2023 PROGRAM BOOK

March 18 - 22, 2023

Lexington, Kentucky

53rd Annual American Society for Neurochemistry Meeting

ASN 2023

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WELCOME MESSAGE

Dear ASN Members,

I want to wish each of you a Healthy and Successful New Year in 2023.

The annual ASN meeting is a one-of-a-kind meeting that brings together young and seasoned scientists to experience cutting edge neurochemistry and neurobiology, and for enhancing the careers of young investigators.

On behalf of the ASN Council/ Officers/ Planning committee, I warmly welcome you to the ASN 2023 Annual Meeting here in Lexington, Kentucky at the Hyatt Regency Hotel.

The 2023 Lexington ASN Meeting offers a dynamic and exciting scientific program presenting 4 internationally renowned Plenary Speakers together with 24 symposia and 10 colloquia covering the most recent evidence on a wide range of research topics. Our Diversity and Inclusion committee is dedicated to having an inclusive venue, collaborative culture, and a safe and professional environment. Multiple social gatherings will unite young and established scientists with diverse expertise and backgrounds. New and emerging scientists will be recognized with prestigious awards while poster sessions will host lively discussions to trigger future collaborations.

We are happy you made it to Lexington, and we hope you enjoy the meeting!

Sincerely,

Seema Kaushayla Tiwari-Woodruff, PhD
President - ASN 2023 Planning Committee

ASN 2023 PROGRAM BOOK

TABLE OF CONTENTS

GENERAL INFORMATION

ASN2023 COMMITTEES

Planning Committee

Seema Kaushalya Tiwari-Woodruff
President

Selva Beltan
Chair

Erhard Bieberich
Treasurer and Local Committee Chair

Marion Buckwalter
Secretary

Program Committee Members

Selva Baltan, M.D., PhD
Babette Fuss, PhD
Ranjan Dutta, PhD
Christian Gonzalez-Billault, PhD
Arturo Ortega Soto, PhD
Astrid Cardona, PhD
Elena Blanco-Suarez, PhD
Vladimir Parpura, M.D., PhD

Local Host Committee Members

Erhard Bieberich, PhD
Linda Van Eldik, PhD
Donna Wilcock, PhD
Stefka Spassieva, PhD
Meifan (Amy) Chen, PhD
Erica Weekman, PhD
Maj-Linda Selenica, PhD
Zhihui Zhu, PhD
Priyanka Tripathi, PhD
John Gensel, PhD
Christopher Norris, PhD
Daniel Lee, PhD
Adam Bachstetter, PhD
Ahmed Elsherbini, PhD
Zainuddin Quadri, PhD

GENERAL INFORMATION

ABOUT ASN ANNUAL MEETING

The ASN Meeting is an annual event that started back in 1970. For the most part, it has been hosted throughout the United States with only a few meetings hosted outside of the US in Canada and Mexico.

ABOUT AMERICAN SOCIETY FOR NEUROCHEMISTRY (ASN)

The American Society for Neurochemistry's Missions:

- To advance and promote cellular and molecular neuroscience knowledge
- To advance, promote, support, encourage and facilitate communication among investigators in neurochemistry and related neurosciences
- To promote, support, encourage and facilitate the dissemination of information concerning neurochemical research through scientific meetings, seminars, publications and related activities
- To promote, support and encourage the research of individual cellular and molecular neuroscientists and to engage in any and all other activities for the advancement of the science of neurochemistry which may be deemed advisable
- To insure that all of its activities remain open to the full participation of scholars of all backgrounds and nationalities

