward the generation of a specific inference. In Experiment 2, targets were weakly-constrained. **Behavioral** results showed faster response times for SC targets and WC targets than targets following neutral texts (i.e., no inference condition). **ERP** results revealed larger N400 potentials for neutral texts relative to SC texts. In the strongly-constrained condition,

N400 potentials were larger in the RH relative to the LH. Lastly, larger P600 potentials were found in both the SC and WC conditions compared to the neutral condition. These results suggest that N400 potentials are significantly attenuated for SC texts, but not for WC texts. Differences in N400 potentials between the hemispheres may suggest that the RH shows greater interference for processing SC inferences compared to the LH. In addition, the degree of textual constraint seems to influence N400 effects, but not P600 effects. Overall, this study provides evidence of behavioral and physiological differences for the processing of SC and WC predictive inferences. This study is funded by the National Science Foundation (grant #1338112).

**A16 PD-B**

**AGE-DEPENDENT DISRUPTION OF PREFRONTAL LOCAL FIELD POTENTIAL RESPONSES BY REPEATED CANNABINOID EXPOSURE DURING ADOLESCENCE IN FEMALE RATS**

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Converging epidemiological findings suggest that frequent exposure to cannabis during adolescence can result in lasting prefrontal cortex deficits and related cognitive impairments later in life. However, the precise neurobiological mechanism(s) underlying the adolescent susceptibility to cannabis exposure remains unclear. We have recently shown in male rats that adolescent (postnatal day – P35-45) exposure to the cannabinoid CB1 receptor agonist WIN produces an enduring, frequency-dependent disruption of prefrontal local field potential responses. Interestingly, such prefrontal disruption was not seen when WIN was given during adulthood (P75-80). Here we examined whether a comparable long-lasting, age-dependent PFC dysregulation occurs in female rats. Similar to what is observed in the male PFC, ventral hippocampal inputs exert a powerful, frequency-dependent inhibitory control of prefrontal local field potential responses through a late adolescent recruitment of local GABAergic transmission. However, this inhibitory modulation of PFC activity fails to emerge following adolescent (P35-40) WIN exposure whereas adult (P75-80)-treated rats show normal patterns of local field potential suppression. Importantly, this age-dependent dysregulation was normalized by acute, local PFC administration of the $GABA_{Aα1}$ positive allosteric modulator Indiplon, suggesting that the diminished inhibition of local field potentials resulting from adolescent cannabinoid exposure is GABA-mediated. Taken together, these data indicate that the PFC of males and females exhibit similar developmental trajectories and sensitivity to cannabinoids during adolescence. As in males, adolescence represents a critical window for the gain of GABAergic function in the female PFC that is highly susceptible to the disrupting effects of cannabis and cannabinoids (CB1R agonists).

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**A17 UG-B**

**EFFECTS OF ACUTE AEROBIC EXERCISE ON BEHAVIORAL AND PREFRONTAL HEMODYNAMIC RESPONSES TO A FATIGUING COGNITIVE TASK**

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**Objectives**: Despite having expended a significant amount of energy, people report lower levels of fatigue after exercise. The purpose of this study was to explore the mechanisms that underlie this apparent paradox by measuring the effects of aerobic exercise on feelings of fatigue and prefrontal cortex (PFC) hemodynamic response during a fatiguing cognitive task. We hypothesized that exercise would reduce feelings of fatigue and improve task performance, changes that would be partially explained by PFC hemodynamic response to the fatiguing task.

**Methods**: Healthy college students (16 males; 15 females) completed three lab visits: a familiarization session with a graded exercise test, a rest session, and an exercise session. The modified Paced Auditory Serial Addition Task (mPASAT) was administered before and after the exercise and rest conditions, and fNIRS was recorded simultaneously. Self-reports of fatigue, block-averaged reaction time weighted for accuracy (WRT) on the mPASAT, and block-averaged hemodynamic response (HbO, HbT, and HHb) were analyzed using a repeated measures analysis of variance (RM-ANOVA) across Condition (Exercise, Rest), Time (Pre-, Post-Condition), and Block (1, 2, 3), with Bonferroni corrections for multiple comparisons. Huyhn-Feldt was used to adjust F-values for differences in variance across the levels of Block. Follow-up simple contrasts were computed to decompose Block effects.

**Results**: Completing the mPASAT was fatiguing across Blocks independent of Condition and Time [$F(3,90)=55.774$, $p< 0.001$, $\eta^2=0.675$]. Fatigue increased across all blocks relative to pre-task baseline (all ps < 0.001). Weighted reaction time on the mPASAT decreased across Blocks independent of Condition and Time [$F(2,58)=16.424$, $p< 0.001$, $\eta^2=0.362$]. Pairwise comparisons revealed significant differences between blocks 1 and 2 ($p=0.003$, and blocks 1 and 3 ($p<0.001$), but not between blocks 2 and 3 ($p = 0.113$). PFC hemodynamic responses to the mPASAT did not change as a function of Block or Time. There were significant effects of Condition for HHb and HbT [$F(1,20)=7.792$, $p=0.016$, $\eta^2=0.256$ and $F(1,20)=4.760$, $p = 0.041$, $\eta^2=0.192$], such that the hemodynamic response was positive for the Exercise condition and negative for the Rest condition.

**Conclusion**: These findings suggest that the mPASAT can be used to manipulate fatigue in a healthy sample of young adults in a laboratory setting. Contrary to expectations, exercise did not protect against the fatiguing effects of the task. The uncoupling of feelings of fatigue, performance, and PFC activity on a fatiguing task after exercise in this sample necessitate further exploration.

*This study was partially funded by a grant from the Wheaton College G.W. Aldeen Memorial Fund*

## *THEME B. DEVELOPMENT*

**B1 G-B**
**OTOLITH DYSFUNCTION DISRUPTS EXPLORATORY MOVEMENT ORGANIZATION ACROSS TWO DEVELOPMENTAL TIME POINTS**

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Animals use multiple sources of information to maintain spatial orientation. Previous work has shown that mouse exploratory behavior organization depends on self-movement cues derived from the vestibular system. The current study examines developmental changes in the role of the otolith organs in an otoconia deficient mouse model (*tilted* mice) for organizing exploration at postnatal days 35 and 84. Under dark conditions, control (n=12) and *tilted* (n=9) mice explored a borderless circular tabletop. A novel sequential analysis was used to segment exploratory movements into progressions and stops. Although behavior was similar both developmental time points, *tilted* mice traveled significantly shorter distances, spent more time stopped, and had larger changes in heading between progressions relative to control mice. These observations suggest that otolith signals influence movement organization early in life, and thus have important implications for development in altered gravitational fields. Further analysis is underway to characterize stop clustering behavior as a measure of home base establishment. This study provides further evidence for the role of the vestibular system in maintaining spatial orientation.

**B2 G-A**
**DEVELOPMENTAL REGULATION OF PREFRONTAL PLASTICITY BY ENDOCANNABINOID SIGNALING IN VIVO**

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Prefrontal cortex (PFC) maturation during adolescence is characterized by structural and functional changes, which involve the remodeling of GABAergic and glutamatergic transmission, as well as changes in endocannabinoid mediated signaling. Despite the modifications that occur within each of these systems, the manner in which the endocannabinoid system interacts with glutamate and GABA transmission in the PFC during the adolescent transition remains unknown. To address this, we conducted local field potential recordings *in vivo* and examined how manipulations of the endocannabinoid-CB1R system affects PFC responses to basolateral amygdala (BLA) and ventral hippocampal (vHipp) stimulation. Pharmacological activation and inactivation of CB1Rs revealed that the recruitment of endocannabinoid-CB1R signaling does not become functionally online until adulthood. Once present, CB1R signaling exerts inhibitory control over both LTP and LTD from afferents originating from the BLA and vHipp. Importantly, both endocannabinoids 2-AG and anandamide are recruited by the vHipp-PFC pathway. However, only 2-AG mediates the inhibitory effect of CB1R signaling of the BLA to PFC transmission. Together, these results show that the endocannabinoid signaling in the PFC is developmentally regulated and emerges to control the gain of afferent drive in an input-specific manner. Funding support: NIH grants R01MH086507 and R01MH105488 to KY Tseng

## *THEME C. DISORDERS OF THE NERVOUS SYSTEM*

**C1 G-B**
**DISCOVERING A NOVEL MECHANISM UNDERLYING SPORADIC ALZHEIMER'S DISEASE.**

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Type 2 Diabetes (T2D) is a risk factor for the development of Alzheimer's disease (AD). According to data collected from the Mayo Clinic Alzheimer's Disease Registry, more than 80% of AD cases also present with either T2D or an impaired glucose metabolism disorder. The aim of the current study is to investigate the mechanism underlying T2D-induced AD in adult diabetic mice. To thoroughly address this, we utilized two models of T2D (1) Obesity-independent *MKR* transgenic mice expressing a mutated form of the human insulin-like growth factor 1 receptor (IGF-1R) (2) Obesity-dependent db/db mice harboring a mutation in the leptin receptor. We observed that treatment of brain endothelial cells with pro-inflammatory cytokines results in dramatic reduction in caveolin-1 (Cav-1) expression. Cav-1 expression is progressively depleted in the brains of diabetic mice, and proinflammatory cytokines are upregulated. Depletion of Cav-1 was accompanied by deficits in recognition memory and in hippocampal neurogenesis in 4-5-month-old diabetic mice. To start to address the mechanism underlying impaired hippocampal plasticity in the diabetic mice we examined the effect of Cav-1 on neurogenesis. For this purpose, we examined neurogenesis in Cav-1 knockout mice. We observed that the number of neural stem cells is significantly reduced, and the expression and membrane localization of critical neurogenic receptors, such as epidermal growth factor and basic fibroblast growth factor is altered. Importantly, we observed a 4-fold increase in BMPR1a, a critical negative regulator of neurogenesis. Interestingly, we show that depletion of Cav-1 induces amyloidogenic processing of amyloid precursor protein (APP), leading to upregulation of beta-amyloid (Aβ) production and hyper phosphorylation of tau. Taken together, these results suggest that progressive loss of Cav-1 expression in the brains of diabetic mice compromises learning and memory and hippocampal plasticity and induces AD pathology.

**C2 PD-B**
**LEVERAGING LRRK2 BIOLOGY TO DEVELOP NOVEL THERAPEUTIC OPPORTUNITIES FOR PARKINSON'S DISEASE**

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Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer's disease. More than 1 million people in the United States and about 10 million people worldwide live with PD. Although there is currently no cure, it is possible to treat this complex disease by expanding our knowledge of the molecular pathophysiology of PD. The identification of genes that are related to PD has offered the molecular tools necessary in furthering this understanding. Mutations in a particular gene called *LRRK2* have provided a great amount of excitement in the recent PD research not only because of its high prevalence in the inherited forms of the disease, but because it confers overall risk for the development of PD in the general population. Moreover, patients with inherited *LRRK2* mutations exhibit clinical and pathological phenotypes indistinguishable from sporadic PD. As such, the knowledge of how *LRRK2* mutations impact cellular processes can provide mechanistic insights into understanding the pathophysiological basis of idiopathic PD, and facilitate the development of therapeutic strategies. Several LRRK2 mutations have been demonstrated to augment LRRK2 kinase activity. This prompted the development of ATP competitive inhibitors that specifically target such increased kinase activity. However, this therapeutic approach comes with several challenges at the preclinical disease models. This highlights the importance of alternative approaches for effective therapeutic innervations that will take into consideration many aspects of LRRK2 biology. In other words, in addition to chemical modulators of LRRK2 enzymatic activity, compounds that modulate LRRK2 cellular functions should be actively pursued. Cell-based high throughput phenotypic screening is an excellent method to identify compounds that modulate mutant LRRK2 function in an unbiased manner. Our previous studies were among the first to demonstrate that one of the most consistent cellular phenotypes that reflect mutant LRRK2 activity is a decrease in neurite outgrowth. As such, we hypothesize that quantitative high content imaging can be used to screen molecule libraries and identify compounds that restore neurite outgrowth of primary dopaminergic neurons by modulating mutant LRRK2 function. By using libraries of molecules with known targets and a history of use in humans (e.g., FDA approved drugs and clinical agents), we aim to identify new targets involved in disease processes and discover existing drugs that may be repurposed for the treatment of PD. In order to achieve this: we validated a quantitative high throughput neurite outgrowth assay, and we are currently performing a high throughput compound library screen with 3,000 chemicals composed of FDA-approved drugs and experimental agents. Overall by 1) leveraging the **high relevance of LRRK2 to PD**, 2) using **unbiased high throughput screening approaches** targeting LRRK2 cellular function, 3) utilizing precise neuronal populations with the **highest disease relevance to PD,** and 4) screening **FDA approved drugs** we believe we have a valuable platform to identify molecules targeting LRRK2 within the disease process. Overall, this approach could be the basis for a mechanistic-based therapeutic approach for a disease that is currently addressed symptomatically

**C3**
**TRANSCRIPTIONAL CHANGES IN FOREBRAIN AND CEREBELLUM OF A CLN2 NEURONAL CEROID LIPOFUSCINOSES MOUSE-MODEL**

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The neuronal ceroid lipofuscinoses (NCLs) or Batten disease are a group of pediatric lysosomal storage disorders that can be due to mutations in over a dozen genes, generating similar neurodegenerative pathology with different times of onset. In humans, homozygous mutant variants in the CLN2 gene are characterized by the loss of tripeptidyl peptidase 1 (TPP1) enzymatic activity encoded by the CLN2 gene, leading to a late infantile NCL disease form. In this study, we are using a mouse model developed by the Lobel laboratory (J. Neurosci. 2004, 24(41)9117), which recapitulates the pathology and clinical features of the human disease. In the CLN2 mouse model, tremors start to be apparent at 2 months (mo.) of age, and more severe symptoms, e.g. ataxia, develop after 4 mo. in homozygous animals. We initially analyzed expression changes in the CLN2 forebrain and cerebellum at 4 mo. compared to control by mRNA *in situ* hybridization, quantitative expression (qPCR), and RNA-seq technology. Transcriptional changes were found in 510 and 1550 genes transcripts in forebrain and cerebellum, respectively, from CLN2 defective tissues compared to age match controls. Several of these expression changes were confirmed by independent quantitative and histological analysis. Analysis of the differentially expressed genes using the Ingenuity pathway software, revealed increased neuroinflammation activity in microglia and astrocyte that leads to neuronal dysfunction particularly in the cerebellum; upregulation in production of nitric oxide and reactive oxygen species; activation of leukocyte extravasation signals and complement pathway; and downregulation of major transcription factors involved in control of circadian rhythm. The identification of differentially expressed novel genes has revealed new lines of investigation in this complex disorder and may lead to identification of novel therapeutic targets.

**C4**

**CHARACTERIZING CEREBRAL ENDOTHELIAL EXTRACELLULAR MATRIX PROTEINS IN THE PRESENCE OF HTRA1 DEFICIENT ASTROCYTES**

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Interactions between endothelial cells and astrocytes are vital for cerebral blood vessel formation and blood brain barrier integrity. These interactions form an extracellular matrix (ECM) that envelopes vascular tubes and helps to stabilize tight junctions between endothelial cells. Consequently, diseases that disrupt this system lead to deleterious health effects in humans. Many cerebral vessel diseases (CVDs) are characterized by a degradation of the blood brain barrier, often coinciding with alterations of the ECM. Cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL) is a rare genetic CVD caused by mutations in the gene HTRA1, a member of the trypsin family of proteases. Although it is believed that the pathology of CARASIL is endothelial in origin, we find that HTRA1 is preferentially expressed in astrocytes and not detected in endothelial cells. We therefore sought to determine how HTRA1 deficiency in astrocytes affects endothelial cells and ECM expression. We conducted immunohistochemical staining on coronal brain sections to examine both ECM protein expression and vascular branching pattern in HTRA1 null mice, and found that perivascular collagen IV protein expression shows an age dependent regulation by HTRA1. We currently are using endothelial/astrocyte Transwell co-cultures to examine how endothelial cell morphology and endothelial produced ECM proteins respond to the deletion of astrocytic HTRA1. Findings from these studies both *in vivo* and *in vitro* will be presented.

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**C5 PD-A**

**DIFFERENTIAL GENOME-WIDE METHYLATION IN ALCOHOL USE DISORDER SUBJECTS: FOCUS ON THE CORTICO-LIMBIC GLUCOCORTICOID RECEPTORS (NR3C1)**

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Glucocorticoids, crucial mediators of the stress response, may be involved in both the development of alcohol abuse and the consequences of long-term alcohol drinking. Glucocorticoids exert their action through specific receptors, i.e. the glucocorticoid receptors, present in the stress/reward-responsive brain regions that are critical in modulating individual reactivity to stress. Glucocorticoid receptors sensitivity is regulated by FK506 Binding Protein 5 (FKBP5), which acts as a negative regulatory element for the receptor's sensitivity. The human glucocorticoid receptors gene (NR3C1) is composed of seven alternatively utilized noncoding first exons/promoters, some of which are within a CpG island known to be susceptible to epigenetic regulation via DNA methylation. In the present work, we investigated whole genome DNA methylation patterns, focusing on the regulation of NR3C1 expression in the prefrontal cortex (PFC) of alcohol use disorders (AUD) subjects. Post-mortem brain samples were obtained from 25 controls and 25 AUD subjects from the New South Wales Tissue Resource Centre (Sidney, Australia). Genome-wide DNA methylation profiling (Infinium® MethylationEPICBeadChip) was assessed in the PFC of AUD. Statistical analyses were carried out using the R package for Differential DNA methylation. Differential DNA methylation (including 5-methyl- and 5-hydroxymethyl-cytosine) was observed at multiple loci in the PFC of AUD subjects, including genes relevant to alcohol and stress-related disorders. Of note, NR3C1 was one of the central genes differentially methylated in the PFC of AUD subjects. This change was associated with reduced NR3C1 mRNA expression. In addition, FKBP5 Mrna expression was increased in AUD subjects. Likewise, in the hippocampus and amygdala of AUD subjects, NR3C1 and FKBP5 mRNAs expression were altered. We also investigated the mRNA levels of several other genes (NR3C2, CRH, CRHR1/2, POMC, OXTR) that are involved in the individual response to stress and found they are all altered in AUD subjects. The allostatic load induced by excessive and repetitive alcohol consumption results in a maladaptive epigenetic NR3C1 regulation. Our study suggests that alcohol-dependent epigenetic regulation of the NR3C1 expression and other stress-related genes in specific stress/reward-responsive brain regions, such as the PFC and the hippocampus, might be involved in the pathogenesis of AUD.