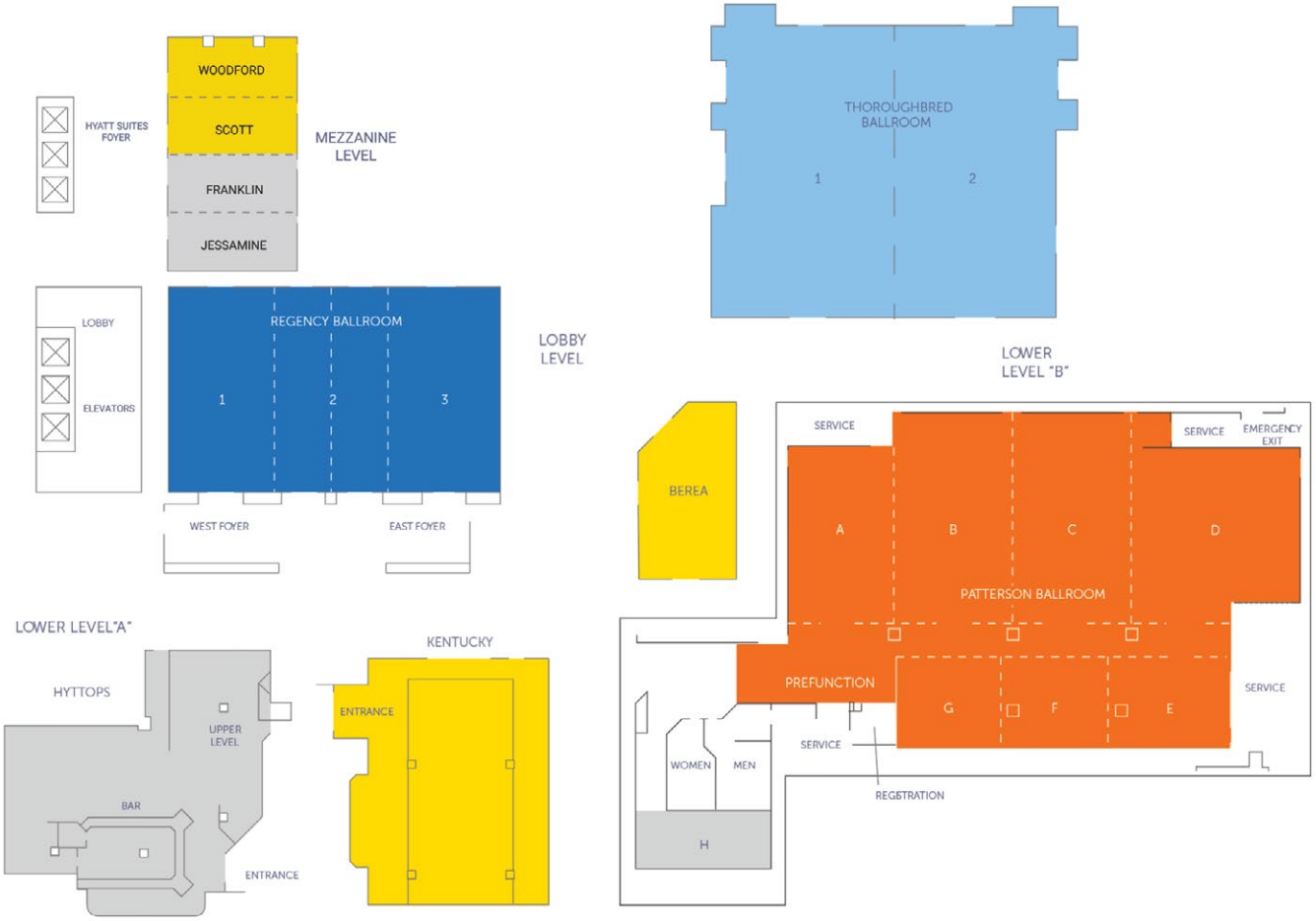
GENERAL INFORMATION

VENUE FLOORPLAN

LOBBY LEVEL
- Hyatt Regency Lobby Foyer
- Regency Ballroom
- Thoroughbred Rooms

LOWER LEVEL B
- Patterson Ballroom
- Berea Room

LOWER LEVEL A
- Kentucky Room



Main Session & Session Room

Session Room

Exhibit & Poster Hall
Welcome Reception & Gala Dinner

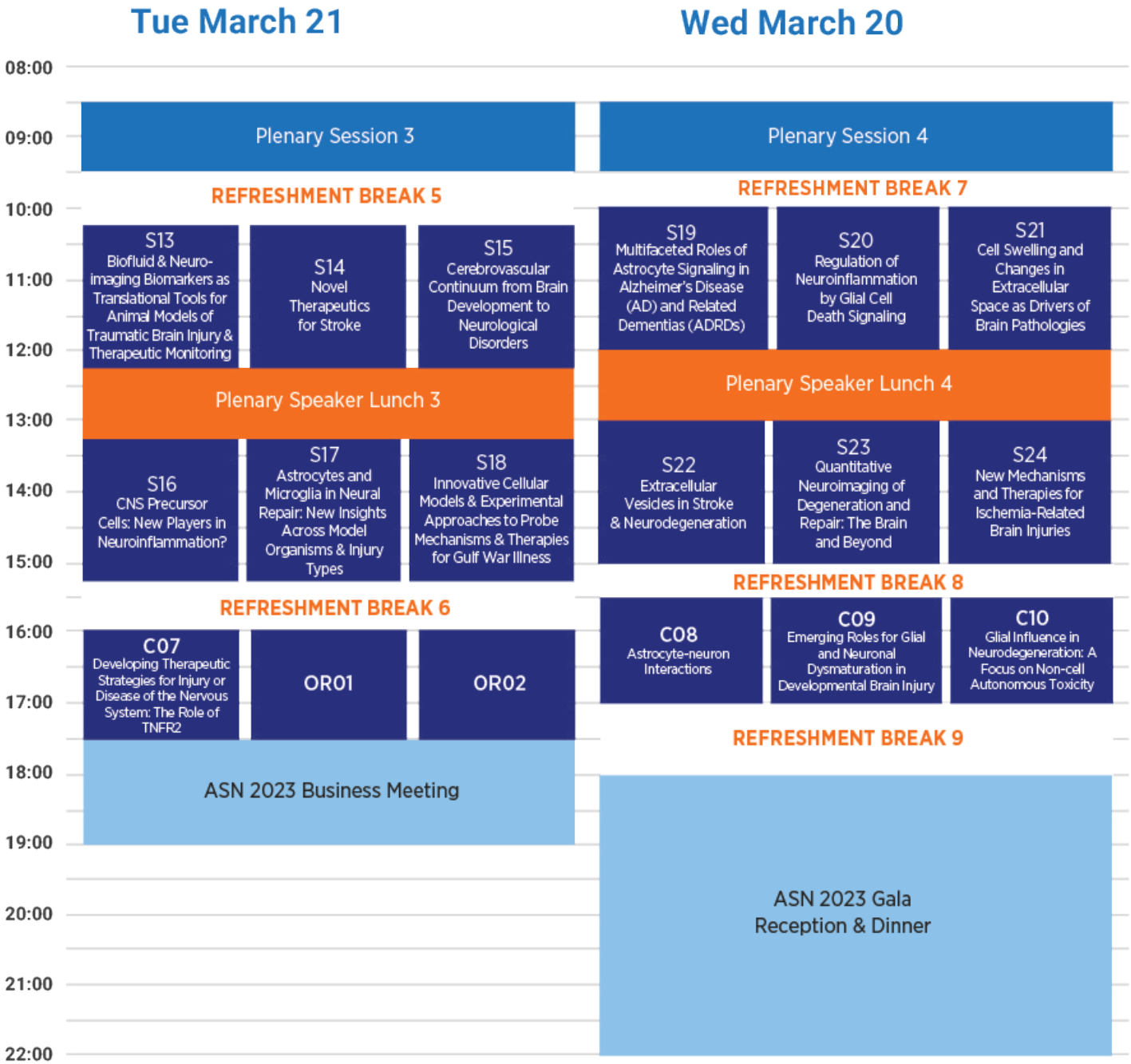
Meeting Rooms

PROGRAM

SCHEDULE AT A GLANCE

PROGRAM

SCHEDULE AT A GLANCE



PROGRAM

PLENARY SPEAKERS

Supported by a grant from NIH National Institutes of Health Turning Discovery Into Health

Nicola Allen, PhD

Salk Institute, San Diego

Astrocyte-Neuron Interaction in Health and Disease

Alejandro F. Schinder, PhD

Leloir Institute, Buenos Aires

Neurogenesis and Circuit Remodeling in the Adult and Aging Hippocampus

ISN Lecture

Amita Sehgal, PhD

University of Pennsylvania, Philadelphia

How and Why We Sleep: Insights From a Small Animal Model

Robert Hill, PhD

Dartmouth College, Hanover

Replacing Dying Oligodendrocytes One Cell at a Time

PROGRAM
SOCIAL EVENTS

SATURDAY, MARCH 18

ASN 2023 First Timers' Reception
5:00pm – 5:30pm
Berea Room
Open to all registered first-time attendees. Name badges must be worn and visible for entry.

Attending ASN for the first time? We welcome you to join other first-time attendees and meet new friends over drinks.

ASN 2023 Welcome Reception
5:30 PM – 7:00 PM
Patterson Ballroom, Exhibit and Poster Hall
Open to all registered attendees. Name badges must be worn and visible for entry.

We are excited to welcome you to ASN 2023 with a drink and some light canapes surrounded by your ASN colleagues! Join us at the Welcome Reception and catch up with some old friends or make new ones.

Sunday, March 19

Plenary Speaker Lunch 1
12:15pm – 1:15pm
Patterson H
Pre-registration required.

Lunch with Dr. Nicola Allen.
***Any pre-registered attendees late for the lunch will forgo their seat which will then be given to anyone on the wait list.**

Women in Neurochemistry Lunch
12:15pm – 1:15pm
Kentucky Room
Pre-registration required.

The Women in Neurochemistry lunch will be an interactive discussion on how to navigate, enjoy and succeed in a career in neurochemistry. Come and meet others, network, and exchange tips on how to create an ever more inclusive and diverse space in which we can all be outstanding scientists and mentors. There will also be an opportunity to set up peer mentoring groups at the luncheon.

Supported by a grant from NIH National Institutes of Health Turning Discovery Into Health

Wine & Cheese Poster Session

5:30pm – 7:00pm
Patterson Ballroom, Exhibit and Poster Hall
Open to all registered attendees. Name badges must be worn and visible for entry.

Learn about new research and network with poster abstract authors.

Supported by ACS Chemical Neuroscience | ACS Infectious Diseases | ACS Medicinal Chemistry Letters | ACS Pharmacology & Translational Science | Journal of Medicinal Chemistry

ASN 2023 Student Post-Doc Mingler

7:00pm – 10:00pm
Infinity Bar, Roof Top Restaurant + Bar View Walking Directions
Open to student/post docs. Pre-Registration required

Meet some of your fellow students or post docs overlooking Lexington, Kentucky in this scenic rooftop lounge.

Monday, March 20

ASN Highschool Day

9:00am – 1:00pm

One of the central missions of the ASN is to introduce academic career choices and promote diversity, equity, and inclusion at early levels of education and scientific training. As part of this effort, we have invited students from a local High School to engage with early career scientists and experience the scientific excitement of the ASN conference. This year, we will welcome students from Frederick Douglass High School, which offers a vibrant biology program and collaborates with the University of Kentucky to promote college readiness of their students. The ASN is enthusiastic about providing this opportunity for young students to experience the latest research in neurochemistry and inspiring them to pursue a scientific career.

Supported by NIH National Institutes of Health Turning Discovery Into Health

Plenary Speaker Lunch 2

12:15pm – 1:15pm
Patterson H
Pre-registration required.

Lunch with Dr. Alejandro Schinder.

*Any pre-registered attendees late for the lunch will forgo their seat which will then be given to anyone on the wait list.

Young Investigators Forum

Leveling Up Your Leadership: A Panel Discussion

5:30pm – 7:00pm

Regency Ballroom

Open to all registered attendees. Name badges must be worn and visible for entry.

Are you a young investigator wondering what your next career move is and how to get there? The Young Investigator Advisory Committee has got you covered! We have brought together scientists from academia, industry, and other career paths you may not have considered to help you chart a path forward. Come for refreshments and learn how our panelists arrived at their current positions and what leadership skills they developed to get there.

Tuesday, March 21

Plenary Speaker Lunch 3

12:15pm – 1:15pm

Patterson H

Pre-registration required.

Lunch with Dr. Amita Sehgal.

***Any pre-registered attendees late for the lunch will forgo their seat which will then be given to anyone on the wait list.**

Young Investigators Lunch

Borrowed Brilliance: Leveling Up Your Leadership

12:15pm – 1:15pm

Kentucky Ballroom

Pre-Registration and advance payment via event brite required.

Join us for lunch on for a presentation and discussion on Leveling up Your Leadership given by Dr. Stefanie Robel, an expert in building better leaders and mentors in academia. Purchase of Eventbrite ticket required

Wednesday, March 22

Plenary Speaker Lunch 4

12:00pm – 1:00pm

Patterson H

Pre-registration required.

Lunch with Dr. Robert Hill.

***Any pre-registered attendees late for the lunch will forgo their seat which will then be given to anyone on the wait list.**

Gala Reception & Dinner

6:00pm – 10:00pm

Patterson Ballroom

Open to all registered attendees. Event ticket required for entry.

Join your fellow attendees with an evening featuring Lexington's delectable flavors. We can't wait to see you on the dance floor as we wind down and celebrate ASN 2023.