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**C6 G-A**

**HYPERBARIC PRESSURE-INDUCED REACTIVE ASTROCYTOSIS RESULTS IN ELEVATED LEVELS OF OXIDATIVE STRESS IN PRIMARY OPTIC NERVE HEAD ASTROCYTES**

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Primary open angle glaucoma (POAG) is associated with elevated intraocular pressure (IOP), manifesting in a pathological triad of optic nerve head remodeling, damage to the optic nerve, and retinal ganglion cell (RGC) loss. In POAG, optic nerve head astrocytes (ONHAs), the primary cell type in the optic nerve head, undergo reactive astrocytosis, characterized by increased motility and proliferation, morphological changes, and alterations in gene expression. Herein, we used hyperbaric pressure to induce reactive astrocytosis in order to study the cellular and molecular consequences on cultured ONHAs. Primary adult rat ONHAs were exposed to ambient or hyperbaric pressure (25-30 mm Hg above ambient pressure) for 2 – 30 hr using a cell culture pressure

chamber. Cell viability and proliferation were quantified using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) and lactate dehydrogenase (LDH) release assays, while levels of oxidative stress and nitric oxide (NO) were quantified using fluorescent sensors. ONHA cultures exposed to hyperbaric pressure for up to 30 hr did not exhibit altered cell viability, and LDH release was similar between cells exposed to ambient or hyperbaric pressure ($P = 0.651$). However, hyperbaric pressure significantly increased the sensitivity to a subsequent oxidative challenge in the MTT assay ($P < 0.01$) and the LDH assay ($P < 0.01$). Subsequent analysis of ROS levels revealed that elevated hyperbaric pressure caused a statistically significant increase in the level of ROS as early as 2 hr after exposure to hyperbaric pressure, as quantified by CellROX® fluorescent staining and dichlorofluorescein fluorescence. Furthermore, hyperbaric pressure resulted in an up-regulation of nitric oxide synthase 2 (NOS2) with a concomitant increase in NO levels, as assessed by 4-amino-5-methylamino-2',7'-difluoro-fluorescein diacetate (DAF-FM). Our data provide evidence for increased levels of ROS and NO in ONHAs in vitro following exposure to hyperbaric pressure. It is thus conceivable that even modest exposure to elevated IOP in POAG may significantly alter the oxidation response of ONHAs and accelerate neurotoxic signaling early during glaucoma pathogenesis.

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**C7**

**AMPA RECEPTOR TRAFFICKING IN THE LIMBIC SYSTEMS OF RATS TREATED WITH PRAMIPEXOLE**

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The D3 receptor (D3R)-preferring agonist pramipexole (PPX) is an effective therapeutic for treating the motor symptoms of Parkinson's disease and restless legs syndrome. Also associated with PPX therapy are behavioral addictions, e.g., problem gambling. Accordingly, we demonstrated that PPX increases gambling-like behavior in rats (Rokosik and Napier. *Neuropsychopharmacology*, 2012). Limbic brains regions such as the medial prefrontal cortex (mPFC), nucleus accumbens (NAc) and ventral pallidum (VP) regulate key aspects of addictions and express D3 receptors. What remains unclear are the molecular consequences that underlie PPX-induced behavioral addictions. One mechanism may involve strengthening of glutamatergic synapses *via* insertion of AMPA receptors in neuronal membranes. Here, we tested the hypothesis that AMPA receptor surface expression is increased in limbic brain regions of rats treated with PPX. In the first study, rats were treated with an acute injection of saline or PPX (2mg/kg, ip), and tissues were harvested 1h post-treatment. In the second study, rats were implanted with subcutaneous osmotic minipumps that infused 1.2mg/kg PPX per day for 14 days; tissues were harvested on day 14 of PPX treatment. Modified Western blot protocols determined the surface/intracellular ratio of GluA1 (a subunit of the AMPA receptor) and total levels of GluA1. Acute PPX administration increased the surface/intracellular ratio of GluA1 in the NAc ($p=0.0191$) but did not alter total GluA1, suggesting an increase of AMPAR insertion to the neuronal membrane. This effect was mitigated by pretreatment with the D3 receptor antagonist PG10307. In contrast, acute PPX had no effect on GluA1 distribution or expression in the VP or mPFC. Chronic PPX administration had no significant effect on GluA1 distribution or expression in any of the brain regions tested. Thus, while acute PPX promoted AMPA receptor trafficking in the NAc, AMPA receptor surface expression was normalized with chronic PPX. Changes in excitatory synapses can also be regulated by NMDA receptors, which may contribute to PPX-induced behavioral addictions. We are currently investigating this possibility.

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**C8 G-B**

**DEVELOPMENTAL SYNAPSE MATURATION IS IMPAIRED IN INFANTILE NEURONAL CEROID LIPOFUSCINOSIS**

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Neural circuits are sculpted by the interplay between synapse formation, maintenance, and refinement. Protein palmitoylation is the reversible attachment of palmitic acid to proteins. In neurons, this posttranslational mechanism is critical for axon pathfinding, synaptic transmission, and plasticity. Depalmitoylation, the process of palmitic acid removal, is required for lysosomal proteolysis. Mutation of the depalmitoylating enzyme, palmitoyl-protein thioesterase 1 (PPT1), causes infantile neuronal ceroid lipofuscinosis (CLN1), a pediatric neurodegenerative disease characterized by visual deterioration and seizure, leading to death by five years. However, the role of protein depalmitoylation in neural circuit formation is unknown. Further, it is unclear how loss of PPT1 function leads to synaptic dysregulation and neurodegeneration in CLN1. Therefore, we examined the *Ppt1*$^{-/-}$ mouse to decipher the role of protein depalmitoylation in visual cortex (VC) maturation. First, in primary cortical cultures, *Ppt1*$^{-/-}$ neurons exhibited morphologically immature dendritic protrusions, including filipodia and elongated, thin spines. Calcium imaging experiments revealed extrasynaptic $Ca^{2+}$ transients in dendritic shafts of *Ppt1*$^{-/-}$ neurons, while wild-type (WT) cells had compartmentalized $Ca^{2+}$ influx within spines. Next, we conducted biochemical analyses of synaptosomes prepared from VCs of developing (postnatal day 11-60), WT and *Ppt1*$^{-/-}$ mice. *Ppt1*$^{-/-}$ VCs had selective decreases in mature components of the N-methyl-D-aspartate receptor (NMDAR) protein complex, the GluN2A subunit and its scaffolding protein, PSD-95, which increase during

development. In contrast, neonatal components of the NMDAR complex, GluN2B and SAP102, were unchanged. Recording of NMDAR-mediated excitatory postsynaptic currents (EPSCs) in layer II/III VC neurons confirmed a reduction in both the amplitude and decay time of the fast, GluN2A-mediated component of the EPSC in *Ppt1*$^{-/-}$ VC. Lastly, *Ppt1*$^{-/-}$ neurons demonstrate increased vulnerability to NMDA-induced excitotoxicity, which is mediated by extrasynaptic, GluN2B-rich NMDARs. Together, these findings suggest a critical role for PPT1 in NMDAR regulation and synapse maturation during development.
*This work is funded by startup funding awarded to Akira Yoshii, MD, PhD.*

**C9 PD-B**

**ACTIVATION OF PPAR-ALPHA BY THE ENDOCANNABINOID N-PALMITOYLETHANOLAMINE (PEA) IMPROVES PTSD-LIKE BEHAVIORAL PHENOTYPE BY NORMALIZING CORTICOLIMBIC ALLOPREGNANOLONE LEVELS**

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Post-traumatic stress disorder (PTSD) is a debilitating condition that affects 8-13% of the general population and 1 in 5 veterans. Nowadays, there is no valid medication in the treatment of PTSD symptoms. SSRIs represent the current drugs of choice; however, these antidepressants fail to work in more than 50% of PTSD patients, suggesting that new pharmacological approaches are even more necessary. One of the biochemical alterations observed in PTSD is the down-regulation of the neurosteroid allopregnanolone (Allo). Our group has previously demonstrated that fluoxetine (FLX) at non serotoninergic doses normalizes corticolimbic levels of Allo and improves fear responses in socially isolated (SI) mice, an animal model of PTSD. The endocannabinoid and neurosteroid systems play a key-role in the regulation of emotions and stress responses. For instance, dysregulation of the endocannabinoid system enhances fear acquisition and impairs fear extinction. As well known, activation of peroxisome proliferator-activated receptor (PPAR)-alpha by the endocannabinoid, N-palmitoylethanolamine (PEA) modulates pathophysiological functions (e.g., neuroinflammation, oxidative stress). PEA induces antidepressant effects comparable to those elicited by FLX; in addition, PEA, similarly to FLX, up-regulates Allo levels in cell cultures and brain stem. All together, these findings suggest that PEA may also induce corticolimbic Allo concentrations and improve anxiety and fear responses in SI mice. We observed that PEA induced a normalization of Allo levels in the hippocampus, amygdala and olfactory bulb of SI mice at the doses of 5, 10 and 20 mg/kg and in the frontal cortex levels at the dose of 20 mg/kg. The same treatment did not change Allo levels in the striatum. PEA, by a reconsolidation blockade, facilitated fear extinction and prevented the spontaneous recovery of fear memory in SI mice. Moreover, PEA induced a marked anxiolytic and anti-aggressive effect, which was mimicked by the PPAR-alpha agonist, GW7647 and blocked by the PPAR-alpha inhibitor GW6471. Of note, PEA did not show any pharmacological effect in PPAR-alpha KO mice. Locomotor activity was not altered by any of these treatments. We suggest that PPAR-alpha may be considered a novel pharmacological target to modulate emotions by stimulation of neurosteroidogenesis and, therefore, may be useful to counteract PTSD symptoms.
*This study is funded by DOD award W81XWH-15-1-0521 to GP.*

**C10 UG-A**

**ASSESSMENT OF DRUG THERAPIES ON A DROSOPHILA PARKINSON'S MODEL AND THEIR EFFECTS ON MOTOR FUNCTION AND AGGREGATE FORMATION**

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Parkinson's disease (PD) is the second most common neuro-degenerative disease that affects humans worldwide. It is characterized by the progressive loss of the main source of dopamine in the nervous system, the dopaminergic neurons. Mutations in several neuronal proteins have been associated with PD, particularly α-Synuclein, a main component of Lewy bodies. Lewy bodies are are caused by the aggregation of proteins, creating oligomers leading to neuronal death. These clusters are associated with the characteristic loss of physical and mental function that occurs with PD. In this study, we are using a Drosophila model of Parkinson's disease to examine the effect of Levodopa (L-Dopa) and novel drug therapies on motor function. Previous studies have demonstrated that Parkinson's disease can be modeled in Drosophila by overexpression of α-Synuclein in neurons. In this model, overexpression of α-Synuclein results in reduced climbing ability that worsens with age. Using this model, we first tested L-Dopa, which is currently used as a treatment for PD, and observed a temporary improvement in climbing ability. In addition, we have also tested p compounds which aggregate formation protein in vitro. Specifically, we have tested black tea extract (BTE), baicalein (Baic), epigallocatechin gallate (EGCG), and nordihydro-guaiaretic acid (NDGA). Preliminary data suggests that treatment with Baic and NDGA may temporarily improve motor function in flies overexpressing α-Synuclein. We are further exploring this phenotype, as well as the effect of these drugs on aggregate formation in the fly brain.
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**C11 G-A**

**MECHANISM OF IRL 1620 INDUCING NEUROGENESIS FOLLOWING SPINAL CORD INJURY IN MICE EX VIVO**

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Spinal cord injury (SCI) is a major medical concern due to the highly debilitating outcomes. Treatments options are failing to advance, resulting in patients unable to return to former functionality. Our research aims to explore SCI using the Endothelin B ($ET_B$) receptor

agonist, IRL-1620, which helps to stimulate the endothelin system and promote the development and maintenance of neurons in the central nervous system. In previous studies looking at the therapeutic potential of using IRL-1620 following cerebral ischemia in mice, IRL-1620 showed significant enhancement of neurogenesis and improved recovery following trauma. Using an *ex vivo* model of SCI we showed that treatment with IRL-1620 enhance proliferation and the expression of markers for neuronal stem cells/neural progenitors. The overall focus of this study is to identify the mechanism of IRL-1620.

Samples treated with IRL-1620 showed an increased expression of NGF and VEGF both of which are known to activate a number of different intracellular signaling pathways, including protein kinase C, mitogen-activated protein kinase (MAPK)/extracellular signal–regulated kinase (ERK), p38 MAPK, and phosphatidyl inositol 3-kinase (PI3K)/Akt/protein kinase B. The ERK signaling cascade is reported to control the proliferation of multiple cell types, including neural stem/progenitor cells. In contrast, Akt signaling is best known for mediating cell survival, cell cycle progression, and stem cell self-renewal. Studies have shown that the activation of both MEK/ERK and PI-3Kinase/Akt is required for promoting neural progenitor cell proliferation.

We hypothesize that a selective stimulation of $ET_B$ receptors using its agonist IRL-1620, will promote neuronal stem cell renewal via activation of the PI3K/AKT and MEK/ERK pathways. Spinal Cords of adult Swiss-NIH mice were collected and 250 300 $\mu$m thick transverse sections were obtained with a tissuechopper and cultured for up to 7 days in either MEM only (control) or MEM + IRL-1620 1mM. Analyzed via western blot analysis. The result showed an increase expression of PI3K, AKT and AKT-P at 1 and 3 days and downstream activation of markers associated with proliferation and cell renewal. A role for IRL-1620 in promoting proliferation is also supported by an increased level of Notch1 and downregulation of P27KIP1 in samples treated with IRL-1620. In conclusion, the activation of the ETB receptor with its agonist IRL-1620 initiate a downstream signaling cascade that ultimately enhances proliferation of neuronal stem cells and neural progenitors via activation of the PI3K/AKT pathway. Further investigation will assess the involvement of the MEK/ERK pathway as well.

**C12 G-B**

## VIRAL VECTOR-MEDIATED A-SYNUCLEIN OVEREXPRESSION RAT MODELS OF PARKINSONIAN AND CEREBELLAR VARIANTS OF MULTIPLE SYSTEM ATROPHY

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Multiple System Atrophy (MSA) is a rare, neurodegenerative disorder with an uncertain etiology and pathophysiology. MSA can be stratified into a Parkinsonian variant (MSA-P) characterized by striatonigral degeneration with associated Parkinsonian-like motor features, and a cerebellar variant (MSA-C) characterized by olivopontocerebellar atrophy with associated ataxia. MSA is a unique synucleinopathy, where α-synuclein accumulates preferentially in oligodendrocytes. MSA is believed to be a primary oligodendrogliopathy in which α-synuclein aggregation is thought to elicit dysfunction in oligodendrocytes, causing disruption in myelin and reduced neurotrophic support leading to secondary neurodegeneration. Traditional animal modeling of MSA relies on transgenic murine models in which human α-synuclein is overexpressed using different oligodendrocyte-specific promoters. In this study, we sought to develop novel viral vector-mediated overexpression models of both MSA-P and MSA-C to establish more clinically relevant models for future use as platforms for drug discovery. α-synuclein or GFP was overexpressed in oligodendrocytes using a recently developed, novel oligotrophic adeno-associated virus vector, Olig001. Sprague-Dawley rats were injected in the striatum to model MSA-P and in the pontine nucleus and middle cerebellar peduncle to model MSA-C. Histological analysis showed 94-97% of the GFP-positive cells co-localizing with oligodendroglial marker Olig2. There was little co-expression in neurons (2.9-4.7%) or astrocytes (0.18-0.49%), indicating the highly oligo-specific tropism of this vector in vivo. Widespread α-synuclein accumulation was seen throughout the injection areas, which were resistant to Proteinase K digestion, indicating the formation of insoluble inclusions. Loss of myelin was observed in white matter regions containing α-synuclein expression. Unbiased stereological counts indicate a ~20% loss of neurons in the striatum of MSA-P rats. Taken together, our data indicates the establishment of novel animal models of the Parkinsonian and cerebellar variants of MSA, recapitulating key aspects of the disease. Long-term studies are underway to evaluate the progression of pathology and development of motor symptoms.

*This project is funded by the Multiple System Atrophy Coalition.*

**C13 PD-A**

## ELUCIDATING THE SYNAPTIC DEFECTS IN HEREDITARY SPASTIC PARAPLEGIAS USING IPSC CO-CULTURE MODELS

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Hereditary spastic paraplegias (HSPs) are a heterogeneous group of neurogenetic disorders characterized by axonal degeneration of cortical motor neurons, a group of large projection neurons (PNs). How the connections between cortical PNs and their targets, spinal motor neurons (MNs), are affected in HSPs remain largely unknown. We have

generated iPSCs from fibroblasts of HSP patients and showed that these iPSC-derived cortical PNs recapitulate the disease-relevant axonal phenotypes. Here, we seek to determine the synaptic defects in HSP by establishing a co-culture model for SPG3A. We first generated the channel rhodopsin 2 (ChR2)-EYFP expressing iPSC lines (both normal and SPG3A iPSCs) using CRISPR/Cas9-mdeiated homologous recombination. These normal or SPG3A iPSCs were differentiated into cortical PNs ($ChR2^+$), which were then co-cultured with normal or SPG3A spinal MNs derived from regular iPSCs ($ChR2^-$), respectively. After immunostaining, we observed a dramatic increase in the numbers of the $Synapsin^+/EYFP^+/PSD95^+$ synaptic clusters in the co-cultures comparing to single culture of cortical PNs. Furthermore, the electrophysiological analysis revealed robust evoked postsynaptic currents in spinal MNs after the activation of co-cultured $ChR2^+$ cortical PNs using blue light stimulation, indicating the formation of functional synaptic connections between co-cultured cortical PNs and spinal MNs. Finally, to evaluate the synaptic defects in SPG3A co-culture model, we compared the synaptic connections between cortical PNs and spinal MNs in different groups using both immunostaining and electrophysiological analyses. The number of $Synapsin^+/EYFP^+/PSD95^+$ synaptic clusters in SPG3A co-culture models was significant reduced comparing to normal co-culture group. The impaired synaptic connections in SPG3A co-culture models were further supported by the dramatic decrease in the frequency of spontaneous excitatory postsynaptic currents (sEPSC) recorded in SPG3A spinal MNs after the activation of ChR2-expressing cortical PNs. Taken together, our data reveal the impaired synaptic connections between cortical PNs and spinal MNs in a SPG3A co-culture model, which will serve as a unique system to study the pathogenic mechanism and explore the treatment for HSPs.