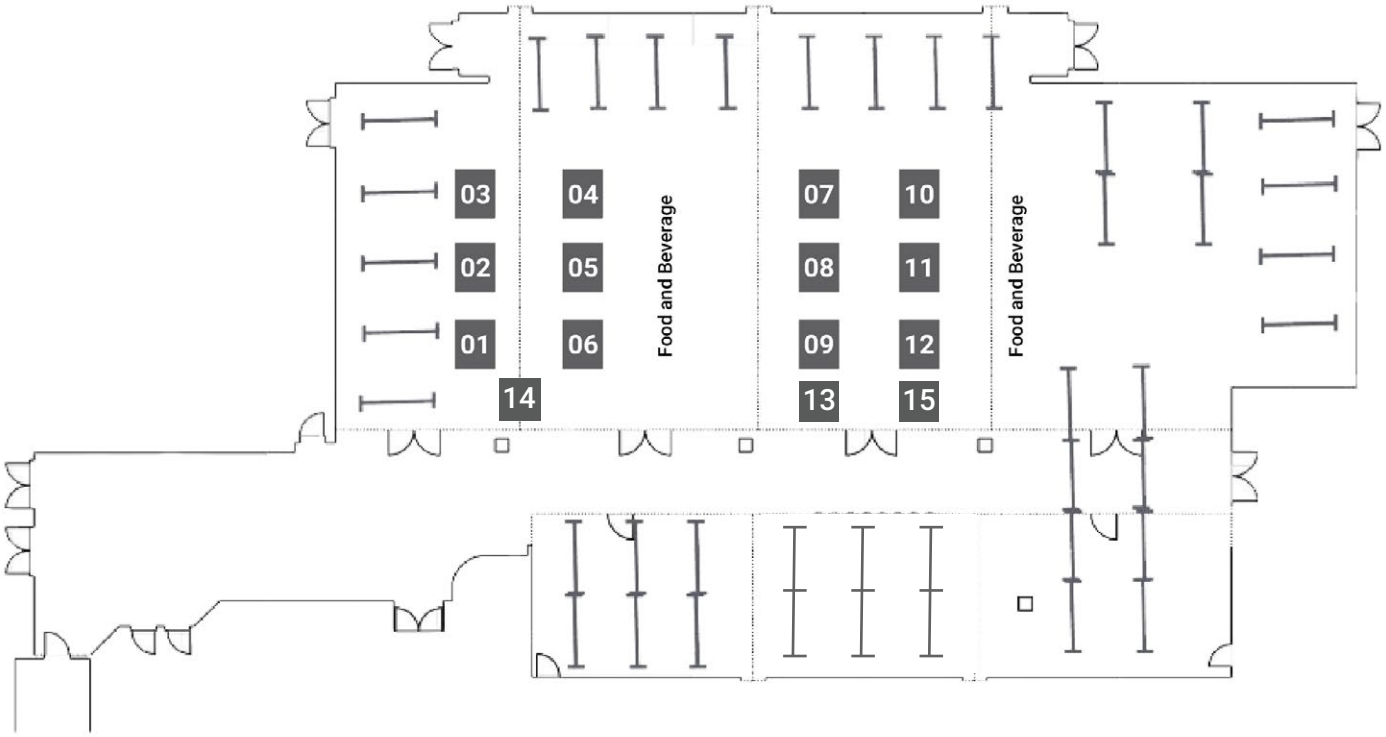
EXHIBITORS

EXHIBITOR INFORMATION

EXHIBIT AND POSTER HALL HOURS

Patterson Ballroom

Saturday, March 18	5:30pm – 7:00pm **(Welcome Reception)**
Sunday, March 19	9:30am – 5:30pm | 5:30pm – 7:00pm **(Poster Reception)**
Monday, March 20	9:30am – 7:00pm
Tuesday, March 21	8:30am – 12:15pm | 12:15pm – 5:30pm **(Posters Only)**



EXHIBITOR	TABLE #
Andor Technology	Booth 10
College of Medicine, Office of Research	Booth 8
Gene Tools, LLC	Booth 7
Glut1 Deficiency Foundation	Booth 1
International Society of Neurochemistry	Booth 3
Kent Scientific Corporation	Booth 9
Miltenyi Biotec	Booth 2
Molecular Instruments, Inc	Booth 5
Neuroscience Associates, Inc	Booths 11&12
Particle Metrix Inc.	Booth 6
Precision Systems and Instrumentation, LLC	Booth 15
RWD Life Science	Booth 14
Stoelting Co.	Booth 13
VectorBuilder Inc.	Booth 4

EXHIBITORS

ASN 2023 EXHIBITORS

BOOTH 10 ANDOR TECHNOLOGY

Andor's a global leader in the manufacture of scientific imaging cameras and microscopy systems, complemented by Imaris software for 3/4D image stitching, visualization, and analysis. We provide workflow solutions for neuroscientists capturing from the nm through to cm scale, including deep imaging, photostimulation tools for optogenetics, axotomy, and investigating dynamic events. Our camera portfolio addresses low light conditions of single molecule detection and vesicle trafficking while also providing solutions for live imaging and large fields of view. Imaris is ideally suited for large light microscopy images with analysis tools for neuron and vessel tracing, spine characterization, and Sholl analysis.

andor.oxinst.com

BOOTH 8 COLLEGE OF MEDICINE, OFFICE OF RESEARCH

The University of Kentucky College of Medicine promotes an inclusive environment providing excellent medical and biomedical education, equitable health care, and transformative research to improve the well-being of Kentuckians and beyond. As one of six health science colleges on campus, our team benefits from interprofessional education and collaborative scientific discovery.

medicine.uky.edu

BOOTH 7 GENE TOOLS, LLC

Gene Tools manufactures Morpholino oligos for blocking translation, modifying splicing or inhibiting miRNA activity. Morpholinos are used in cell cultures, embryos or, as Vivo-Morpholinos, in adult animals. Morpholinos are effective, specific, stable and non-toxic. Backed by Ph.D.-level customer support, Gene Tools designs and synthesizes Morpholinos and offers cytosolic delivery options.

www.gene-tools.com

BOOTH 1 GLUT1 DEFICIENCY FOUNDATION

The Glut1 Deficiency Foundation is a nonprofit patient advocacy organization dedicated to improving lives in the Glut1 Deficiency community through its mission of increased awareness, improved education, advocacy for patients and families, and support and funding for research.

www.g1dfoundation.org

BOOTH 3 INTERNATIONAL SOCIETY OF NEUROCHEMISTRY

The International Society for Neurochemistry (ISN) is a nonprofit membership organisation and the only international society focused on neurochemistry. With a proud history dating back to its establishment in 1965, ISN strives to promote all relevant aspects of molecular and cellular neuroscience.

www.neurochemistry.org

BOOTH 9 KENT SCIENTIFIC CORPORATION

For over 30 years Kent Scientific has served medical and research scientists as a worldwide provider of integrated solutions for pre-clinical research and drug discovery advancement. As the world leader in noninvasive blood pressure, physiological monitoring and anesthesia systems for rodents, we enable researchers to achieve fast, consistent, accurate results.

www.kentscientific.com

BOOTH 2 MILTENYI BIOTEC

For over 30 years, Miltenyi Biotec has been a leader in the development of products that empower the advancement of biomedical research and enable cell and gene therapy. We provide innovative tools to help with your sample preparation, cell isolation, cell culture, and cell analysis needs. Our solutions are designed to support everything from basic research to translational research and clinical application for immunology, stem cell biology, neuroscience, and cancer.

www.miltenyibiotec.com

BOOTH 5 MOLECULAR INSTRUMENTS, INC

Molecular Instruments® (MI) designs and synthesizes kits for multiplexed, quantitative, high-resolution bioimaging in academic research, drug development, and clinical pathology and diagnostics. MI offers custom assay design and development services for life sciences and pharmaceutical laboratories and companies, as well as nucleic acid sequence design and analysis services for agricultural and veterinary enterprises. Our mission is to make multiplexed, quantitative imaging of biomolecules routine and accessible.