*This work has been supported by the Blazer Foundation and the NIH (R21NS089042).*

**C14 G-A**

**ENVIRONMENTAL AND GENETIC CONTRIBUTIONS IN AN ALS RAT MODEL: FAILED RECOVERY AND ENHANCED VENTRAL HORN INFLAMMATION AFTER PERIPHERAL NERVE INJURY**

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**Background:** ALS is poorly understood and no effective therapeutics exist to stop its insidious progression. Patients may present initially with single limb weakness or difficulty talking or swallowing. Once it starts, the disease progresses up and down the spinal cord until respiratory failure leads to death or mechanical ventilation. Clinicopathologic human studies have shown a clear relationship between disease onset and lower motor neuron loss, with the most severe lower motor neuron loss at the site of disease onset. One long entertained observation is that ALS may be precipitated by nerve or brain injury. In fact, many patients with ALS are athletes (Lou Gehrig) or veterans, and may have suffered minor nerve injuries in the limb where ALS first presents. However, it is unclear whether and how nerve injury plays a role in ALS development. Our lab and others have previously shown an increase in spinal cord microglial activation in post-mortem ALS patient tissue, as well as at early stage in animal models of ALS, suggesting microglial activation contribution in early stage of neurodegeneration. These cells are also the primary immune cells of the nervous system and are known to respond to nerve injury. **Objectives:** Our current study seeks to link an environmental factor (nerve injury) with a genetic factor (SOD1 mutation) to induce symptom onset and disease progression while characterizing the pattern of pathological glial cell response to injury. **Methods:** We performed sciatic nerve crush injuries in SOD1 G93A rats and age, sex and background matched wild type controls at 10 weeks of age, prior to any known symptoms. Functional recovery using the EPT test was tracked weekly following surgery. Spinal cord tissue was collected at different stages of disease for tissue staining and quantitative analysis of glial cells and motor neurons. **Results:** Unlike in wild types, injury induced permanent functional impairment in SOD1 animals, which showed faster rates of motor decline and decreased survival compared to uninjured SOD1 littermates. Significantly enhanced and sustained microglial activation was seen in the ventral horns of SOD1 rats 1-2 weeks after injury that spread to nearby, uninjured motor neuron pools. Microglial activation subsided by 6 weeks, however, astrocyte activation was increased at this time in injured SOD1 animals compared to uninjured SOD1 controls. One consequence of this prolonged glial activation is significant synaptic loss on lower motor neuron cell bodies in the injury-affected region of the spinal cord. **Discussion and Conclusions**: These studies take a unique approach to understand the effects of early environmental contributions (nerve injury) in a genetic model of ALS. They provide mechanistic insight of inducible glial activation and cell damage, as evidenced by synaptic loss, that may be responsible for functional impairments and decreased survival. This animal model could serve as an important new system for drug development that focuses on disease onset and progression rather than traditional models of survival and therefore may translate better to the human condition.

**C15 UG-B**

**LOW-DOSE ASPIRIN STIMULATES DOPAMINE PRODUCTION: IMPLICATIONS FOR PARKINSON'S DISEASE**

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It is believed that motor signs first appear in patients with Parkinson's disease (PD) when about 30-50% of substantia nigra (SN) dopamine neurons are lost. Therefore, increasing the function of residual dopaminergic neurons in the nigra of PD patients is an important area of research as it may eventually compensate the loss. Although tyrosine hydroxylase (TH) is the rate-limiting enzyme in the dopamine (DA) biosynthesis pathway, there are no effective drugs/molecules to upregulate TH and increase the production of DA in nigral dopaminergic neurons. Acetylsalicylic acid, commonly known as aspirin, is one of the most frequently used pharmaceutics in medical practice

and is available over the counter. This study underlines the importance of aspirin in stimulating the expression of TH and increasing the level of DA and its metabolites in dopaminergic neurons. Aspirin dose-dependently increased the expression of TH and the production of DA in mouse MN9D dopaminergic neuronal cells with maximum upregulation seen at 5 µM concentration. Time-dependent studies also showed maximum increase in TH and DA at 2 h of aspirin stimulation. While investigating mechanisms, we found the presence of cAMP response element (CRE) in the promoter of TH gene and the rapid induction of cAMP response element binding (CREB) activation by aspirin in dopaminergic neuronal cells. The abrogation of aspirin-induced expression of TH by siRNA knockdown of CREB suggests that aspirin stimulates the expression of TH in dopaminergic neurons via CREB. These results highlight a new property of aspirin in stimulating the TH-DA pathway, which may be beneficial in PD patients.

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**C16**

## TRANSECTION RAPIDLY REORGANIZES THE CORTICAL ACTIN CYTOSKELETON OF THE NEURITE IN VITRO

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Traumatic brain injury is a heterogeneous, neurological disorder that may involve edema, ischemia, hemorrhage, skull fracture, contusion and diffuse axonal injury (DAI). DAI occurs when axons are hyper-extended during impact. This insult triggers neurite dystrophy. Specifically, the normal cylindrical shape of the neurite transforms into a beads-on-a-string morphology. Dystrophy is conventionally attributed to axonal transport failure arising from microtubule fracture. This study addressed the hypothesis that actin reorganization also plays a role in post-traumatic neurite dystrophy. Human induced pluripotent stem cell derived neurons (hiPSCNs) were cultured on glass coverslips and stained with SiR-Actin to visualize F-actin fibers. Neurites were transected using a glass microneedle mounted on a micromanipulator and then imaged over time with high speed, 3D, single channel fluorescent structured illumination microscopy. The degree of dystrophy was quantified as the standard deviation of the caliber along the length of the neurite within a user-defined region of interest. Trauma rapidly increased the dystrophy of the F-actin cytoskeleton but there was no statistically significant influence of jasplakinolide on this trend. Jasplakinolide reorganized the microstructure of the F-actin cytoskeleton, as quantified by the lacunarity, but there was no statistically significant influence of trauma on this trend. The F-actin concentration of dystrophic structures appeared to vary in this data set. To explore this phenomenon, new cultures were stained with SiR-Actin and DiI, a membrane stain. They were transected and then fluorescently imaged over time across both channels in 3D using lattice light sheet microscopy. These images reveal that the F-actin cortex forms a shell inside some dystrophies after trauma. In some cases, the lipid bilayer remains adjacent to the F-actin cortex within dystrophies while in others, it separates from the F-actin cortex. These results suggest that post-traumatic neurite dystrophy may not be a homogeneous phenomenon explained by a single mechanism. Furthermore, the actin cortex is not obliterated at neurite dystrophies but rather changes shape along with the neurite. This result raises the possibility that the actin cortex may influence the formation of some neurite dystrophies by modulating the stiffness and tension at the surface of the neurite.

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**C17 PD-B**

## MITOCHONDRIAL OXIDATATIVE STRESS REGULATES TGF-β2 DEPENDENT REMODELING OF THE EXTRACELLULAR MATRIX IN HUMAN TRABECULAR MESHWORK CELLS

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Primary open angle glaucoma (POAG) is the leading cause of irreversible blindness worldwide. POAG is characterized as progressive optic neuropathy, elevated intraocular pressure (IOP) is a major risk factor for the progression of the disease. Numerous studies have also implicated elevated levels of oxidative stress markers in aqueous humor (AH) of POAG patients, along with altered expression of antioxidant defenses in the trabecular meshwork (TM). Selective oxidative damage to TM mitochondria (Mt) elicits TM cell dysfunction. Dysregulation of the TM is thought to initiate extracellular matrix (ECM) remodeling and impair AH outflow, culminating in the aberrant elevation IOP. Whereas elevated levels of TGF-β2, a profibrotic cytokine, is known to enhance ECM remodeling in TM, the mechanism by which this occurs is unknown. Mt-generated reactive oxygen species (ROS) are required for TGF-β induced changes in gene expression in other cell systems. Here, we investigated the role TGF-β2 plays at promoting Mt-ROS regulated ECM remodeling in primary or transformed human TM cells. TGF-β2 (5 ng/ml) elicited a dose- and time-dependent increase in intracellular ROS, as semi-quantified using CellROX or DCFDA fluorescent probes. Challenging TM cells with TGF-β2 similarly increased the expression of collagens (Col1α1 and Col4α1) and connective tissue growth factor (CTGF), as quantified by RT-qPCR using human specific primers. Pre-treating TM cells with SB-431542 (10 µM), an ALK-5 inhibitor, or with Mt-targeted antioxidants prevented TGF-β2 induced increases in intracellular ROS generation, as well as TGF-β2 induced transcription downstream of Smad signaling and expression of collagen and CTGF gene expression. These findings support a role of ROS as an important

mediator of TGF-β2 signaling and ECM remodeling in human TM. Thus, we propose that Mt-targeted antioxidants may represent a novel strategy to manage elevated IOP associated with POAG.

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**C18 G-B**

**SINGLE NUCLEOTIDE POLYMORPHISMS OF GCH1 ASSOCIATES WITH SICKLE CELL DISEASE PAIN IN AFRICAN AMERICANS**

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The inadequate therapeutic management of pain in sickle cell disease (SCD) can be attributed to our limited knowledge of its multifaceted nature. We explored the association of SCD pain with 5 GTP-Cyclohydrolase (*GCH1)* single nucleotide polymorphisms (SNPs) of interest. GCH1 the rate-limiting enzyme in tetrahydrobiopterin biosynthesis- a cofactor involved in the synthesis of several pain modulators. Blood/buccal swab samples collected from 132 subjects were genotyped using MassARRAY iPLEX platform. Composite pain index (CPI) scores obtained from pain assessment tool, PAINReportIt®, and acute care utilization scores were used as markers for chronic and acute pain respectively. CPI scores were fitted using multiple linear regression (MLR) and utilization scores using negative binomial regression (NBR), for additive, dominant and recessive models. SNPs were in Hardy-Weinberg equilibrium (p>0.05). The A allele of rs3783641 was associated with increased utilization in the additive and recessive NBR models for acute pain (IRR= 1.39, 1.84; 95%CI= [1.07, 1.82], [1.13, 3.10]; p= 0.017, 0.015). It was also found that the C allele of rs8007267 was associated with decreased CPI scores for the additive and dominant MLR models (B= -3.69, -5.41; 95% CI= [-7.21, -0.17], [-10.17, -0.66]; p= 0.040, 0.026). Additionally, we identified two haploblocks based on linkage disequilibrium plot. For haploblock rs10483639[G>C]-rs752688[C>T]-rs4411417[T>C], haplotype CTC was associated with high utilization (4 or more) compared to the reference haplotype GCT (OR= 2.05, p= 0.049). For haploblock rs3783641[T>A]-rs8007267[T>C], haplotype TC was less likely to have high utilization than the reference haplotype AT (OR= 0.30, p= 0.001). These data indicate that genetic polymorphisms of *GCH1* may contribute to some of the pain heterogeneity in SCD.

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**C19 G-A**

**LY6K PROMOTES GLIOBLASTOMA TUMORIGENESIS BY ENHANCEMENT OF ERK SIGNAL TRANSDUCTION**

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Glioblastoma (GBM) is the most malignant brain cancer, with a median survival of approximately 15 months for GBM patients. Distinct molecular subtypes, characterized as proneural (PN), classical, and mesenchymal (MES), as well as inherited heterogeneity render GBM tumors resistant to current therapies. In addition to the genetic heterogeneity arising from the differentiated cells, GBM tumors also contain a small population of glioma-initiating or stem-like cancer cells (GSCs). Gene expression profiling studies from our lab and others showed that patient-derived GSCs can also be classified into subtypes phenotypically similar to GBM. We found over three thousand genes that are differentially expressed between PN and MES-like GSCs. Of these, *Lymphocyte Antigen 6 Complex, Locus K (LY6K)* was one of the top differentially expressed genes. LY6K is a GPI-anchored protein from the LY6 family. Various members of the LY6 family, including LY6D, LY6E, and LY6K have been implicated in human cancers such as breast, esophageal, and lung cancers. In our preliminary studies, we tested the roles of *LY6K* upregulation in GBM tumorigenesis and examined the underlying mechanism responsible. We found that **1)** high levels of *LY6K* expression correlates with poor prognosis of patients with GBM; **2)** modulation of *LY6K* expression in GSCs significantly altered tumorigenic behavior *in vitro* and *in vivo*; **3)** modulation of *LY6K* expression affected p-ERK1/2 activation, which is likely a downstream effect of enhanced EGFR signaling. These data indicate that LY6K enhances GBM tumorigenesis by enhancing EGFR signaling, thereby promoting p-ERK1/2 activation. Our results also provide critical insight for advancing our understanding of how oncogenic EGFR signaling could be enhanced by other tumor-associated genes such as LY6K, thereby promoting GBM tumorigenicity and therapy resistance.

**C20**

**KNOCKDOWN OF CORTICOTROPIN RELEASING HORMONE RECEPTOR 1 (CRF1) IN THE ROSTRAL PERI-CELLULAR REGION OF THE LOCUS COERULEUS (LC) NORMALIZES ANXIETY-LIKE BEHAVIOR IN FEMALE RATS SUBJECTED TO CORONARY ISCHEMIA AND REPERFUSION INJURY (IR)**

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12-15% of patients develop post-traumatic stress disorder (PTSD) following myocardial infarction (MI). Women develop more severe symptoms of post-MI PTSD, and symptom severity is predictive of cardiovascular morbidity. Similarly, we found that female rats exposed

to MI show deficits in fear extinction while males do not. During exposure to conditioned fear cues, amygdala projections release corticotropin releasing hormone (CRF) near the rostral LC to cause norepinephrine release in the medial prefrontal cortex (mPFC). In the absence of an aversive event, this process leads to fear extinction. To assess the role of CRF in MI-induced extinction deficits, freezing to conditioned auditory fear cues was examined 8 weeks after IR-induced MI or sham surgery in female and male rats previously treated with bilateral rostral LC injection of viral vectors encoding either an anti-rat CRF1 shRNA sequence or a scrambled sequence. Freezing was measured during 10 extinction trials. One day later, rats were anesthetized with ketamine and perfused with fixative to enable in situ detection of CRF1 mRNA in rostral and caudal peri-LC regions. The IR surgery impaired left ventricular (LV) function more in males than females (23.3±3.1 [n=17] vs 33.7±4.4% [n=18] fractional shortening). During extinction, females with LV damage (fractional shortening ≤40%) froze more than sham females (99.7±12.3 [n=4] vs. 41.6±7.3 seconds [n=6], P<0.01). Treatment with anti-CRF1 shRNA reduced freezing in IR females (43.3±7.7 seconds [n=6], P<0.05). Males showed similar trends that were not significant (82.9±16.0 [n=9] vs. 57.1±9.7 [n=10] and 58.6±14.4 seconds [n=9]). Rostral peri-LC CRF1 mRNA was 66% higher in sham females than sham males treated with control virus (P<0.01). ShRNA treatment reduced rostral peri-LC CRF1 mRNA by 22.9% (P<0.05) and 42% (P<0.01) in sham female and male rats respectively. Rostral, but not caudal peri-LC CRF1 mRNA levels were reduced in female and male IR rats treated with control virus compared to their respective sham groups injected with control virus (-58%, P<0.01 and -38%, P<0.05 respectively). CRF1 mRNA levels were not different between IR rats treated with shRNA-encoding- or control virus. Rostral peri-LC CRF1 mRNA levels were inversely correlated with freezing across all females treated with control virus (P<0.01). Together, the data suggest that exposure to fear cues causes activation (and downregulation) of rostral peri-LC CRF1 mRNA. Exaggerated activation of this pathway in female IR rats appears to contribute to deficits in fear extinction.

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**C21**

**EXAMINING THE STRIATAL PROTEOME IN DOPAMINE DEFICIENT MOUSE MODELS: IMPLICATIONS IN PARKINSON'S DISEASE**

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Dopamine (DA) deficiency in the striatum underpins the motor symptoms of Parkinson's Disease (PD). How the striatal proteome is remodeled in the absence of DA has not been examined in depth and examining this profile may provide valuable targets for pharmacological manipulation.

To evaluate this, we have recently developed two DA deficient conditional mouse models in which tyrosine hydroxylase (TH), the rate limiting enzyme for DA synthesis, is eliminated selectively in midbrain DA neurons. The first uses the dopamine transporter (*Dat*) to drive Cre recombinase expression and eliminate TH embryonically (Dat::Cre, Th cKO), whereas the second, uses Dat::CreER$^{T2}$, a tamoxifen inducible Cre recombinase, to eliminate Th in an adult mouse (Dat::CreER$^{T2}$, Th cKO). Dat::Cre, Th cKO mice survive with 95% DA depletion in the striatum and display a motor phenotype in which they show no motor learning ability as assessed by latency to fall on an accelerating rotarod. For Dat::CreER$^{T2}$, Th cKO, after tamoxifen regimen, we show a ~60-70% DA depletion as well as a decrease in motor performance on the rotarod as compared to controls, though this phenotype is not as severe as the Dat::Cre, Th cKO mice. A proteomics study of the striatum of the Dat::Cre, Th cKO was analyzed by tandem mass spectrometry (MS). Preliminary MS analysis demonstrated that ~4,000 different proteins were detectable in mutants and control samples, with several proteins, including TH, showing differential expression. One pathway of interest showed multiple proteins upregulated significantly, the mitogen activated protein kinase (MAPK) pathway which is involved in many cellular processes including regulation of transcription. By comparing the Dat::Cre, Th cKO and Dat::CreER$^{T2}$, Th cKO through western blot we can validate the proteomics and target important potentially maladaptive pathways such as the MAPK pathway. Modulating the MAPK pathway through the metabotropic glutamate receptor 5 (mGluR5) may ameliorate the motor phenotype seen in these DA depleted models.