www.molecularinstruments.com

BOOTH 11-12 NEUROSCIENCE ASSOCIATES

NeuroScience Associates (NSA) uniquely provides mass production neurohistology. Proprietary MultiBrain® technology enables NSA to embed, section and stain up to 40 neuronal tissues simultaneously in one MultiBrain® block. With facilities to process up to 16 MultiBrain® blocks daily, NSA can section thousands of brains per week. NSA has 30+ years of experience applying classic histological stains (ICS, H&E, Nissl), plus IHC with custom antibodies. These staining services complement MultiBrain® processing to significantly reduce client's R&D cycle times. NSA provides slide digitization and remote viewing of scans for downloading via Internet (Proscia). No other company provides this distinctive combination of services.

www.neuroscienceassociates.com

BOOTH 6 PARTICLE METRIX

Particle Metrix manufactures the ZetaView® Nanoparticle Tracking Analysis system for measurement of particle size distribution, number concentration and zeta potential, including the targeted measurement of sub-populations for exosomes & viruses, using fluorescence NTA. Our new ultra-fast laser/filter switching capability provides the determination of biomarker colocalization scores with our TWIN-laser systems.

www.particle-metrix.de

BOOTH 15 PRECISION SYSTEMS AND INSTRUMENTATION, LLC

For over 20 years, Precision Systems and Instrumentation, LLC has manufactured, sold, and supported the finest spinal and cortical impactors worldwide. Our primary products include the Infinite Horizon Spinal Cord Impactor and TBI-0310 impactors. We also provide a full range of service options and custom accessories.

www.presysin.com

BOOTH 14 RWD LIFE SCIENCE

Since 2002, RWD Life Science has been the world leading manufacturer of laboratory instruments for scientific research. We specialize in providing solutions for animal surgery and modeling, neural signals and in vivo Imaging, histopathology and molecular biology for researchers. For more information, please visit our website.

www.rwdstco.com

BOOTH 13 STOELTING CO.

Stoelting has been a leader in development, design, and sale of neuroscience research equipment since 1886. Our line of Stereotaxic Instruments is world-renowned. Moreover, our intuitive, feature-rich, and all-inclusive ANY-maze behavioral tracking software is unmatched (www.ANY-maze.com). We also offer solutions for Animal Identification including Ear Tags and Markers.

www.StoeltingCo.com

BOOTH 4 VECTOR BUILDER INC

VectorBuilder is a global powerhouse in genetic engineering solutions: we are the world's largest provider of custom vectors for viral and non-viral gene delivery, constructing over 80,000 vectors a year, delivered to researchers in six of the seven continents. Our vector packaging systems include lentivirus, AAV, adenovirus, MMLV, baculovirus, VSV, HSV, PiggyBac, Sleeping Beauty, and more. Besides vectors and virus packaging, we offer a wide range of services pertaining to gene editing (Crispr and shRNA), pre-made shRNA and gRNA libraries for multiple species, as well as custom library construction, in vitro and in vivo library screening services, stable cell line generation, and mRNA synthesis/encapsulation. Come join us in the gene delivery revolution

en.vectorbuilder.com

ASN 2023
FULL PROGRAM

SATURDAY MARCH 18, 2023

TIME	DETAILS	DESCRIPTION
1:00pm - 3:00pm	Local Symposium/Pre-Meeting Workshop - Healing, Pain and the Opioid Crises **Regency Ballroom** **Chair:** John C Gensel, PhD University of Kentucky **Co-chair:** Stefka Spassieva, PhD University of Kentucky	Opioid treatment of chronic pain can lead to addiction, which caused one of the worst public heath crises in the Commonwealth of Kentucky and in the United States. In this symposium, researchers from the University of Kentucky, including scientists from the HEALing community study, and their collaborators will present forefront research on understanding and addressing opioid addiction and finding novel treatments to manage pain.
4:00pm - 6:00pm	Public Forum - Healthy Brain Aging - Addressing Risks and Disparities in Our Community **Regency Ballroom** **Chair:** Dr. Linda van Eldik, PhD Dr. Erhard Bieberich, PhD, University of Kentucky **Speakers:** Dr. Lauren Whitehurst, PhD Dr. Erin Abner, PhD Dr. Frederick Schmitt, PhD Dr. Ima Ebong, MD	Most of our nerve cells are not renewed throughout life and therefore, our brain is most vulnerable to aging. Age-related nerve degeneration can lead to dementia and Alzheimer's disease, a public health problem prevalent in the Commonwealth of Kentucky and disproportionally impacting African-American and underserved communities. In this public forum, researchers from the University of Kentucky will discuss the latest research on risk factors affecting healthy brain aging, lifestyle choices that can restore the healthy brain, and strategies to address health disparities in our community.

SUNDAY MARCH 19, 2023

TIME	DETAILS	DESCRIPTION
8:00am - 9:30am	**ASN 2023 Opening Remarks & Plenary Session 1** **Regency Ballroom** **PL.01 - Astrocyte-Neuron Interaction in Health and Disease** Nicola Allen (supported by the Basic Neurochemistry text)	
10:15am - 12:15pm	**S01 - Gut Microbiome and Neurological Diseases: From Association to Function** **Regency Ballroom** **Session Chairs:** Ashutosh Mangalam Javier Ochoa-Reparaj S01.01 - Role of the Gut Microbiome in Ad-Like Pathology in Mouse Models Sangram Sisodia S01.02 - The Immunomodulatory Role of Gaba-Producing Bacteria in CNS Inflammatory Demyelination Javier Ochoa-Reparaj S01.03 - Intersections of the Maternal Microbiota and Nervous System Development Helen Vuong S01.04 - The "Gut Feeling": Breaking Down the Role of Gut Microbiome in Multiple Sclerosis Ashutosh Mangalam	Trillion of bacteria residing in our gut (microbiome) play a critical role in maintaining our health through regulation of number of host physiological processes including neurodevelopment. Alteration in gut microbiome (dysbiosis) had been linked with number of diseases including neurological disorders such as Alzheimer disease (AD) and Multiple Sclerosis (MS). Gut dysbiosis lead to the development and exacerbations of various neurological disorders due to the possible modulation in bidirectional communication between gut bacteria/bacterial metabolites and the CNS (microbiota–gut–brain axis). The mechanisms through which gut dysbiosis and alterations in microbiota–gut–brain axis can predispose individuals to neurological disorders are being actively pursued by researchers across the globe. A better understanding of the mechanism through which gut microbiota regulate gut-brain axis will help in harnessing enormous potential of gut microbiota as potential diagnostic and therapeutic agents. To illustrate this important topic Sangram Sisodia, University of Chicago, Javier Ochoa-Repáraz, Boise State University, Helen Vuong), University of Minnesota, and Ashutosh Mangalam, University of Iowa will present work on the role of gut microbiome in the nervous system development and pathobiology of various neurological diseases including AD and MS.