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**C22 UG-A**

**INSIGHT INTO PARKINSON'S DISEASE FROM YEASTS: GROWING EVIDENCE FOR SUMOYLATION AS A PROTECTIVE FACTOR AGAINST A-SYNUCLEIN TOXICITY**

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Parkinson's disease (PD) is a neurodegenerative disorder linked to the loss of dopaminergic neurons in the midbrain. A key pathological marker of PD is the presence of Lewy bodies, which are mainly composed of misfolded alpha-synuclein protein. Alpha-synuclein is a highly post-translationally modified protein. While phosphorylation and nitration of alpha-synuclein is well studied as aids to PD pathology, less is known about sumoylation, which is proposed to be neuroprotective based on limited studies. The majority of sumoylation takes place on the lysine-96 and lysine-102 sites of alpha-synuclein and it increases the protein's solubility. The goal of this research was to better understand the role of sumoylation in regulating alpha-synuclein toxicity, and we performed four studies towards it. First, we evaluated the effects of blocking sumoylation on alpha-synuclein in the well-established

budding and fission yeast models for PD and found that alpha-synuclein becomes more aggregated and toxic and localized less at the plasma membrane. Second, we evaluated the effects altering sumoylation pathways by using yeast strains with reduced ($ulp1^{ts}$) or excessive sumoylation ($smt3^{ts}$), and found that alpha-synuclein aggregates more with reduced sumoylation, but becomes less toxic with increased sumoylation. Third, we asked how altering phosphorylation of alpha-synuclein would alter sumoylation's protective role and found that blocking phosphorylation reduced alpha-synuclein toxicity. Finally, we evaluated whether blocking sumoylation on familial PD mutant versions of alpha-synuclein would exacerbate its toxicity, but we have found little evidence to that effect. In the future, we will conduct further studies to understand how sumoylation affects other variants and modifications of alpha-synuclein.

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**C23 PD-A**

**REPURPOSING AN FDA APPROVED CARDIAC GLYCOSIDE FOR MYELIN REPAIR THERAPY IN COMBINATION WITH IMMUNE TOLERANCE IN MULTIPLE SCLEROSIS.**

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Multiple Sclerosis (MS) is a CNS autoimmune disease characterized by demyelination and neurodegeneration. Currently, there are no available therapies marketed for myelin repair in MS. The aims of my work included prevention of disease progression and promotion of CNS repair and neuroprotection. In an effort to repurpose FDA approved medication to expedite therapies to patients, we tested a cardiac glycoside (Na+/K+ ATPase) and revealed it promoted an increase in the oligodendrocyte cell lineage *in vitro* and *in vivo*, in the non-T cell-mediated Cuprizone model of demyelination/remyelination promoted a quicker restoration of myelin integrity, and improved clinical score throughout the autoreactive Th1/Th17 driven C57BL/6 Chronic experimental autoimmune encephalomyelitis (EAE) time course. Currently available disease modifying therapies for MS are global immunosuppressants and have limited efficiency. We are able to induce immune tolerance, through induction in autoreactive T cells using i.v. infusion of nanoparticles coupled with or encapsulating myelin peptides (Ag-PLG), to selectively regulate known immune responses without compromising the entire adaptive immune system. We have demonstrated an effective means of ameliorating disease in a mouse model of MS that effectively reduces disease burden in relapsing-remitting (RR-EAE) and chronic-progressive (C-EAE) mouse models. Prophylactic administration can prevent disease induction, but more importantly therapeutic administration can stop disease progression in mice. The hypothesis was that to effectively target disease course and severity in MS, regulated by autoimmunity and neurodegeneration, a combination of selective immune regulation and myelin repair therapy is required. Combination therapy using Ag-PLG immunoregulatory therapy and the cardiac glycoside completely ameliorated clinical disease severity. These promising results provide pre-clinical evidence for future clinical studies in MS undertaking this combinatorial therapeutic approach.

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**C24 G-B**

**ACTIVATED MESENCHYMAL STEM CELLS INCREASE LONG-TERM RECOVERY FOLLOWING ISCHEMIC STROKE VIA REDUCTION OF MICROGLIA ACTIVATION AND INDUCTION OF OLIGODENDROGENESIS**

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Stroke is the most common cause of adult disability worldwide with few available treatment options. Natural brain repair mechanisms fail to promote recovery after stroke and little is known about why these mechanisms fail. To address this lack of understanding we utilized a rat model of ischemic stroke. In contrast to previous literature, we see no changes in the neurogenic processes in vehicle treated animals following stroke. Also, microglia activation is sustained in the ipsilateral hemisphere without treatment after stroke. Because mesenchymal stem cells (MSCs) secrete various neurotrophic factors and are known to dampen immune responses we examined their efficacy for treating stroke. Additionally, *ex vivo* activation with interferon-γ has been shown to enhance the paracrine effects of MSCs so our studies utilized both naïve MSCs as well as interferon-γ-activated MSCs. Following MSC treatment, there is an increase in proliferating cells in the subventricular zone (SVZ) as well as an increase in oligodendrocyte progenitor cells suggesting that MSC treatment induces oligodendrocyte differentiation. Additionally, there is a significant reduction in microglia activation with MSC treatment. Lastly, rats administered MSCs have a more rapid and sustained functional improvement compared to vehicle treated animals. Taken together, these results suggest that MSCs improve functional recovery by increasing oligodendrogenesis and reducing microglia activation after ischemic stroke.

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**C25 UG-B**

**DIFFERENTIAL REGIONAL BUILDUP OF DISTINCT AB OLIGOMER SPECIES IN THE 5XFAD MOUSE MODEL OF ALZHEIMER'S DISEASE**

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**Purpose.** Alzheimer's disease (AD) is a progressive, neurodegenerative disease and is the sixth leading cause of death in the U.S. Amyloid beta oligomers (AβOs) have been found to contribute to AD pathogenesis and have been shown to cause pathologies linked to AD such as synaptic loss. Extracellular and membrane-associated AβO species have been found in the AD brain and evidence indicates that these AβOs vary in abundance during AD progression. However, it is not known which AβO type is most prevalent throughout AD pathogenesis. My long-term research goal is to characterize the abundances of different AβO types that are present in the AD brain. In this project, I have been mapping the buildup of different AβOs in the 5xFAD mouse model as the mice age through multiple antibodies and immunoassays. **Methodology.** The 5xFAD mouse model has been shown to exhibit many pathologies characteristic of AD, including AβO buildup. I have been utilizing the 5xFAD mouse model at selected time points (3, 6, 9 months) to measure the abundances of water-soluble and membrane-associated AβOs present during AD progression. I also am testing three brain regions: hippocampus and cortex, which are associated with AD and the cerebellum, which is not. Antibodies (NU2, NUsc1) with AβO specificity have been used in dot immunoblots and ELISA assays to measure the amounts of water-soluble AβOs, membrane-associated AβOs and insoluble Aβ present. Our antibody NUsc1 is of interest due to its specificity for a toxic subpopulation of AβOs.

**Findings.** As expected, we have found that the amount of AβOs in the 5xFAD mouse brain increases as the mice age. Preliminary data also indicates that the abundance of water-soluble AβOs, membrane-bound AβOs, and insoluble Aβ varies with brain regions. Water-soluble and membrane-associated AβOs are more abundant in the hippocampus and the cortex in comparison to the cerebellum. Water-soluble AβOs increased in abundance in the hippocampus and the cortex over time, with the majority of water-soluble AβOs accumulating in the hippocampus. Membrane-associated AβOs remained constant in the hippocampus, while also showing sudden accumulation in the cortex at 9 months. This may be indicative of water-soluble and membrane-associated AβO regional specificity. We have also found a NUsc1-reactive AβO subpopulation present in the hippocampus of 3- and 6-month 5xFAD mice. **Practical Implications.** This data can lead us to understand if water-soluble and membrane-associated AβOs are abundant during each stage of AD and in each brain region. This data can also be used to diagnose AD stages if a ABO species progression trend is identified. Additionally, the antibody NUsc1 has shown specificity for a toxic AβO population, which could potentially lead to its use as a diagnostic and/or therapeutic tool.

**C26 G-A**

**CLOSED-HEAD INJURY MODEL OF REPEAT SUBCONCUSSION IN THE ADULT RAT**

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Although concussions have become a large focus of neurological study, especially in athletes and the military, brain injuries that occur with higher frequency but are less studied are repeat subconcussions. A subconcussion is loosely defined as an impact to the head that does not result in a diagnosable concussion or concussive symptoms. Repeat subconcussions consist of multiple hits to the head and have been shown to produce significant cognitive and behavioral impairments along with neurological pathology (DeFord et al., 2002; Bailes et al., 2013). This study was designed to model repeat subconcussive events in the adult rat. Using a model of closed head single and repeat concussions created in our lab (Jamnia et al., 2017), this study modified the intensity of the impact to creat a subconcussive impact. Rats received either a single concussion, single subconcussion, repeat subconcussion, or sham injury. Using behavioral tests including air-righting, balance beam, foot fault, and novel object recognition, we compared responses among groups over a 30-day timeline. Animals with a single concussion and with repeat subconcussive events showed deficits in locomotion, righting reflexes, and recognition memory, while animals with a single subconcussion did not. In both the air-righting test and novel object test, similar deficits were observed between the single concussion and repeat subconcussion groups ($p<0.001$; $p<0.01$). In the foot fault test the repeat subconcussive group showed worse deficit than the single concussion group ($p<0.05$). Foot fault and novel object test deficits persisted until post-injury day 30 ($p<0.05$). Our model of repeat subconcussive events result in deficits that are similar to or worse than a single concussion, which suggests that this model would provide a good comparison to clinical responses seen after repeat subconcussive events. Further experiments are underway to determine if there are any sex differences in the behavioral responses to repeat subconcussions along with an examination of the neuropathological response.

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**C27**

**XANTHOHUMOL EXERTS CELL-TYPE SPECIFIC EFFECTS AGAINST OXIDATIVE STRESS *IN VITRO***

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Dry age-related macular degeneration (AMD) is the most common cause of irreversible blindness in the developed world and comprises more than 90% of all patients diagnosed with AMD. Dry AMD is characterized by a slow progressive photoreceptor loss in the macula and changes in the retinal pigment epithelium, ultimately leading to blindness. Currently, there are no treatments for dry AMD, highlighting the urgent need for discovery and development of novel pharmacological interventions. It is generally accepted that oxidative stress is an important mediator in the pathogenesis of several retinal diseases, including AMD. Therefore, activation of the endogenous antioxidant system may be a feasible approach to slow the progression of dry AMD. Xanthohumol (2',4',4-trihydroxy-6'-methoxy-3'-prenylchalcone, Xn) is a polyphenol chalcone from hops (*Humulus lupulus*) that exerts potent neuroprotective effects that have been attributed to the selective activation of the transcription factor, *Nrf2*, resulting in an increase in endogenous antioxidant potential. Herein, we tested the effects of Xn *in vitro* in 661W photoreceptor cells. Specifically, 661W photoreceptor cells were seeded in 96-well plates at 10,000 cells per well. Cells were serum-deprived and treated with either vehicle (DMSO) or Xn (0.1 μM or 0.5 μM) 24 hr prior to exposure to exogenously-applied chemically-induced oxidative stress using *tert*-butylhydroperoxide (*t*BHP; concentration range 10 μM to 1.5 mM). 661W cells showed a significant increase in proliferation in response to DMSO at concentrations ranging from 0.01 – 0.1% (v/v) as assessed by the MTT conversion assay. Concomitantly, 661W cells were sensitized to the deleterious effects of exogenously-applied oxidative stress. Xn treatment shifted the $IC_{50}$ for *t*BHP from 399.1 μM (DMSO) to 495.2 μM and 520.4 μM (0.1 μM Xn and 0.5 μM Xn, respectively), but did not result in a statistically significant protection of 661W cells against *t*BHP-induced oxidative stress, as assessed by Two-Way ANOVA ($P = 0.75$; n = 3 separate experiments). For comparison, Xn exerted strong dose-dependent glioprotection against *t*BHP-induced oxidative stress in primary adult rat optic nerve head astrocytes (ONHAs) ($P < 0.01$). In conclusion, Xn exerts cell-type specific effects against oxidative stress *in vitro*. *In vivo* experiments are currently underway that are determining the potential neuroprotective effects in the mouse light-induced retinal neurodegeneration model and related *in vivo* ocular models characterized by increases in oxidative stress.

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## *THEME E. HOMEOSTATIC AND NEUROENDOCRINE SYSTEMS*

### E1 UG-B
### ACTIVATION OF HYPOTHALAMIC OXYTOCIN NEURONS IN RESPONSE TO FEAR CONDITIONING

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Oxytocin (OT) is a hypothalamic neuropeptide that modulates not only social behavior but fear and anxiety-like behaviors. OT neurons from the hypothalamus project to the dorsolateral bed nucleus of the stria terminalis (BNSTdl), a region involved in the modulation of fear and anxiety-like behavior. Utilizing a fear-potentiated startle (FPS) paradigm, we previously showed that blocking OT receptors (OTR) in the BNSTdl attenuated acquisition of cued fear. This suggests that OT is released in the BNSTdl during fear conditioning and that neurons producing OT are engaged during fear learning process. To determine whether the OT neurons in the hypothalamus were activated in response to fear conditioning, we exposed adult Sprague-Dawley rats to cued fear conditioning (ten presentations of cue light co-terminating with a foot shock) or contextual fear conditioning (ten presentations of foot shock alone), and compared them to controls placed in the conditioning chambers with no shock or cue presentations. Following fear conditioning, rats were perfused and brain sections from the entire hypothalamus were double-immunolabeled with antibodies against OT and the immediate early gene, cFos, and imaged using confocal microscopy with Olympus FV10i. Relative to controls, rats exposed to cued or contextual fear conditioning showed greater activation of OT neurons within all hypothalamic nuclei. Within the PVN, rats exposed to contextual fear conditioning showed a greater percentage of OT neurons co-expressing cFos relative to control rats, while no difference was seen in rats exposed to cued fear conditioning. In the supraoptic nuclei, animals exposed to either contextual fear conditioning or cued fear conditioning displayed a greater percentage of OT neurons co-expressed cFos compared to control animals. Similarly, in accessory hypothalamic nuclei, a significantly greater percentage of OT neurons co-expressed cFos in rats exposed to cued or contextual fear conditioning relative to control animals. These results suggest hypothalamic OT neurons are activated in response to both cued and contextual fear conditioning and may be the source of OT release into the BNSTdl during fear
learning.

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*DePaul-RFUMS seed research grant to JD, as well as start-up funds from the Chicago Medical School, Rosalind Franklin University of Medicine Science to JD.*

### E2 UG-A
### CONTINUOUS SOCIAL ISOLATION IMPAIRS FEAR EXTINCTION LEARNING: POSSIBLE ROLE OF THE OXYTOCIN RECEPTOR

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Environmental stressors are known to modify neuronal activity (Kolb & Whishaw, 1998). Human studies suggest that social isolation induces

anxiety and prolongs recovery from psychological trauma (Cacioppo et al., 2014). Conversely, having strong social support enhances stress-coping strategies and reduces the risk of developing anxiety and post-traumatic stress disorder (Hansen et al., 2017; Malicka et al., 2016). While most studies quantify the effects of rodent isolation on fear as time spent freezing, the translation of such findings to humans is challenging. We instead utilize the fear-potentiated startle (FPS) paradigm, indicative of fear in both rodents and humans (Grillon et al., 1993). In the FPS, an acoustic startle reflex (ASR) is significantly potentiated by an exposure to a conditioned stimulus (CS+, cue) that has been previously paired with foot shock (unconditioned stimulus, US). Twenty-four hours after fear conditioning, during an FPS testing session, rats are subjected to startle-eliciting, 95 dB white noise bursts, with (CS+) or without (CS-) presentations, mixed in a pseudorandom order. To determine the effects of social isolation on fear and anxiety-like behavior, our objective was to measure cued and non-cued fear, both of which we hypothesized would be potentiated by isolation. To this end, male Sprague Dawley rats (n=44) aged 55-60 days were housed singly with no haptic and limited olfactory contact with conspecifics, while control animals were housed socially in pairs or trios. Social isolation did not potentiate the acoustic startle reflex and it did not affect fear memory recall tested 24 hours after fear conditioning. However, socially isolated rats displayed potentiated cued fear compared to socially housed rats during second FPS test, suggestive of impaired cued fear extinction learning. As oxytocin (OT) is a hypothalamic hormone and a neuromodulator, which has been shown to regulate social behavior, these findings led us to hypothesize that OT may facilitate fear extinction in socially housed male rats. Thus, we acutely blocked peripheral and central OT receptors (OTR) using an OTR antagonist (systemic injection of L-368,899, 5 mg/kg) in male rats (n=48) one hour before their first FPS testing. Our results show that OTR is involved in the modulation of fear extinction in socially housed but not socially isolated rats. These results highlight the critical role of social environment in the modulation of fear memory.

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**E3 UG-B**

**DO BEHAVIORALLY INHIBITED INDIVIDUALS EXHIBIT GREATER MINUTE VENTILATION AND BREATHING FREQUENCY THAN NON-INHIBITED INDIVIDUALS WHEN BREATHING ENHANCED CO2?**

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Behaviorally inhibited (BI) individuals tend to avoid and withdraw from unknown situations and environments, and respond differently to stress than non-BI individuals. After a stressful situation, BI individuals are also more susceptible to developing severe anxiety disorders such as Post-Traumatic Stress Disorder (PTSD) (North et al., 2008); however, it is unknown if there is a difference in the physiologic response to stress between BI and non-BI individuals. Our current studies will test the hypothesis that BI individuals have a heightened physiological response to a stressful stimuli which also exceeds the duration of the stimuli. In each participant, we will induce physical stress using a respiratory challenge (inspired 7% $CO_2$ gas) and we will asses BI by utilizing the Adult Measure of Behavioral Inhibition (AMBI) and Retrospective Measure of Behavioral Inhibition (RMBI). During each trial, we will record the participant's tidal volume, minute ventilation and breathing frequency during a 15 minute control period, followed by 5 minutes of 7% $CO_2$ gas. We predict that minute ventilation and breathing frequency rates will increase when all participants are given 7% $CO_2$; however, we speculate that BI individuals will exhibit a greater increase in both minute ventilation and breathing frequency which will take longer to return to control levels when compared to non-BI individuals. The significance of this study is that it can potentially help gauge the physiological responses of BI individuals in high-stress environments such as military combat or space travel. Our future studies will also include measurement of salivary cortisol and amylase to asses the neuroendocrine response to stress in BI versus non-BI individuals.

**E4 G-B**

**MODULATION OF BASOLATERAL AMYGDALA INPUTS INTO NUCLEUS ACCUMBENS BY GSK3B INHIBITION AND ANTERIOR CINGULATE CORTEX STIMULATION TRAINS**

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Human neuroimaging studies have identified greater white matter connectivity from the basolateral amygdala (BLA) to the nucleus accumbens (NAcc) to be a predictor of hypomania disorder. These projections are responsible for facilitating emotional learning and behavioral responses elicited by emotional states, and perhaps inappropriate BLA-NAcc communication is a contributor to mood dysregulation symptoms. Thus, understanding both the neurophysiology of BLA-NAcc projections and the factors that govern their communication, may uncover mechanisms that initiate or exacerbate positive and negative emotional states. Glycogen synthase kinase 3 beta (GSK3β), a downstream target of three distinct mood-stabilizing therapies, has piqued interest for its involvement in the pathophysiology of many neurological diseases, including manic-depression. The anterior cingulate cortex (ACC) is a potential catalyst in emotional valancing, as different patterns of ACC activation shift preference for opposing facial expressions. We hypothesized that inhibition of GSK3β and high frequency ACC stimulation would diminish BLA-NAcc interactions. *In vivo* extracellular electrophysiology was utilized to gauge alterations of BLA-NAcc projections by systemic GSK3β inhibition and various ACC stimulation patterns. Preliminary findings suggest that GSK3β inhibition (AR-A014418; i.p. injection, 30 μmol/kg) enhances the slope of evoked local field potentials in the NAcc resulting from BLA stimulation. While comparisons between ACC stimulation

patterns have thus far produced no significant results on BLA-NAcc interactions, early results suggests that train stimulation of ACC may reduce the probability and latency variability of BLA-stimulated evoked single-unit response in NAcc. These data provide promising evidence that GSK3β expression can augment BLA-NAcc projections, which may play a role in promoting affective behaviors mediated by BLA-NAcc interactions.

**E5 UG-A**
**A CIRCADIAN OUTPUT CENTER CONTROLLING FEEDING RHYTHMS IN DROSOPHILA**

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The circadian system produces ~24-hr rhythms in physiology and behavior and can be divided into three major components: a core molecular clock in the brain, input pathways that synchronize the molecular clock to environmental cycles, and output pathways that couple the clock to overt physiological and behavioral outputs. Most circadian studies in *Drosophila* have been conducted with respect to locomotor behavior, and little is known about circadian control of other outputs such as feeding behavior. We recently showed that specific populations of cells within the *Drosophila* pars intercerebralis (PI) function as part of the output pathway controlling locomotor activity rhythms. Here we examine the role of cell populations within the PI in the modulation of circadian feeding behavior using a newly-developed automated feeding monitoring system. Specifically, we investigate *Drosophila* insulin-like peptide (Dilp)-expressing cells and SIFamide (SIFa)-expressing cells, both of which are synaptically connected to the central clock and are known to be involved in the control of feeding behavior and metabolism. We find that genetically-induced constitutive activation or inhibition of Dilp-expressing cells has no effect on feeding rhythms. In contrast, constitutive activation and inhibition of SIFa-expressing cells result in decreased strength of feeding rhythms and increased overall duration of feeding events. Similar phenotypes are observed in mutant flies lacking SIFa peptide. These results in conjunction with previous findings suggest that the PI serves as a key integrator of circadian signals and that circadian control of different behaviors relies on different output circuits that access molecularly-distinct PI output populations.

**E6**
**C2-DOMAIN PHOSPHORYLATED PROTEIN (C2CD5) INVOLVED IN HYPOTHALAMIC PROTEIN TRAFFICKING AND ENERGY BALANCE**

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Obesity is arguably the most crucial health concern, as 34% of the population in the United States is now obese. Melanocortin receptor 4 (MC4R), contributes to appetite control in hypothalamic neurons and is a promising target for anti-obesity treatments or drug development, but is hindered by gaps in our understanding of its regulation. MC4R trafficking (to plasma membrane and/or endocytosis) has been shown to be key to regulation of energy balance, and is altered in obesity and in presence of lipids, but the cellular and molecular mechanisms of altered trafficking are largely unknown.

Our studies identified a novel C2-domain phosphorylated protein of 138kDa (C2CD5) that appears to contribute to the regulation of MC4R trafficking. We found that 1) the expression of C2CD5 is altered by changes in diet, 2) baseline expression of C2CD5 is lower in genetically obese and high-fat fed mice compared to controls, 3) C2CD5 colocalize and interacts with MC4R complex, and, 4) C2CD5 knock-out mice exhibit pronounced obesity due to an increase in food intake compared to control mice when fed a high-fat diet.

Based on these, we hypothesize that C2CD5 could be a trafficking protein involved in central regulation of energy balance by modulating hypothalamic receptors via nutritional status. These studies provide evidence for a novel pathway and targets, to develop therapeutic drugs aimed at efficiently deceasing body weight in obese patients.

**E7 UG-A**
**IDENTIFICATION OF CIRCADIAN OUTPUT GENES THAT AFFECT REST:ACTIVITY RHYTHMS IN DROSOPHILA**

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Most organisms have endogenous circadian clocks that produce behavioral and physiological rhythms. The circadian system has three main parts: input pathways, the core clock, and output pathways. The core clock in the fruit fly, *Drosophila melanogaster*, is comprised of several populations of neurons in the brain that contain a molecular clock. Input pathways relay information about environmental cues, such as light, to the cells of the core clock. These cells then communicate to output pathways, which mediate behavioral rhythms. Output pathways are the least understood component of the circadian system. We recently identified the *Drosophila* pars intercerebralis (PI) as a major circadian output center that lies downstream of the core clock cells, and have used single-cell RNA sequencing to identify neuronal signaling genes expressed by PI cells. Here we determined the contribution of these genes to behavioral rhythms by monitoring locomotor activity following cell-specific RNA interference (RNAi) to knock down gene expression within the PI. We have identified several genes whose expression in the PI is necessary for normal locomotor activity rhythms. These include the calcium-activated voltage-gated potassium ion channel *slowpoke* and the *GluRIIE* glutamate receptor, and we hypothesize that these genes have a role in setting neuronal activity levels of PI output cells. Intriguingly, a subset of core clock neurons

releases glutamate and we have previously shown that these clock neurons synapse directly onto PI output cells. Thus the GluRIIE receptor likely underlies the ability of PI neurons to respond to clock cell outputs. Our experiments have identified novel circadian output genes and provide insight into the manner through which circadian output cells receive and transmit circadian information.

**E8 UG-B**

**EFFECTS OF PERINATAL POLYCHLORINATED BIPHENYLS (PCBS) ON DOPAMINERGIC AND INFLAMMATORY GENE EXPRESSION IN THE ADOLESCENT HYPOTHALAMUS AND PREFRONTAL CORTEX IN RATS.**

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Polychlorinated biphenyls (PCBs) are endocrine disrupting compounds present throughout the environment. Their effects on mesocorticolimbic (MCL) dopamine cells are well-studied, but effects on hypothalamic dopaminergic cell populations are unknown. PCBs are also known to alter immune responses in tissues like blood and spleen, but the effects are not well described in the brain. The relationship between neuroinflammation and neuroendocrine systems is complex and bidirectional. If PCBs induce inflammatory responses in the brain, inflammatory signaling may be a mechanism for both dopaminergic and endocrine effects of PCBs. To test this hypothesis, pregnant Sprague-Dawley rats were fed a PCB mixture chosen to represent human exposures (20ug/kg, 1:1:1 Aroclor 1242, 1248 and 1254 on wafers) or vehicle daily throughout gestation. Male and female offspring were injected with an immune challenge (lipopolysaccharide, LPS, 0.05mg/kg, ip) 2-4 hours prior to euthanasia at postnatal day (P) P42 or P84 and brain tissues were collected. RNA was isolated from gross dissections of the whole hypothalamus or prefrontal cortex (PFC), transformed to cDNA, and used in rt-qPCR to quantify expression of receptors for dopamine (*Drd1a, Drd2*) and LPS (*Tlr4*). Differences between groups in relative expression of these genes were determined with a two-way ANOVA (PCB treatment x LPS treatment) within a sex and brain region. In the adolescent PFC, there was a significant interaction between PCB and LPS exposure on expression of *Tlr4*: LPS exposure reduced expression of *Tlr4* only in PCB-exposed males. In contrast, in adolescent hypothalamus, PCB exposure caused greater expression of dopamine receptors, independent of LPS treatment, and no effect on *Tlr4* expression. Of note is that these adolescent effects were found in males, but not females. In adult hypothalamus, interactions between PCBs and LPS were observed on both *Tlr4* expression in females, and *Drd1a* expression in males: the normal response to LPS was absent in animals that had been exposed to PCBs. As expected with hormone sensitive systems, sex- and age- specific effects were observed. Overall, these effects reveal PCB-induced alterations in hypothalamic dopamine receptor expression and a mechanism by which PCBs might alter responses to inflammatory molecules that typically bind *Tlr4*. Continued work is investigating potential PCB effects on microglial activation, the resident immune cells of the brain. As neuroinflammation has been implicated in depressive, addictive, and neurodegenerative diseases, these findings highlight another mechanism by which PCB exposure may have long lasting effects on health.

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## *THEME F. NEURONAL EXCITABILITY, SYNAPSES AND GLIA*

**F1 PD-B**

**GENOME-WIDE TRANSCRIPTIONAL CHANGES IN THE RAT HIPPOCAMPUS DURING WITHDRAWAL FROM CHRONIC ALCOHOL DRINKING**

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Withdrawal from chronic alcohol drinking causes depression and anxiety that may promote relapse to problematic alcohol use. The hippocampus is an especially alcohol-sensitive region of the brain that is involved in depression. In order to discover genes that may regulate depression-like behavior during alcohol withdrawal, we performed a whole-genome analysis of transcriptional changes occurring in the rat hippocampus during withdrawal from chronic alcohol drinking using RNA sequencing (RNA-Seq) and performed qPCR validation on a subset of these genes. We also examined whether altering histone acetylation at the promoters of these genes by treating with the histone deacetylase inhibitor SAHA would reverse the changes occurring during ethanol withdrawal. Analysis of the RNA-Seq data identified 354 genes in the hippocampus that were significantly altered in the withdrawal vs. control condition, out of 18,118 genes identified. WGCNA analysis of all 18,118 genes demonstrated that the genes fit into 53 co-expression modules. Genes in module 1 showed significantly higher expression during withdrawal compared with control conditions. We selected 6 genes from this module (Tnfrsf1a, Stat3, Relb, Plat, Serpine1, and Timp1) for further validation by qPCR. All of these genes except Plat were increased during withdrawal, similar to what was observed in the RNA-Seq experiment. In addition, expression of Tnfrsf1a, Stat3, Relb, Serpine1, and Timp1 were normalized to control levels by SAHA treatment. These results demonstrate that transcriptional changes occur in the hippocampus during withdrawal from chronic ethanol exposure. Some of these changes can be reversed by treatment with the histone deacetylase inhibitor SAHA. Reversal of the gene expression changes by SAHA suggests that these genes might be regulated by histone acetylation during withdrawal. Future experiments will examine the epigenetic changes that cause increased expression of Tnfsfr1a,

Stat3, Relb, Serpine1, and Timp1, and whether these genes regulate depression-like behavior during withdrawal from alcohol.

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**F2 G-A**
**REGULATION OF NMDAR TRAFFICKING BY PROTEIN PHOSPHATASE 1**

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N-methyl-D-aspartate receptors (NMDARs) are glutamate receptors that are responsible for the molecular basis of learning and memory through their ability to control processes such as synaptic plasticity and synaptic maturation. NMDARs are able to achieve these phenomena through their ability to initiate specific intracellular cascades in response to receptor activation. A major determinant of which intracellular cascades are triggered upon NMDAR activation is receptor localization. Receptors that are located within the synapse engage pathways that are important for cell survival and maintenance. On the other hand, overactivation of the extrasynaptic NMDARs has been linked to initiation of cell death pathways. Thus, a key aspect of NMDAR regulation and function is tight control over trafficking of the receptor.

Posttranslational modifications are a crucial component of receptor trafficking. We have previously investigated how phosphorylation within the PDZ-binding domain of the GluN2B subunit of NMDARs results in the disruption of NMDARs from scaffolding proteins to reduce the synaptic content of the receptor. More recently, we sought to understand how the complementary reaction, dephosphorylation of this site, is controlled. Using primary cortical neurons and acute cortical slices, we have identified that calcium influx into the cell following activation of both synaptic and extrasynaptic GluN2B-containing NMDARs triggers the dephosphorylation of surface-expressed GluN2B-containing NMDARs.

In an effort to better understand the mediators of this dephosphorylation, we screened protein phosphatases and have identified protein phosphatase 1 (PP1) as the phosphatase responsible for dephosphorylating the GluN2B PDZ-binding domain. However, because PP1 is a constitutively active phosphatase, there exist a number of molecular modes of regulation to control its activity. These include inhibitory PP1 phosphorylation, association with endogenous inhibitor proteins, and subcellular substrate targeting. Because canonical PP1 regulation involves a mixture of these forms of regulation, we are examining how each of these mechanisms may play a role in regulating PP1 activity against GluN2B's PDZ-binding domain. Our data suggests a model in which locally inhibited PP1 becomes activated following calcium influx due to activation of the synaptic and extrasynaptic populations of NMDARs. Ultimately, we believe that understanding how PP1 regulates this posttranslational modification and its consequence on NMDAR trafficking and localization will help to understand how physiological NMDAR trafficking occurs and how this is altered in disease.

**F3 PD-A**
**UPTAKE AND ANTI-INFLAMMATORY EFFECTS OF MOLECULAR FORMS OF DOCOSAHEXAENOIC ACID IN MICROGLIA**

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Docosahexaenoic acid (DHA) is a potent neuroprotective compound that plays a critical role in brain function, and its deficiency is known to contribute to neurological disease such as Alzheimer's disease (AD). Since the brain cannot synthesize DHA, it needs to import it through the blood brain barrier (BBB). However, there is a controversy with regard to the molecular form of DHA transported into the brain. Recent studies demonstrated the presence of a transporter at the BBB that specifically transports DHA into the brain in the form of lysophosphatidylcholine (LPC-DHA). Other studies reported that the free DHA is the preferred form of DHA that enters the brain. The aim of this study is to determine the uptake and anti-inflammatory effects of various molecular forms of DHA and the generation of neuroprotectins by LPS-treated microglia. BV2 microglial cells were incubated with free DHA or LPC DHA for 24 h, and the DHA content of the cells, as well as the molecular species DHA-containing lipids were analyzed by GC/MS and LC/MS/MS respectively. The uptake of DHA by BV2 glial cells was 2-fold higher with LPC-DHA, compared to free DHA. The incorporation pattern of DHA into molecular species of PC, PE and PS in BV2 glial cells was also different between two forms of DHA. Cells incubated with LPC DHA showed a significant increase in DHA PC and DHA PS molecular species (2-fold), compared to free DHA treated cells. The increase in DHA PE molecular species was 3-fold greater than the increase in DHA PC species after incubation with LPC DHA. Both sn-1 acyl and sn-2 acyl isomers of LPC DHA inhibited the synthesis of pro-inflammatory eicosanoids in LPS treated BV2 microglia. LPC DHA reduced the levels of pro-inflammatory cytokines IL-1 (-40%) and IL-6 (-55%) in LPS treated BV2 microglial cells, compared to free DHA and control. In addition, the concentrations of neuroprotectins NPD1 (+45%), RVD1 (+50%), RVD2 (+47%) and Mar1 (+54%) were significantly increased in LPS treated BV2 microglial cells as compared to free DHA and control. These results show that LPC DHA is superior to free DHA in the attenuation of microglial activation, and in providing neuroprotection.