TIME	DETAILS	DESCRIPTION
10:15am - 12:15pm	**S02 - Brain Immunology and Glia** **Thoroughbred 1** **Session Chair:** Michael R. Nichols S02.01 - Glia Shape Developmental and Pathological Plasticity Sarah Ackerman S02.02 - Microglio-Vascular Interactions in Health and Disease Ukpong Eyo S02.03 - Neuron-Glial and Glial Glial Interactions in Development, Health, and Disease Jaeda Coutinho-Budd S02.04 - Neuro-Immune Interactions in the Meninges Felipe Almeida de Pinho Ribeiro	This session will cover physiological and pathological aspects of brain immunology and glia. Speakers come from the Centers for Brain Immunology and Glia (BIG) at Washington University in St. Louis and University of Virginia. They include Sarah Ackerman (glial plasticity), Ukpong Eyo (microglia-vascular interactions), Jaeda Coutinho-Budd (neuron-glial interactions) and Felipe Almeida de Pinho Ribeiro (meninges neuro-immune interactions).
10:15am - 12:15pm	**S03 - In Memoriam Dr. Robert K. Yu** **Thoroughbred 2** **Session Chair:** Thomas Seyfried S03.01 - A Long, Eventful Journey With a Dear Friend; a Former Student Who Became a Teacher (Recorded Presentation) Robert Ledeen S03.02 - Is Ganglioside GD3 Linked to Fermentation Energy Thomas Seyfried S03.03 - From Gangliosides to Ceramide and Back: The Power of Sphingolipids in Neural Development and Disease Erhard Bieberich S03.04 - GM1 Ganglioside: A Multiplicity of Functions Makes It a Potentially Ideal Therapeutic for Parkinson's Disease Jay Schneider	Dr. Robert K. Yu (Bob) passed away on May 18 2022 after a long struggle with Parkinson's disease. Bob was a Professor in the Dept. of Neuroscience and Regenerative Medicine, Medical College of Georgia, Augusta University and was the 17th President of the ASN. Bob's research mainly involved the neurochemistry of glycosphingolipids in health and disease. Bob was a leader in isolation and characterization of glycolipids in the nervous system. His research groups from the Yale University School of Medicine, the Medical College of Virginia, and Medical College of Georgia has characterized about 1/3 of all glycolipids in the nervous system. These structures included all of the "c-series" gangliosides (known as the A2B5 antigens) in fish and embryonic mammalian brains, and other bioactive glycolipids, such as the sulfoglucuronosyl glycolipids that are known as markers for neural stem/progenitor cells and human natural killer cells (HNK-1 glycolipid antigens). More recently, his laboratory had focused on characterizing stage-specific glycosphingolipids in neural stem cells with the goal of elucidating the relationship between glycosphingolipid expression and cell fate determination during development and disease. Bob left behind not only his loved-personal family but also his academic family of former students, postdocs, collaborators, and teachers. All of the graduates from Bob Yu's Neurochemistry Lab in the all over the world are missing him very much. This symposium is dedicated to the memory of Dr. Robert K. Yu (1938–2022) who was a giant in ganglioside science, a prominent neurochemist, mentor, colleague, friend, husband, father, grandfather, and student.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S04 - Mitochondrial Perspectives of Neurodegenerative Diseases** **Regency Ballroom** **Session Chairs:** Brad Hubbard Samir Patel S04.01 - Mitochondria and Mitochondrial Cascades in Alzheimer's Disease Russell Swerdlow S04.02 - Mitoceutical and Mitochondrial Delivery Approaches to Combat Energy Depletion After Spinal Cord Injury Alexander Rabchevsky S04.03 - The ATP Synthase C-Subunit Leak Channel Regulates Cellular Metabolism in Neurodevelopment Elizabeth Jonas S04.04 - Too Much, Too Little, Too Late; the Importance of Therapeutic Windows of Opportunity for Targeting Mitochondria After Brain Injury Patrick Sullivan	This session will highlight two of the priority areas for the conference, including "Neurodegenerative Disease" and "Metabolism, Cellular and Molecular Neurobiology." This session brings together world-renowned researchers from across the nation while also highlights the research strengths of the local affiliate institution, the University of Kentucky. This session is focused on mitochondrial biochemistry in the CNS and understanding how function changes neurodegenerative disease. Our various speakers will pinpoint unique underpinnings of cellular mitochondrial activity and bioenergetics in the CNS. Speakers in this session will also highlight mitochondrial regulation and how to target bioenergetic alteration to improve outcomes. Insights into these mechanisms will be ascertained from foundational work in the areas of traumatic brain injury, spinal cord injury, fragile X syndrome, and Alzheimer's disease. This session is chaired by early career investigators and contains confirmed speakers.
1:15pm - 3:15pm	**S05 - Rebuilding the Brain After Injury** **Thoroughbred 1** **Session Chairs:** Cesar V. Borlongan S05.01 - Targeting G-Protein Pathways for Tissue Repair Following Stroke Sharon Owino S05.02 - Interrogating the Neurovascular Unit with Stem Cells Cesar V. Borlongan S05.03 - Oligodendrocyte Precursor Cell Transplantation for Brain Repair Yongting Wang S05.04 - Multi-Modal Functional Mapping of Brain Injury and Recovery Xin Yu	Stroke remains a significant scientific and medical challenge, with many gaps in knowledge. This session is designed to provide a multi-disciplinary survey of recent advances in the field of neurorecovery after stroke, comprising the latest findings in molecular biology, neurovascular unit science, glial biology, and in vivo imaging. The speakers have all contributed to recent primary research papers in these areas. Dr. Owino recently identified the diverse and complex roles of G-protein pathways in regulating various cellular and molecular mechanisms of neurogenesis and neuroplasticity. Dr. Borlongan's long-standing interest in stem cells has been extended to probing the impairment of the neurovascular unit after stroke and its repair with exogenous stem cell transplantation and mobilization of endogenous stem cells. Dr. Wang has led efforts to translate oligodendrocyte precursor cell transplantation as a therapeutic approach for enhancing oligodendrogenesis, in order to promote functional recovery after cerebral ischemia. Dr. Yu is a leader in the development of multi-modal functional imaging platforms for brain injury and recovery, which may provide clinically feasible and direct measures of brain function in animal models and humans.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S06 - Astrocyte Functional Heterogeneity in Neural Circuits and Behaviors** **Thoroughbred 2** **Session Chairs:** Xinzhu Yu Margaret Ho S06.01 - The Roles of Astrocyte Calcium Signaling in the Prefrontal Circuit and Anxiety-Like Behavior Xinzhu Yu S06.02 - Glia-Mb Acetylcholine-Mediated Synaptic Connections Underlie Drosophila Long Term Memory Margaret Ho S06.03 - A Role for Astrocytes in Disordered Breathing In Rett Syndrome Michelle Olsen S06.04 - Mapping the Contribution of Astrocytes to the Effects of Neuromodulators on Synapses and Behavior Thomas Papouin	As the most numerous glial cell types in the central nervous system, astrocytes closely interact with neurons, blood vessels and other glial cells, and contribute to essential physiological processes. Recent evidence has revealed a functional heterogeneity of astrocytic molecular responses that are specific to experimental challenges and neural circuits where they reside. However, major open questions concerning how astrocytes regulate neural circuit functions and behaviors that are relevant in disease and whether the same or different astrocytic mechanisms are employed remain to be addressed. In this Colloquium, we will share recent discoveries and unpublished data that support context- and circuit-specific mechanisms by astrocytes to modulate synaptic transmission, neuromodulation, respiratory control, memory formation, and emotion. These exciting findings further indicate the possibility that astrocyte biology could be leveraged to ameliorate the symptoms of patients suffering from neuropsychiatric and neurodevelopmental diseases. Specifically, Dr. Yu will focus on astrocyte calcium-dependent signaling and reveal its novel roles in regulating specific neuronal projections in the prefrontal circuit and anxiety-like behavior. Dr. Ho will present a novel mechanism mediated by astrocytic acetylcholine receptors in synaptic transmission and long-term memory formation. Dr. Olsen will present evidence that brainstem astrocyte dysfunction contributes to disordered breathing in a preclinical mouse model of Rett Syndrome. Dr. Papouin will discuss how well-documented effects of neuromodulators on neuronal circuits are, in fact, entirely implemented by astrocytes and how astrocyte-based neuromodulation is involved in cognitive functions known to be altered in schizophrenia.
4:00pm - 5:30pm	**C01 - Ferroptosis in Neurodegenerative Diseases** **Regency Ballroom** **Session Chairs:** Wenzhang Wang Pamela Maher C01.01 - Using the Oxytosis Ferroptosis Pathway to Understand and Treat Age-Associated Neurodegenerative Diseases Pamela Maher C01.02 - Targeting Ferroptosis to Mitigate Neurodegenerative Disease Qitao Ran C01.03 - Ferroptosis as a Mechanism for Neurodegeneration in Alzheimer's Disease Scott Ayton C01.04 - Ferroptosis Underlying Neurodegeneration With Brain Iron Accumulation Wenzhang Wang	Ferroptosis is a novel mechanism of regulated cell death that selectively removes dysfunctional cells caused by iron overload related to lipid peroxidation of cellular membranes, alongside impaired antioxidant capacity. Given that ferroptosis is intensively being implicated in the pathogenesis of a wide range of neurodegenerative diseases, our speakers represent its intriguing roles in rare familial forms of neurodegeneration that occur early in life as well as its more common role in aging related neurodegeneration. Dr. Pamela Maher is an early investigator who explores the oxytosis/ferroptosis pathway, and she will discuss recent progress using small molecules to manipulate the ferroptosis pathway in age related neurodegeneration processes. Dr. Qitao Ran's talk will explore the well-established ferroptosis pathway in the pathogenesis of Alzheimer's disease, which is the most common neurodegenerative disease in the country. Dr. Scott Ayton will focus on the intriguing role of abnormal iron regulation and related alterations of ferroptosis mechanisms in Parkinson's disease and Alzheimer's disease. Dr. Wenzhang Wang will discuss the contribution of ferroptosis mechanism in the pathogenesis of mitochondrial protein-associated neurodegeneration, a genetic disease characterized as massive iron accumulation in the brain of young cases. In summary, these interesting topics will cover both the basic mechanisms and therapeutic translational opportunities by focusing on the ferroptosis pathway in neurodegenerative diseases.