**F4 UG-A**

**ATP-INDUCED EXTRACELLULAR ACIDIFICATION FROM RETINAL MÜLLER GLIA IS SENSITIVE TO EXTRACELLULAR CONCENTRATIONS OF SODIUM AND POTASSIUM.**

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In recent years, significant interest has developed in the role glia play in actively regulating neuronal signaling in the central nervous system. This study characterizes a molecular pathway by which a type of glial cell in the retina, known as a Müller cell, causes a robust extracellular acidification when exposed to ATP, which is likely to be co-released with glutamate at neuronal synaptic terminals. Alterations in extracellular pH have been shown to regulate synaptic transmission in the retina, and we hypothesize that the pH modulation by Müller cells is a key regulator of visual signals in the retina. Prior studies of this regulatory pathway implicated activation of a P2Y pathway relying on elevation of intracellular calcium to induce the glial cell mediated extracellular acidification. This study, which utilizes a novel $H^+$-selective self-referencing technique to measure proton fluxes from isolated tiger salamander Müller cells, analyzes the dependency of this ATP-induced pathway on the presence of the extracellular ions $Na^+$ and $K^+$. Self-referencing studies involving the removal of extracellular $Na^+$ demonstrated a significant decrease in the ATP-induced acidification. In addition, the sodium transport blocker amiloride and the sodium-hydrogen exchanger blocker cariporide each reduced the effect in an analogous manner to the removal of extracellular $Na^+$. Together, these results strongly imply a role of sodium-dependent mechanisms, and the sodium-hydrogen exchanger, as a proton carrier in the ATP-evoked pathway. The ATP-induced acidification was also sensitive to extracellular $K^+$ concentrations. The reintroduction of extracellular $K^+$ from a nominally 0mM $K^+$ Ringer's solution resulted in a large potentiation of the ATP-induced acidification. This $K^+$ sensitivity points toward a positive feedback mechanism through which increased extracellular $K^+$ levels, likely correlated with increased neuronal activity, could further enhance the glial cell mediated ATP-evoked proton efflux and act to attenuate overactivity of retinal neurons. Collectively, our results indicate that the proton efflux activated by ATP likely involves multiple carriers with $Na^+$ and $K^+$ dependencies. This characterization of ionic dependencies of the ATP-evoked extracellular acidification sheds further light on an important mechanism through which glia shape visual signals in the retina.

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**F5 PD-B**

**INPUT-SPECIFIC MODULATION OF PREFRONTAL AFFERENT DRIVE BY α7nAChR SIGNALING IN VIVO**

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Abnormal elevation of the tryptophan metabolite kynurenic acid in the prefrontal cortex (PFC) is thought to contribute to the development of cognitive deficits in schizophrenia and related psychiatric syndromes. At the synaptic level, we have recently determined that increasing kynurenic acid levels in the PFC is sufficient to disrupt local processing of ventral hippocampal drive through an α7nAChR-dependent mechanism. The aim of the present study is to further investigate the impact of α7nAChR signaling in the regulation of PFC processing of afferent drive and determine to what extent such a modulation is input-specific. We found that the characteristic high-frequency stimulation-induced facilitation of basolateral amygdalar-evoked local field potential (LFP) responses in the PFC is markedly attenuated following prefrontal infusion of the α7nAChRs antagonist MLA. However, this inhibitory effect by MLA is no apparent when a protocol of LFP facilitation was elicited from the ventral hippocampus. Instead, prefrontal administration of MLA markedly diminished the inhibitory component of the hippocampal drive such that a shift from LFP suppression to LFP facilitation emerges in the PFC. Accordingly, the normal inhibitory regulation of amygdalar-induced LFP facilitation by the ventral hippocampus is no longer observed following PFC infusion of MLA. This shows that blockade of α7nAChR signaling limits the influence of ventral hippocampal-mediated inhibitory drive and its control over basolateral amygdalar transmission to the PFC. Collectively, these results indicate that α7nAChR signaling in the PFC differentially regulates hippocampal and amygdalar afferent information and their interactions in an input-specific manner.

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**F6**

**METAPLASTICITY IN THE MOUSE MODEL OF FRAGILE X SYNDROME**

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Fragile X syndrome (FXS) is a neurodevelopmental disorder that results in numerous neurological problems including intellectual disability and autism spectrum disorder (ASD). FXS is primarily caused by the expansion of a trinucleotide CGG repeat in the 5' UTR of the FMR1 gene leading to the silencing and loss of expression of the Fragile X mental retardation protein (FMRP). Synaptic plasticity has been studied extensively in the mouse model of FXS (Fmr1 KO). There is evidence indicating that long-term potentiation (LTP), the molecular basis of learning and memory is still present, but the threshold for LTP induction

is elevated in the hippocampus in Fmr1 KO mice. Metaplasticity, the "plasticity of synaptic plasticity", sets the state dependence of synapses and primes synapses to be either more or less likely to undergo LTP. Therefore metaplasticity is known to set the threshold for synaptic plasticity however, it is not known if this is disrupted in Fmr1 KO mice and little is known about the intracellular signaling pathways that underlie priming induced metaplasticity. We have found that priming of synapses with low frequency activation suppresses subsequent induction of LTP in the CA1 region of the hippocampus. This NMDA receptor dependent metaplasticity is enhanced in Fmr1 KO mice. We hypothesize that priming changes the excitability of dendrites by increasing trafficking of a potassium channel and that this mechanism is altered in Fmr1 KO mice. We tested this hypothesis by measuring several synaptic parameters before and after priming. There was no significant difference in the relative NMDA receptor component of transmission in Fmr1 mice. However, enhanced metaplasticity in Fmr1 KO mice was restored by treatment with heteropodatoxin, an inhibitor of the voltage gated potassium channel, Kv4.2. Therefore, our results indicate that 1) NMDA receptor mediated metaplasticity is mediated in part by elevated Kv4.2 in CA1 neurons 2) this pathway is enhanced in Fmr1 KO mice. This study provides novel insight into the mechanism that underlie changes in the threshold for synaptic plasticity in FXS and may provide potential therapeutic targets for this devastating disorder.

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**F7 G-B**

**DEFECTS IN LYSOSOME-AUTOPHAGOSOME REGULATION AND SYNAPTIC VESICLES IN ALZHEIMER'S DISEASE MOUSE MODELS AND HUMAN INDUCED NEURONS**

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Vacuolar H+ ATPase (V-ATPase) is a conserved proton pump that regulates the acidic environment necessary for intracellular organelle function, such as lysosomes and synaptic vesicles. The V-ATPase maintains an acidic lysosomal pH needed for the catabolic autophagosome lysosome pathway to degrade cellular proteins. Synaptic vesicles require an acidic environment for neurotransmitter uptake and synthesis. In Alzheimer's disease (AD), V-ATPase disruption can lead to abnormal β-amyloid and tau accumulation, and deficient synaptic vesicle store. Our hypothesis is that altered composition of V-ATPase influences lysosomal and synaptic vesicle functionality thereby contributing to pathogenic protein aggregation and disrupted synaptic transmission in AD.

Using immunohistochemistry, this study reveals that V-ATPase (V1B2, V0a1), lysosome (Lamp1), and pre-synaptic vesicle (Synaptophysin) markers are diminished in hippocampus and cortex of 3-month old AD mice models (3xTg) relative to non-transgenic (NTg) controls, whereas mature autophagosome (LC3B) expression is increased. These phenotypes were restored to NTg levels after a 30-day Ryanodex (NAM of ryanodine receptor (RyR); 10mg/kg) treatment in 3xTg-AD mice. These findings suggest that prior to overt histopathology or cognitive deficits, decreased V-ATPase expression on acidic compartments and increased autophagosomes in 3xTg-AD mice reflect impaired lysosomal and synaptic vesicle functionality, which is mediated through upstream RyR-$Ca^{2+}$ dyshomeostatis.

Additionally, lysosomal pH upon RyR stimulation with caffeine (20mM) was measured in RyR-overexpressing HEK cells and induced human neurons (iN) using Lysosensor DND-160. This shows that increased aberrant RyR-$Ca^{2+}$ signaling alkalizes lysosomes. Upon 500nM bafilomycin (V-ATPase inhibitor) treatment, iN shows increased levels of β-amyloid and hyperphosphorylated tau. Therefore, increased RyR-mediated $Ca^{2+}$ release alkalizes lysosomal pH resulting in abnormal protein aggregates, thereby demonstrating that aberrant intracellular $Ca^{2+}$ signaling in early AD influences synaptic transmission and accumulation of aberrant proteins in part through altering functionality of critical organelles.

**F8 G-A**

**A COMPUTATIONAL MODEL OF TWO-PHOTON CALCIUM IMAGING IN SPINES AND DENDRITES OF CA1 PYRAMIDAL NEURONS USING A GENETICALLY ENCODED CALCIUM INDICATOR**

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Fluorescent calcium indicators are commonly used to image neuronal activity, as changes in calcium concentration accompany voltage-mediated events such as action potentials and dendritic spikes. The GCaMP family of calcium sensors has opened the door for genetically targeted, high resolution neuronal activity to be recorded in awake, behaving animals in vivo. However, interpreting the calcium signal to infer more specific details about the underlying activity remains a challenge.

We present a morphologically detailed computational model of the CA1 pyramidal cell, featuring calcium buffering dynamics and simulated GCaMP6 fluorescence throughout the dendrites and spines. The GCaMP construct combines green fluorescent protein (GFP) and calmodulin (CaM). We therefore model the calcium-binding kinetics of GCaMP based on those of CaM (Faas 2011). Additionally, our model incorporates sources of calcium influx into the cytosol such as voltage-gated calcium channels (R-type and T-type), AMPA (Spruston 1995, Beaulieu-Laroche 2018) and NMDA receptors (Stern 1992, Tao 2018), as well as endogenous buffers and transmembrane pumps that extrude calcium from the cell (Müller 2005).

We verify that the model reproduces a number of reported results from two-photon imaging experiments of CA1 pyramidal neuron

dendrites and spines. In particular, the somatic, dendritic and spine compartments exhibit appropriate levels (both absolute and relative) of calcium concentration changes and indicator fluorescence in response to simulated synaptic stimulation. A unitary event, with a depolarization of 18mV in the spinehead and .3mV in the soma (Harnett 2012, Magee 2000), evokes a fluorescence level that is sub-visible (dF/F < 10%), as expected. We also reproduce fluorescence changes between 40-400% in dendrite and spine for dendritic spikes and action potentials, with a larger fluorescence increase in the spinehead in the case of coincident synaptic input (Sheffield 2015, Sheffield 2017).

We also discuss how the model can be used to inform our understanding regarding the mechanisms associated with dendritic integration by providing a more detailed interpretation of calcium imaging experiments. In particular, it provides a way to test the causal mechanisms giving rise to observed calcium signals such as sodium-mediated depolarizations, regenerative calcium-mediated events and NMDA spikes.

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**F9 G-B**

**NEURINFLAMMATORY CONTRIBUTION OF ASTROGLIAL CELLS IN A BINGE ETHANOL IN VITRO MODEL**

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Poly [ADP-Ribose] Polymerase-1 (PARP-1) is a DNA repair enzyme that has been implicated in parthanatos, a regulated necrotic form of cell death. This lab previously reported that PARP-1 is upregulated in binge ethanol-treated adult rat hippocampal entorhinal cortical (HEC) organotypic slice cultures. The data has been supported *in vivo* using a PARP 1 inhibitor. Moreover, $Ca^{2+}$ dependent phospholipase A2 (cPLA2) is an enzyme that cleaves fatty acids from the cell membrane at the *sn*-2 position – this includes the pro-inflammatory omega-6 fatty acid, arachidonic acid. HEC slice cultures show increased protein expression of cPLA2 after 3 days of binge ethanol treatment. Analysis of mRNA revealed an increase in cPLA2 mRNA after 2 days of binge ethanol-treatment in HEC slices.

However, HEC slices contain multiple cell types (i.e neurons, glia, microglia, etc.). The purpose of this study was to identify the cellular origin of PARP-1 and cPLA2 upregulation following binge ethanol-treatment. To this end, we used cell type specific cell lines as *in vitro* models for ethanol treatment. Exposure to 100mM ethanol for a period of 4 days (16 hour treatment / 8 hours withdrawal) resulted in statistically significant upregulation of PARP-1 expression, in cells of neuronal origin (HN2-5 cells) as well as C6 astroglioma cells. Our data are in accordance with *in vivo* studies showing neuron-specific upregulation of PARP-1 in response to ethanol exposure. Furthermore, cPLA2 protein increases only in c6 astroglioma cells and not HN2-5 cells. This suggests glial cells play a key contribution to neuroinflammation associated with binge ethanol exposure.

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**F10 G-A**

**MODELING THE DEVELOPMENT AND BINOCULAR MATCHING OF ORIENTATION SELECTIVITY IN THE MOUSE VISUAL CORTEX**

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In mouse visual cortex, at eye opening binocular cells have different preferred orientations for input from the left and from the right eye. With normal visual experience during a critical period (P19-P30), these orientations shift and eventually become well matched. Recent experiments have shown that the binocular matching process is completely blocked by monocular deprivation spanning the entire critical period; however, environmental enrichment can rescue the matching to the level seen in normal mice. To gain insight into the matching process, we employed a computational model of a cortical cell that receives oriented inputs from the two eyes via synapses that exhibit spike-timing dependent plasticity. The model captures the experimentally observed matching of the orientation preferences, the dependence of the matching process on the ocular dominance of the cell, and the inverse relationship between the resulting binocular orientation selectivity and the mismatch. Moreover, our model puts forward testable predictions: i) the binocular orientation preference at the onset of the matching period is predictive of the final matched orientation preference; ii) the matching speed increases with the initial ocular dominance, suggesting ocular dominance as a key driver of the binocular matching process; iii) matching proceeds faster than the sharpening of the orientation selectivity, suggesting that orientation selectivity is not a driving force for the matching process; iv) there are two dominant routes for matching: the monocular preferred orientations either drift towards each other or one of the orientations switches quite suddenly. The former occurs for a small initial mismatch, while the latter is specific for a large mismatch. We expect that these results provide insight more generally into the development of systems that integrate inputs from multiple sources in order to generate normal neuronal functions.

**F11 PD-A**

**RAPID THREE-DIMENSIONAL SUPER-RESOLUTION IMAGING OF DENDRITIC SPINULE DYNAMICS IN CORTICAL MOUSE NEURONS**

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Dendritic spinules are thin, transient membranous protrusions that originate from neuronal dendritic spines and can project into invaginations of adjacent axon terminal or glial cell membranes. Current literature indicates that dendritic spinules are induced by synaptic transmission, and their proposed functions include synaptic plasticity, retrograde signaling, and membrane recycling. Their nano-scale necessitates resolution beyond the light diffraction limit, and hence our knowledge of spinule structure and function has been derived primarily from static serial section electron microscopy. Recently developed super-resolution light microscopy techniques, e.g. structured illumination microscopy (SIM), have theoretically enabled live nano-scale imaging, but their practical application for studying rapid biological processes has so far been limited. New improvements in SIM, though not yet widely available, have overcome slow acquisition speed, which is the main barrier to time-lapse imaging with this technique. Herein, we utilized rapid 3D SIM to track individual dendritic spinule lifespans and dynamics in cultured mouse pyramidal cortical neurons expressing a red cell-filling fluorescent protein. Spinules were most frequently detected in large mushroom spines and they typically recurred at the same location on the spine head. The majority of spinules were short-lived and existed for <60 seconds, while a smaller subset was long-lived, existed for ≥60 seconds, and occasionally exceeded the 1000 second duration of imaging. Long-lived spinules were substantially greater in length and less dynamic than short-lived spinules, and they displayed unique morphologies. We also investigated the role of $Ca^{2+}$ in spinule formation by treating neurons expressing the ultra-sensitive $Ca^{2+}$ indicator, GCAMP6-GFP, with the cytosolic $Ca^{2+}$ chelator, BAPTA-AM, followed by fast enhanced-resolution A1 confocal microscopy. BAPTA-AM-treated neurons displayed a substantially decreased number of spinules per spine compared to negative controls. There was also a striking positive correlation between mean and maximum $Ca^{2+}$ peak intensity in the spine head and number of short-lived, but not long-lived spinules per spine in the negative controls. These data indicate that short-lived spinules are highly responsive to local changes in activity, while long-lived spinules are resistant, hinting at differential synaptic functions. Our study demonstrates the utility of rapid 3D enhanced-resolution imaging for the study of dendritic spinules and reveals distinctive spinule classes, which differ in their lifespan, dynamics, morphology, and regulation.

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## *THEME G. NOVEL METHODS AND TECHNOLOGY DEVELOPMENT*

**G1**

**MUSCLE-NERVE-NERVE GRAFTING IMPROVES FACIAL REANIMATION IN RATS FOLLOWING FACIAL NERVE INJURY**

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Loss of facial nerve innervation can be devastating for otolaryngology patients. Primary neurorrhaphy and interposition (IP) nerve grafting are common methods for reinnervation, but rarely if ever restore full function. We have previously demonstrated comparable return of facial function in rats using a lesser known alternative, muscle-nerve-muscle (MNM) grafting, which involves implanting an autogenous nerve graft as a conduit between an innervated "donor" muscle and a denervated "recipient" muscle. In this study, we investigated a variation of this technique, muscle-nerve-nerve (MNN) grafting, where instead of implanting the graft into the denervated "recipient" muscle, it is implanted at the nerve. We sought to determine the effect of MNN neurotization relative to MNM and IP grafting, on functional recovery following rat facial nerve injury. Our work used thirty male, Sprague-Dawley rats, assigned to 4 groups: no graft (negative control), IP graft (positive control), MNM graft, and MNN grafts. Harvested right buccal and mandibular nerve branches were used as nerve grafts. We assessed functional recovery with behavioral observations of vibrissae movement and orientation, and muscle reinnervation with weights of vibrissae muscle pads. The MNN group demonstrated statistically significant improvement in vibrissae movement as compared to controls, similar to that of IP grafting ($p<0.01$). MNN grafting also demonstrated significantly reduced days to forward vibrissae movement (60 days) compared with controls (91 days) ($p<0.01$) and significant improvement in vibrissae orientation. Fifty percent of animals in the MNN group demonstrated forward, coordinated vibrissae movement at 16 weeks, which was superior to IP grafting (37.5%), MNM grafting (12.5%), and control (0%). The MNN group had a significant increase in muscle pad weight (70.9±2.78 g) at 16 weeks compared to controls (55.9±0.63 g), which was similar to IP grafting (72.7±4.4 g). This is the first study to evaluate the efficacy of MNN neurotization for facial nerve injury, and showed to be a viable technique for repair of unilateral peripheral nerve paralysis. For patients with unilateral peripheral nerve injury, particularly those who are not candidates for primary neurorrhaphy, this study provides a promising new technique for the return of function.

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**G2**

## PRE-IMPLANT MODELING FOR DEPTH LEAD PLACEMENT IN WHITE MATTER FOR MAXIMIZING DIRECT NEUROSTIMULATION THERAPY

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A critical step towards optimizing direct neuromodulation of refractory focal-onset epilepsy is to effectively interface with an epileptogenic circuit with two or more communicating epileptogenic sources using a maximum of two 4-contact depth electrode leads. Our objective was to predict, preoperatively, the maximum extent to which responsive neurostimulation therapy (RNS, NeuroPace) can propagate through an epileptic brain circuit for stabilizing the epileptogenic network by placing virtual electrodes at the grey-white matter interface. A classical approach to determine the volume of brain activation is to calculate the electric field (E-field) immediately surrounding the stimulating electrode and select a "magnitude threshold" above which activation occurs. While this model gives information on possible activated tracts when placed in white matter (WM), it fails to account for stimulation effects on the axonal membrane potential. However, an understanding of the membrane biophysics is crucial for predicting activation in axon bundles adjacent to the electrode. Our model can differentiate between regions of depolarization and hyperpolarization produced by the applied stimulus by computing an activation function (AF), derived from the core-conductor model which considers three factors: 1) electric potential (EP), 2) directionality of the E-field, and 3) axon bundle orientation. The model was generated for five RNS patients with refractory focal-onset epilepsy implanted at our institution. The AF was computed for each patient and then compared with the classical E-field model. Validation of both models was addressed post-operatively by performing a stimulation activated SPECT (SAS). This technique captured transient blood flow changes during delivery of focused cortical RNS using a relatively high therapeutic charge density without generating an after-discharge.

For the five patients, the AF model generated irregular volumes of activation surrounding the depth contacts due to hyperpolarization and depolarization, these regions were used as seeds for creating modulated circuit tractography (MCT) maps for each model. In contrast, the E-field model showed spherical shaped regions. The MCT activated by the AF predicted the extent to which WM-connected epileptic sources were influenced during RNS. MCT was validated by SAS (performed in 2 of the 5 patients) and RNS electrocorticography.

The preimplant AF-based model offers the potential to predict optimal implant sites for two 4-contact depth leads influencing up to 3 distant communicating epileptogenic sources in an epileptogenic network. As importantly, the model provides an ability to identify regions of depolarization and hyperpolarization.

**G3 G-B**

## PROTEASOME-TARGETED NANOBODIES ALLEVIATE PATHOLOGY IN A SYNUCLEIN-BASED PARKINSON'S DISEASE MODEL

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Parkinson's disease (PD) is a synucleinopathy with a significant loss of dopaminergic neurons in the substantia nigra (SN) and abrogation of dopaminergic tone along the nigrostriatal pathway. Therapeutics designed to target α-synuclein (α-syn) aggregation may be critical in halting the progression of pathology in PD patients. Nanobodies are single-domain antibody fragments that can be expressed intracellularly and specifically bind to target regions critical for protein accumulation. Nanobody fusion with a proteasome-targeting PEST motif can modulate monomeric concentrations of aggregate proteins while maintaining aptamer stability. Here we aimed to validate and compare the in vivo therapeutic potential of gene therapy delivery of two proteasome-directed nanobodies selectively targeting α-syn in a synuclein-overexpression based PD model: VH14*PEST (non-amyloid component region) and NbSyn87*PEST (C-terminal region). Stereotaxic injections of AAV5-α-syn into the SN were performed on Sprague-Dawley rats that were sorted into three cohorts based on pre-operative behavioral testing (cylinder test and stepping test). Rats were treated with unilateral SN injections of vectors for VH14*PEST, NbSyn87*PEST, or injected with saline 3-weeks post-lesion. Post-mortem assessments of the SN showed both nanobodies markedly reduced the level of phosphorylated-Serine129 α-syn labeling relative to saline treated animals. VH14*PEST showed considerable maintenance of striatal dopaminergic tone in comparison to saline- and NbSyn87*PEST-treated animals as measured by Tyrosine Hydroxylase immunoreactivity (optical density), dopamine transporter immunoreactivity (optical density), and dopamine concentration (HPLC). Microglial accumulation and inflammatory response, assessed by stereological density of Iba-1-labeled cells, was modestly increased in NbSyn87*PEST-injected rats but not in VH14*PEST- or saline-treated animals. Both nanobody constructs significantly improved stepping test performance, with NbSyn87*PEST-treated group also showing improvement in cylinder test compared to the saline-treated group, although there was pronounced variability amongst individual animals. These data show novel in vivo therapeutic efficacy of vector-delivered intracellular nanobodies targeting α-syn misfolding and aggregation in synucleinopathies such as PD.

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**G4**

## PARAMETRIC SUBTRACTED POST-ICTAL DIFFUSION TENSOR IMAGING FOR GUIDING DIRECT NEUROSTIMULATION

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The correct identification of anatomic targets for responsive neurostimulation therapy (RNS) depth leads represents a crucial step for interfacing with a communicating epileptogenic circuit. Diffusion Tensor Imaging (DTI) has the potential to detect transient post-ictal changes in axonal water diffusion and define critical nodes in an epileptogenic circuit. Parametric Subtracted Post-Ictal DTI (pspiDTI) is an imaging tool for detecting patient-specific ictal-related water diffusion abnormalities in white matter (WM) following stereotypic dyscognitive seizures without generalization secondarily. This technique can identify inter- and post-ictal differences between fractional anisotropy (FA) and mean diffusivity (MD) measurements. Such differences can outline ictal-associated WM propagation pathways to identify critical nodes in a potentially extensive epileptogenic network for implantation up to two RNS depth leads.

Six patients with medically intractable epilepsy, experiencing stereotypic dyscognitive seizures without generalization secondarily, who were candidates for RNS therapy were included in the study. Each patient underwent a post-ictal (up to 4hrs following seizure termination), and an inter-ictal (no electrocerebral seizures for at least 24hrs) DTI study.were pre-processed and registered to a T1 MRI. Fractional anisotropy (FA) and mean diffusivity (MD) maps were obtained. Patient-specific voxel-wise t-tests were then conducted on the FA and MD maps to identify abnormal diffusivity regions.

All patients presented statistically significant ($p < 0.01$) regions of post-ictal FA decrements. In 5/6 patients (83.33 %), each hyper-perfusion finding on ictal SPECT spatially correlated with a significant FA decrease. Three of 6 (66.67 %) also FA changes contralateral to hyper-perfusion findings. In 5/6 patients (83.33 %), statistically significant post-ictal increases in MD were observed. The clinical relevance of abnormal diffusivity regions (FA and MD) complemented ictal SPECT and electroencephalographic data. This multimodal evaluation facilitated imaging seizure propagation pathways for incorporating into a presurgical planning system and defining targets for implanting a limited set of two RNS depth leads.

PspiDTI is a novel post-processing imaging technique that facilitates localizing abnormal WM regions of transient water diffusion involved in the activation of an ictogenic network. When incorporated into a presurgical workflow, such regions can define critical nodes in a potentially extensive epileptogenic network with up to 3 epileptogenic sources for implanting a maximum of 2 RNS therapy depth leads.

**G5 UG-B**

## OPTIMIZING PREDICTION MODEL FOR A NONINVASIVE BRAIN COMPUTER INTERFACE PLATFORM USING CHANNEL SELECTION, CLASSIFICATION AND REGRESSION

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A Brain Computer Interface (BCI) platform can be utilized by a patient to control an external device without making any overt movements. An EEG-based computer cursor control task is commonly used as a testbed for BCI applications. Traditional computer cursor control schemes are based on sensorimotor rhythm. Recently, a new scheme was developed using imagined body kinematics to achieve natural cursor movement. This article attempts to explore optimal decoding algorithms using imagined body kinematics. The study is based on offline analysis of 32 healthy subjects' training data. Various machine learning techniques were implemented to evaluate the prediction model for cursor velocity compared to a user's intentions. Individual channels of EEG signals were each tested to determine the level of contribution to overall predictability. Also, a directional classifier was proposed as a potential addition to the control model. Our results showed that using a linear regression least squares model yielded the highest scores in cursor prediction (71% horizontal prediction and 40% vertical prediction using a Theil-Sen regressor). By testing each channel for individual prediction capabilities, clear patterns in relevant channels for horizontal and vertical movements were found. By using different combinations of relevant channels, we were able to increase overall prediction accuracy. Features from different frequency bands of brain activity were used in the directional classifier to a high degree of classification accuracy (80% average across subjects). The findings of the current study provide novel techniques for designing optimized BCI platforms.

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**G6 G-A**

## HUMAN INDUCED NEURONS MODEL HALLMARKS OF ALZHEIMER'S DISEASE

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Despite investing significant resources into Alzheimer's Disease(AD) research, we have yet to find a therapeutic intervention, suggesting a need for better models. Human induced neurons(HuiNs) generated from terminally differentiated AD patient fibroblasts allow us to study

the disease in live human tissue. Here we show that HuiNs model several aspects of AD, and represent an attractive model for therapeutic testing. Using fibroblasts from both AD and healthy patients, we have generated and characterized human induced pluripotent stem cells (iPSCs). Selected iPSCs were then transduced with lentiviral vectors containing neurogenin-2 (NGN2) for conversion to HuiNs. The neurons generated have been shown to express known neuronal maturation markers, and both spontaneous and evoked action potentials have been recorded. This confirms successful conversion, and suggests the HuiN make functional synapses. The power of this system lies in its ability to model AD; AD HuiNs show amyloid  42 production, tau hyper-phosphorylation, and exaggerated calcium release in response to caffeine. AD HuiNs also showed increased expression of Ryanodine Receptor 2(RYR2), and the aberrant calcium release could be mitigated by the RYR2 antagonist dantrolene. This work demonstrates that the HuiN system is a powerful tool for modeling AD. Importantly, this system can be used to test potential therapeutics, which may translate to patients more readily than animal models. In future studies, we will be exploring the contribution of pro-inflammatory cytokines and microglia to AD pathogenesis applying a co-culture system.

**G7 UG-A**
**BINOCULAR INNATE VISUAL LEARNING THROUGH SPONTANEOUS ACTIVITY PATTERNS**

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Spontaneous activity occurs in the retina prenatally and manifests amorphous, spatiotemporal patterns. Efficient coding of these spontaneous activity patterns reveals linear filters that resemble V1-like receptive fields. Such patterns have be proposed as a mechanism of preparing for vision analogously to learning soon after the eyes open, but using endogenously generated activity. Spontaneous activity is also known to occur in the lateral geniculate nucleus (LGN) through eye-specific layers with cross-talk between eye-layers provided by reciprocal connections to V1.

This work demonstrates that spontaneous activity in the LGN/V1 provides the necessary spatiotemporal patterns and subtle eye-specific differences to prepare the organism for binocular vision. This is done by generating binocular spontaneous activity patterns using an abstract, but physiologically plausible model of activity generation. Then, the patterns are efficiently to produce receptive field filters. Then these filters are used to perform a simple depth discrimination take using autostereograms, and the performance is scored based on the ability to perceive depth. The best depth perception performance occurring just below the percolation threshold of the LGN/V1 model. The spontaneous activity generated by the LGN/V1 model with those parameters closely resembles the spontaneous activity measured in vivo. This strongly indicates that spontaneous activity in the LGN/V1 prepares the visual pathway for binocular vision.

*Albert MV, Schnabel A, Field DJ (2008) Innate Visual Learning through Spontaneous Activity Patterns. PLoS Comput Biol 4(8): e1000137. doi:10.1371/ journal.pcbi.1000137*

**G8 G-B**
**IDENTIFYING SMALL MOLECULE INHIBITORS OF THE RNA EDITING ENZYME, ADAR2.**

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RNA editing is a post-transcriptional process that regulates the transmission of glutamate and serotonin receptors, in addition to modifying the replication of some retroviruses, including HIV. Abnormal RNA editing of glutamate and serotonin receptors in the brain results in physiological and behavioral deficits, including seizures, reduced social interaction and abnormal brain development. Currently, there are no FDA-approved drugs that inhibit the RNA editing process, which is catalyzed by Adenosine Deaminase acting on RNA (ADAR) enzymes. We used molecular modeling to study drug-ADAR2 interactions. Compounds that can interfere with the activity of ADAR2 will be valuable probes for studying mechanisms by which RNA editing regulates brain development. Computational molecular docking allows *in silico* exploration and study of 3-D models to identify possible binding sites, conformations, and modes between a target biomolecule and small molecules. We used Autodock software to quantify chemical interactions and binding energies of all conformations of the drug to the target binding site. Significant binding interactions were detected between the antipsychotic drug haloperidol and ADAR2. No significant binding interactions were detected between ADAR2 and the antipsychotic drug Clozapine. While we obtained these results *in silico*, we also demonstrate that treatment with haloperidol (1mg/kg) but not clozapine (5mg/kg) in mice results in reduced RNA editing of the GluA2 subunit of the AMPA glutamate receptor in the frontal cortex and hippocampus. Haloperidol treatment also reduced RNA editing in yeast cells expressing ADAR2 and a reporter construct of the GluA2 double-stranded RNA loop. Ongoing studies aim to identify molecular probes of RNA editing with greater specificity for ADAR2.

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**G9**
**DEVELOPMENT OF MOUSE DERIVED DRG EXPLANT TO INVESTIGATE HSV-1 PATHOGENESIS**

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In this study we developed mouse derived *ex vivo* dorsal root ganglia (DRG) explant model and single cell neurons (SCNs) to investigate the molecular mechanism of herpes simplex virus (HSV) entry and the associated inflammatory response in the nervous system. Using confocal microscopy, we provided a visual evidence for the expression of heparan sulfate (HS) and 3-*O*-sulfated heparan sulfate (3-*O*S HS) followed by their interactions with HSV-1 glycoprotein B (gB) and glycoprotein D (gD) during cell entry. Upon heparanase treatment of DRG derived SCN, a significant inhibition of HSV-1 entry was observed suggesting the involvement of HS role during viral entry. Finally, a cytokine array profile generated duringHSV-1 infection in DRG explant indicated an enhanced expression of chemokines (LIX, TIMP-2, and M-CSF)—known regulators of HS. Taken together, these results highlight the significance of HS during HSV-1 entry in DRG explant. Further investigation is needed to understand which isoforms of 3-*O* sulfotransferase (3-*O*ST)-generated HS contributed during HSV-1 infection and associated cell damage.

*This study was funded by Midwestern University start-up funds to VT.*

**G10**

**INTRACELLULAR UPTAKE OF CARBON NANOTUBES AUGMENTS NEURAL CONDUCTIVITY AND THE EXTENT OF ACTIVATION DURING DIRECT BRAIN STIMULATION THERAPY**

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**Rationale:** A depth electrode implanted for direct brain stimulation (dBS) therapy can only activate neurons within a 4mm radius of its surface, extending its radius by 1mm can increase the volume of tissue activation by over 95%. One method to increase the extent of brain activation is to enhance tissue conductivity (σ) and potentially reduce its impedance adjacent to the electrode. Our study demonstrates the influence of fluorescein-thiosemicarbazide-functionalized metallic carbon nanotubes (fCNTs) altering tissue impedance, their intracellular uptake, stable long-term neural localization, and biocompatibility. Four experiments were carried out as follows: electrical impedance spectroscopy to evaluate fCNT-induced changes in σ; *in vitro* fCNT cytotoxicity assay; *in situ* immunofluorescence (IF) after fCNT stereotaxic injection; and computational Hodgkin and Huxley (HH) cable modeling.

**Methods:** *Impedance Spectroscopy:* To assess changes in σ, electrical impedance spectroscopy was performed in a 0.6% agarose gel. fCNTs were injected at 5mm from the depth electrode's first contact. Impedances were measured after injection and statistically compared to controls. *Cytotoxicity Testing:* Cortical human astrocytes were treated with raw and fCNTs of 90, 95, and 99% purity. Cell viability was measured via alamar blue assay. *Cellular Uptake Test:* IF staining was performed to observe the influence of 95% pure fCNTs injected into the left hippocampal formation of F344 rats 4 weeks before sacrifice. Astrocytes (GFAP), neurons (anti-synaptophysin), and fCNTs were localized using confocal microscopy (100x). *Cable Modeling:* A computational HH impulse-propagation cable model evaluated the effect of fCNT-induced σ changes in the axonal intracellular medium. Changes in action potential production and propagation velocity were analyzed.

**Results:** Impedance spectroscopy showed a statistically significant difference between the fCNT sample and controls ($p < 0.05$). The fCNTs cytotoxicity assay demonstrated no significant difference in viability. IF staining revealed fCNT clusters' uptake by astrocytes and neurons near the injection point. The HH cable model revealed that intracellular σ changes enhance axon excitability and propagation speed.

**Conclusions:** fCNTs are biocompatible following injection into the brain, remain anchored intracellularly, and chronically modify the biophysical properties of neural cells. fCNT can impact the excitability properties of neural tissue by altering the local tissue σ and augmenting the maximal extent of cortical activation by a depth lead placed in white matter for delivering dBS therapy.

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## *THEME H. SENSORY AND MOTOR SYSTEMS*

**H1**

**ENERGY OUTPUT OF MITOCHONDRIA LOCATED NEAR SYNAPTIC RIBBONS IN INNER EAR HAIR CELLS**

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The mitochondrion plays an essential role in every cell. ATP, the primary energy source in the body, is produced by ATP synthase molecules located in the cristae of the mitochondria. In this study, we utilized EM tomography to create 3D models of mitochondrial cristae from hair cells in the vestibular nervous system. Analyzing these models provided insight into the role of these cristae in activity near ribbon synapses and synaptic vesicles. When action potentials reach nerve terminals, synaptic vesicles fuse with the membrane, releasing neurotransmitters. After this, vesicles are quickly re-internalized and refilled with neurotransmitters through endocytosis, which requires ATP (Guatimosim and von Gersdorff, 2002). In this study, we wanted to determine whether ribbon synapses and their vesicles utilized large amounts of energy from adjacent mitochondria to transmit signals. Our calculations of energy output in this mitochondrion with lamellar-shaped cristae yielded estimates of ATP production of about 1000 zeptomoles/sec and energy production at 25–30 femtojoules, consistent with other hair cell mitochondria of similar size. Using the 3D models that we constructed, we also counted the number of crista

junctions (CJs) in the locations where the crista meets the inner membrane of the mitochondria in both hair cell mitochondria and in afferent mitochondria. We observed that the ratio of CJs facing the ribbon synapse to those facing away was approximately 5:4. We concluded that there was no significant relationship between the location of the CJs and that of the ribbon synapse.