TIME	DETAILS	DESCRIPTION
4:00pm - 5:30pm	**C02 - History, Theory, Applications, Use, and Misuse of the G-Ratio** **Thoroughbred 1** **Session Chairs:** Doug Feinstein Anne Boullerne C02.01 - History of the G-Ratio and Early Measurements Across Species and Evolution Anne Boullerne C02.02 - Statistical Considerations to Interpret G-Ratios in Disease States Alexander Gow C02.03 - The Efficacy of G Ratio as an Indicator of Myelination Jeff Dupree C02.04 - Are There Alternatives for Measuring Myelin Thickness? Wendy Macklin	The g-ratio, being the ratio of axonal diameter to fiber thickness, has been used for over 50 years to provide an assessment of axonal health. Changes in the g-ratio occur during demyelinating and remyelinating events, and have been used to monitor efficacy of remyelinating therapies. However, while widely used, there is a large disparity in the methods used to measure parameters, in data presentation and analysis, and in the interpretation of what those changes reflect. In this session, the 4 speakers will address these topics. Dr Boullerne will provide an overview of the origins of the g-ratio, its theoretical basis, and give examples of measurements made across species, brain areas, during development, aging, and in human demyelinating diseases. Dr Dupree will discuss if and how changes in g-ratios reflect functional outcomes. Dr Gow will discuss some of the theoretical aspects underlying the importance of the g-ratio, relationships between fiber diameter and conduction velocity, and a statistical framework for detecting and interpreting myelin pathology. Dr Bouhrara will discuss recent methodologies to measure g-ratio in human subjects using MRI and DTI imaging protocols during aging and neurodegeneration.
4:00pm - 5:30pm	**C03 - Glycolipids in Neurobiology and Neurodegenerative Disease** **Thoroughbred 2** **Session Chair:** Yutaka Itokazu C03.01 - Gangliosides: Old Dogs With New Tricks Simonetta Sipione C03.02 - Inhibition of Glucosylceramide Synthase Modulates Disease Phenotypes in Preclinical Models of Neurodegeneration James Dodge C03.03 - Glycolipids of Neural Stem Cells: Fountain of the Youth Yutaka Itokazu	Patients with central nervous system CNS disorders suffer from impaired cognitive, sensory, and motor functions that can have adverse effects on daily life activities and create challenges for caregivers. With accumulating evidence, it has become increasingly clear that glycosphingolipids (GSLs) play important roles in CNS function and repair. GSLs are abundant in the CNS and are known to play essential roles in cell-cell recognition, adhesion, signal transduction, immunomodulation, and cellular migration, as well as other important functions that are crucial to CNS development and neurogenesis and later to CNS maintenance and function. Despite intense investigations into CNS repair mechanisms, the roles of GSLs in this process remain unclear. Here we will focus on the potential roles of glycolipids in disease pathogenesis, disease progression, and CNS repair. Mice lacking glucosylceramide, lactosylceramide or gangliosides show significant structural and functional CNS pathology. More importantly, patients with GSL deficiencies exhibit severe clinical phenotypes. Neurodegenerative diseases and mental health disorders are associated with altered GSL expression. Accumulating studies demonstrate that GSLs play central roles in the maintenance and biological functions of neurons and glia. We will discuss the ways in which proper GSL composition modulates behavior of a variety of molecules leading to development and potentially the treatment of CNS disorders. GSL-replacement therapy and normalization of GSL profiles could be a paradigm-shift in the approach to treating CNS disorders.