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**H2 G-A**

**UNDERLYING FACTORS CONTRIBUTING TO REACHING FUNCTION IN CHRONIC STROKE: PRELIMINARY RESULTS OF A PREDICTIVE MODEL**

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Chronic stroke survivors demonstrate a myriad of motor impairments impacting reaching function including loss of independent joint control, spasticity (i.e., hyperactive stretch reflexes), and weakness. The relative and concurrent contributions of each of these underlying factors has not yet been quantitatively explored in depth. Eight individuals (3 females, mean age = 57.88 years) with chronic stroke (1.4 to 11.53 years post-stroke) participated in the study. The Upper Extremity Fugl-Meyer Motor Assessment scores ranged from 25 to 49 indicating moderate to severe generalized impairment. Reaching function was quantified with a robotic device and defined as maximum reaching distance against gravity. A multiple regression model was implemented to investigate the constitutive elements of reaching function. The regressors included: 1) maximal shoulder abduction and elbow extension strength that were normalized to the unaffected side, 2) spasticity-related biceps activation that was measured as the increase in EMG occurring after the onset of elbow extension during reaching, and 3) loss of independent joint control (flexion synergy) that was measured as the highest abduction load at which the participant could successfully lift their arm and reach to a near and far target. Dynamic reaching was completed in a custom robotic device, the ACT$^{3D}$. Average reaching distance was 73.22 percent of maximal distance determined by limb measurements. The 5 regressors included the flexion synergy takeover threshold (0.844 ± 0.171), flexion synergy emergence threshold (0.348 ± 0.270), spasticity-related biceps activity (0.0768 ± 0.115), normalized maximum elbow extension torque (0.460 ± 0.211), and normalized maximum shoulder abduction torque (0.572 ± 0.139). While still preliminary, a significant regression equation was found ($F(5,2) = 25.144$, $p = 0.039$), with an $R^2$ of 0.945. Participants' predicted reaching distance was equal to -0.598 + 1.384(synergy takeover threshold) + 0.941(synergy emergence threshold) + 1.049(reaching flexor spasticity) – 0.139(elbow extension strength) – 0.317(shoulder abduction strength). The synergy takeover ($p = 0.028$) and emergence ($p = 0.031$) thresholds were the only significant predictors. More individuals will be enrolled in the study to ensure that each variable is normally distributed, to expand the number of regressors, and to increase the generalizability of the study. This study will attempt to identify the most important constitutive elements predicting reaching function. It is imperative to understand the magnitude of each factor's contribution in order to better inform clinical trial design and current practice.

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**H3 G-B**

**ORIGIN OF RECURRENT MOTOR CIRCUITS IN ZEBRAFISH SPINAL CORD**

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Locomotor movements are a fundamental aspect of behavior and are generated by pools of motor neurons distributed throughout the spinal cord. Spinal motor neurons innervate peripheral fast- and slow-twitch fibers to elicit the muscle contractions responsible for movements; however, some motor neurons also exhibit axonal collaterals that terminate within the spinal cord. These intraspinal axon collaterals form synaptic connections with other motor neurons and inhibitory Renshaw cells, providing monosynaptic 'recurrent excitation' and disynaptic 'recurrent inhibition', respectively. Recurrent motor circuits have been identified in vertebrates ranging from fish to humans; however, the functional role of these circuits is still mysterious. Here, we are beginning to examine the role of recurrent motor circuits in zebrafish to provide a better idea of their evolutionary origins and the precise function of these structures in motor control. Thus far, we have determined that only subsets of zebrafish motor neurons possess axon collaterals, and we have confirmed their function as output structures using in vivo synaptophysin labeling. In addition, we have identified putative motor neuron to motor neuron connections and connections to a group of inhibitory interneurons derived from the same progenitor domain that gives rise to Renshaw cells in mammals. Our results suggest that motor neurons in zebrafish, as in mammals, likely participate in the patterning of locomotor activity beyond pure muscle control by providing 'recurrent' excitation and 'recurrent' inhibition to other motor neurons. The relative simplicity of zebrafish spinal cord and its accessibility to a number of techniques that allow for in vivo assessments of circuit function set the stage for a greater understanding of these fundamental circuits.

**H4 UG-A**

**THERMOSENSORY EFFECTS ON DROSOPHILA CIRCADIAN RHYTHMS**

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Circadian rhythms are the daily cycles in an organism's behavioral, physiological and biochemical processes which are driven by a molecular oscillator known as the circadian clock. Temperature has a profound effect on circadian behavior and day/night changes in ambient temperature serve as a cue to synchronize the clock to the environment,

a phenomenon known as temperature entrainment. Additionally, when an animal is exposed to constant hot or cold temperature (such as in different seasons), the period of circadian rhythms remains reasonably close to 24 hrs despite the expected slowing/speeding up of biochemical processes, a phenomenon called temperature compensation. However, the exact mechanism by which the clock senses external temperature remains unknown, even though both entrainment and compensation are well described.

Our goal is to study how the neural network that controls behavioral rhythms in activity in the Drosophila brain (the "clock neurons") receives and processes temperature information.

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**H5 PD-B**
**INACTIVATING $K_V$ CURRENTS IN TYPE II HAIR CELLS OF THE MOUSE UTRICLE**

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Hair cells of the mammalian utricle detect head motions and tilt, contributing to the coordination of movement and balance. The mechanosensitive hair cells are of two types, I and II. Like most hair cells, type II cells have large outwardly rectifying $K^+$ conductances that are activated by depolarizing receptor potentials and tend to repolarize the hair cell. Despite their prominence among currents that contribute to the receptor potential, their molecular composition is not fully known. $K_V1$ voltage-gated potassium channels are likely candidates based on their voltage dependence, expression data in mouse utricular hair cells (Scheffer et al., Journal of Neuroscience 35(16):6366-80, 2015), and characterization of pigeon type II hair cells (Correia et al., Neuroscience, 152(3):809-820, 2008). Here we have investigated $K_V1$ contributions in mouse type II hair cells with the Kv1-selective channel blocker, $\alpha$-dendrotoxin ($\alpha$DTX), a component of mamba snake venom.

We used the patch clamp method to record whole-cell voltage-gated currents from identified type II hair cells in semi-intact preparations of the mouse utricle (postnatal days 5-7). Recorded cells were identified as type II and mapped relative to the distinct central and peripheral zones (striola and extrastriola, respectively) of the utricular epithelium. Recordings were obtained with conventional external and internal solutions, at room temperature, in response to conventional voltage protocols. Depolarizing steps evoked an inactivating outward current, $I_A$, which we measured by subtracting steady-state current from peak current. For steps to +36 mV, the extent of inactivation of $I_A$ was significantly more prominent in extrastriolar type II hair cells than in striolar type II hair cells (p=0.01).

$\alpha$DTX differentially affected the inactivating and sustained current components. For steps from –124 mV to –4 mV, $\alpha$DTX blocked $I_A$ by 44±10% (11 cells) at 10 nM and by 61±7% at 100 nM (9 cells). Similar $\alpha$DTX-sensitivity was observed for steps to +16 mV. These data yield an IC50 of ~10 nM, in the range of $K_D$'s for $\alpha$DTX-sensitive $K_V1$ subunits (1-25 nM). In contrast, the IC50 for steady-state current was >100 nM. The voltage dependence, inactivation and $\alpha$DTX-sensitivity suggest that $I_A$ in mouse utricular type II hair cells may flow through heteromeric channels comprising Kv1.1 and Kv1.4 $\alpha$ subunits and Kv$\beta$1 subunits (Rudy et al., Encyclopedia of Neuroscience, 10:397-425, 2009).

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**H6**
**GENE EXPRESSION ANALYSIS AS A TOOL TO MEASURE RECOVERY OF THE NASOPHARYNGEAL REFLEX**

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The mammalian diving response is characterized by bradycardia, apnea, and systemic vasoconstriction that diverts blood to oxygen-sensitive CNS and heart tissue. The afferent signal initiating the diving response is thought to be carried via the anterior ethmoidal nerve (AEN). Similar to the diving response, the nasopharyngeal reflex can be initiated in anesthetized animals after stimulation of the nasal passages with water or ammonia vapors. The AEN is also important for initiation of the nasopharyngeal reflex, as bilateral AEN sectioning severely attenuates this response. More recently, however, it was found that 3 days after bilateral AEN sectioning the nasopharyngeal response is not different from sham operated rats with intact AENs. A literature review provided several workable hypotheses for the mechanism of nasopharyngeal reflex response recovery. 1) Another trigeminal nerve is primed to assume the role of the AEN in activating the reflex after AEN transection. 2) Previously "silent" synapses and pathways within the trigeminal nucleus could become activated after AEN transection. 3) Changes occurring in the trigeminal ganglion (TG) enable recovery of the reflex. Peripheral nerve injury (PNI) results in a measurable change in genetic activity. A number of genes showing increased expression following PNI have been labeled as "regeneration-associated" due to their neurotrophic roles at the level of the sprout, axon, or synapse. Here we study the changes in expression of three different genes within the TG 3 hr after AEN transection. These genes, Insulin Growth Factor 1 (IGF-1), Microtubule Associated Protein Tau (MAPT), and Angiotensin II Receptor type 2 (AGTR2) have some evidence to support neurotrophic roles. We hypothesized that a measurable upregulation of gene transcription would occur following nerve transection as the AEN responded to transection injury. Six rats were anesthetized and underwent AEN sectioning. Three hr later the rats were sacrificed and TGs were harvested. qPCR was performed to quantify RNA. Probes for IGF-1, MAPT, and AGTR2 gene expression showed +1.39, +1.46, and

+1.27 fold changes, respectively, when compared to control glyceraldehyde-3-phosphate dehydrogenase (GAPDH) expression. Although statistical significance was not reached given the small sample size, this investigation carries implications for future studies. Regeneration associated gene expression can be studied at various time intervals before and after restoration of the nasopharyngeal reflex, leading to the possibility of isolating a particular genetic role involved in this restorative process.

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**H7 UG-B**
**MITOCHONDRIA IN VESTIBULAR EFFERENT BOUTONS**

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Mitochondria play a crucial role in our cells, as they are the primary producers of ATP, which powers the cell. Mitochondria contain cristae, which are enfoldings within the mitochondria that expand the surface area of the mitochondria. Our focus was to look at the mitochondria within hair cells in the inner ear. Using IMOD software, we segmented ultrastructural sections of mitochondria, cristae and dense core vesicles in vestibular efferent boutons. These mitochondria were derived from rats, and studied using EM tomography in a high-voltage microscope at the NCMIR at UCSD. Sections 300 nm in thickness were examined. The objects of interest in this particular study were efferent buttons, which contact vestibular Type I and Type II hair cells in the inner ear. The assumption is that the more centrally located Type I hair cells are more sensitive than those which are located more peripherally. The ultimate goal is to see the effect of ototoxic agents, such as aminoglycoside antibiotics and chemotherapeutics, on each of the different types and classes of mitochondria. Using cells from both the liver and the brain of rats, we are currently working out parameters for determining metabolic activity in mitochondria, which we will then apply to isolated mitochondria from inner ear hair cells. After running these experiments at various cell densities, we are attempting to find the perfect density in which antibiotics are effective on the cells. Running the mitochondrial stress test produces a chart that displays the effect of each agent added, with respect to the oxygen consumption rate. The agents used were oligomycin, FCCP, Antimycin A and Rotenone. We split our samples into various fractions, then normalize the cell counts in the different fractions to compare them. Once normalized, we will attempt experiments in which ototoxic agents added, and determine any potential loss in oxygen consumption rates (pMoles $O_2$/min) after the addition of the particular ototoxic agent. Results from this study should inform us about the effects of ototoxic agents on particular classes of inner ear hair cell mitochondria, and point a way toward optimal therapeutics for hair cell loss.

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**H8 G-A**
**A RETINAL GANGLION CELL USES INTRINSIC PROPERTIES TO CONTROL ITS FEATURE SELECTIVITY**

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Feature selectivity of higher order sensory neurons is computed through both synaptic input and intrinsic properties. Retinal Ganglion Cells have a diverse range of feature selectivity and are the final information processing step between the retina and the brain. Here we describe two cell types, the Transient Suppressed-by-Contrast (tSbC) and the Sustained Off Alpha (sOffA) that have similar synaptic input yet are selective for different features of the visual environment. The difference can be accounted for by the different intrinsic properties of these neurons. The tSbC undergoes depolarization block in response to light while the sOffA does not. In addition, it is likely that there is a difference in distribution and type of ion channels. By examining the interplay of synaptic inputs and intrinsic cell properties, we can illuminate possible mechanisms for cell specific computations throughout the nervous system.

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**H9 G-B**
**FUNCTIONAL DEPENDENCE ON CONTRALESIONAL HEMISPHERE AND ITS RELATIONSHIP WITH CORTICO-RETICULOSPINAL STRUCTURE AND HAND/ARM IMPAIRMENT IN MODERATE TO SEVERE CHRONIC STROKE**

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Hand function, particularly opening, is often significantly impaired following stroke, particularly in moderate to severely impaired individuals. Unfortunately, lifting the arm, a crucial component of many activities of daily living, usually further reduces hand opening ability in this population. This is a consequence of involuntary abnormal coupling between the shoulder abductors, elbow, wrist, and finger flexors termed the “flexion synergy”. Evidence from primates and, more recently, diffusion imaging in humans implicates increased reliance on the contralesional cortico-reticulospinal (CRS) pathway as a possible reason for this synergy-related impairment. However, evidence connecting functional activity related to the arm/hand and structural changes in the CRS pathway is currently missing.

To fill the above gap, we recruited 14 individuals with moderate to severe chronic hemiparetic stroke and 3 healthy age-matched controls. Individuals first took part in an MRI scan, including structural T1-weighted and Diffusion-weighted images. Individuals then participated in a high-density EEG experiment in which subjects performed 2 tasks on an ACT3D robot: 1. Hand opening and 2. Hand opening while lifting

against 50% maximum shoulder abduction (SABD) force, using the paretic (stroke) or dominant (control) hand. We then quantified the following: 1. Cortical activity related to hand opening with or without lifting; 2. Fractional Anisotropy (FA) of the contralesional reticular formation.

Individuals with stroke demonstrated an increased functional reliance on the contralesional hemisphere compared to controls during hand opening as evidenced with the EEG measurements. Furthermore, the addition of lifting caused an even greater reliance on the contralesional hemisphere in stroke, but not controls. Use of the contralesional hemisphere was positively associated with higher FA in the contralesional reticular formation, a potential indicator of white matter integrity. These findings suggest that greater functional reliance on the contralesional hemisphere was associated with greater white matter structural integrity in the contralesional reticular formation.

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**H10 G-A**

**GENETICALLY MODIFIED MICE TO STUDY SCHWANN CELL-SPECIFIC PATHWAYS *IN VIVO* IN A MODEL FOR PAINFUL OBESITY-INDUCED NEUROPATHY**

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*Department of Cell and Molecular Physiology, Stritch School of Medicine, Loyola University Chicago, Maywood, IL*

Painful diabetic neuropathy (DN) is one of the most common complications of obesity and type II diabetes. Twenty-five to 50% of diabetic patients will experience neuropathy at some point during the disease course (Abbot et al., 2011), severely affecting their life quality. The complex molecular and cellular neurobiology triggering the disease is still unknown and no pharmacological treatment is available as of today. It has been proposed that a disruption of the cross-talk between Schwann cells (SC) and sensory neurons may be the underlying cause of DN. Therefore, to study whether SC machinery is affected in DN, we created a mouse model to qualitatively investigate mRNA and gene expression as well as quantitatively analyze mitochondria specifically in SC following high-fat diet or acute lipid injections. The first step was to optimize the cell specific method and validate it. To this end, we used B6.Cg-Gt(ROSA)26Sortm9(CAG-tdTomato)Hze/J mice (td tomato) that have a loxP-flanked STOP codon followed by a td Tomato reporter gene and crossed them with MPZ-Cre-recombinase mice, which have Cre-recombinase under a SC-specific promoter. The offspring expressed td Tomato reporter gene specifically in SC. This cell-specific labelling allowed us to sort SC td Tomato positive – MPZ-tdTomato$^{+/+}$ – using Fluorescence-Activated Cell Sorting (FACS) and to perform further analyses specifically in SC and not in the sensory neurons associated with them. The FACS results clearly showed two different cell populations in the offspring, MPZ-tdTomato$^{+/+}$ and MPZ-tdTomato$^{-/-}$ cells, indicating that the mouse breeding approach utilized produced highly specific tomato expression. Conversely, both wild-type and td tomato mice had only MPZ-tdTomato$^{-/-}$ cells, as expected. We also performed an estimate of the number of copies of mitochondria based on mitochondrial DNA qPCR analyses and immunofacs methodology. Our data demonstrated that only Schwann cells were extracted from the mice MPZ-tdTomato$^{+/+}$ sciatic nerve. In addition, mice on 12 weeks of high fat diet (HFD) demonstrated a reduced number of mitochondria in SC when compared to normal chow fed mice. In conclusion, this study shows that the presented Schwann cell-specific sorting technique can be employed to specifically evaluate the changes induced by obesity in mice Schwann cells, as well it suggests that mitochondrial pathways may be involved in obesity-induced neuropathy.

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1st Place: **Stuart Lukz,** Adlai Stevenson High School
2nd Place: **David Liang**, Adlai Stevenson High School
3rd Place: **Krishnachandra Nair**, Illinois Math and Science Academy

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*Thanks to the Judges of the **Graduate Student Symposium**:*

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Thanks to **Emma Childs, Ph.D. and Doug Wallace, Ph.D.** for coordinating the **Postdoctoral Fellow Poster Competition**

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# NOTES

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