MONDAY MARCH 20, 2023

TIME	DETAILS	DESCRIPTION
8:30am - 9:30am	**Plenary Session 2 – Neurogenesis and Circuit Remodeling in the Adult and Aging Hippocampus** **Regency Ballroom** Alejandro Schinder	
10:15am - 12:15pm	**S07 - The Emerging Transcriptome in Brain Health and Disease** **Regency Ballroom** **Session Chairs:** Ashutosh Dharap Suresh Mehta S07.01 - Alzheimer's Risk Factor BIN1 Regulates Pathology-Specific Microglial Activation Aryeh Sudwarts S07.02 - Regulation and Function of Microexons in Specific Neuronal Cell Types Adam Norris S07.03 - Noncoding RNA Crosstalk and Post-stroke Outcome Suresh Mehta S07.04 - Conditional Expression of miR-20a-3p in Neurons vs Astrocytes and Its Impact on Acute Stroke Outcomes Taylor Branyan	The advent of deep sequencing has provided insights into the mammalian transcriptome at an unprecedented depth, revealing sequences, structures, expression patterns, and gene networks that had escaped prior detection using conventional methods. Some of these discoveries include new classes of RNAs such as circular RNA (circRNA) and enhancer RNA (eRNA), and novel mRNA splice variants produced by context-dependent alternative splicing events. In the CNS, such events at the transcriptional and post-transcriptional levels have been tightly linked to neural state and physiology, and their dysregulation has been linked to brain injury and disease. This session is centered around the latest advances in brain transcriptomics with the goal of highlighting the roles of the newly discovered RNAs and RNA processing mechanisms in the maintenance (and disruption) of biochemical, physiological, and cellular processes in the healthy and diseased brain. The panel of speakers is comprised of experts in neuroscience and transcriptomics, specifically circRNA biology (Dr. Suresh Mehta; published the first study on circRNAs in stroke), eRNA biology (Dr. Nancy Carullo; pioneered the study of eRNAs in neurons), and alternative splicing (Dr. Chaolin Zhang and Dr. Adam Norris; at the forefront of research on neural-specific alternative splicing and RNA regulatory networks).

TIME	DETAILS	DESCRIPTION
10:15am - 12:15pm	**S08 - Myelin Disruption in Diverse Neurological Disorders** **Thoroughbred 1** **Session Chairs:** Jayshree Samanta S08.01 - Finding the Missing Link Between Disrupted Myelin and Abnormal Learning in RASopathies Alejandro Lopez-Juarez S08.02 - TPPP Forms Liquid Condensates and Aggregates in Multiple System Atrophy Meng-meng Fu S08.03 - Enhancing Remyelination by Endogenous Neural Stem Cells in Multiple Sclerosis Jayshree Samanta S08.04 - APOE4 Impairs Cholesterol Trafficking and Myelination Joel Blanchard	Myelin is disrupted in many neurological diseases but the mechanisms of disruption and the cellular consequences vary in each disease. This session will focus on myelin injury in three different neurological diseases- RASopathy, Multiple System Atrophy and Multiple Sclerosis. First, Alejandro Lopez-Juarez will discuss the mechanisms of myelin disruption in two RASopathy models, - mice carrying mutations known to cause Neurofibromatosis Type 1 and Costello Syndrome - and the resulting behavioral abnormalities. Next, Meng-meng Fu will elucidate the mechanisms of oligodendroglial protein aggregates in Multiple System Atrophy, a neurodegenerative disorder characterized by widespread demyelination and cytoplasmic alpha-synuclein inclusions in oligodendrocytes. Finally, Jayshree Samanta and Kaylene Young will discuss how myelin loss in Multiple Sclerosis affects neural stem cells and vascular cells using mouse models and induced pluripotent stem cells derived from human patients.
10:15am - 12:15pm	**S09 - Bringing the Bench to the Bedside: Translational Use of Patient Care Opportunities for Real-Time Research in Stroke** **Thoroughbred 2** **Session Chair:** Keith Pennypacker Amanda Trout S09.01 - Integration of AI Technology and Translational Bench Techniques to Evaluate Stroke Managements and Outcomes Justin Fraser S09.02 - Cellular Analysis of Intracranial and Systemic Blood to Uncover Immune Response in Patients During a Stroke Ann Stowe S09.03 - Computational Analysis of Proteomics and Transcriptomics of Blood From Stroke Patients for Prognostic Biomarkers and Therapeutic Targets Keith Pennypacker S09.04 - Identification of EVs in Intracranial and Systemic Blood From Patients Undergoing Thrombectomy Amanda Trout	The University of Kentucky's Center for Advanced Translational Stroke Science has been enrolling patients with emergent large vessel occlusion (ELVO) who undergo mechanical thrombectomy for stroke into a prospective tissue bank, where we collect the arterial blood both proximally in the neck, and distally in the brain blood (prior to reopening of the vessel). The bank also records all relevant demographic, clinical and radiographic data about each subject (with an independent neuroradiologist performing radiographic assessments). We have utilized machine learning and advanced statistical modeling to identify cellular and molecular biomarkers that predict clinical outcomes and are potential therapeutic targets for adjuvant treatments administered with thrombectomy. Our transcriptomic and proteomic analyses have yielded the first insight into the inflammatory signaling that occurs in the intracranial and systemic blood in a human during a stroke. Keith Pennypacker, PhD will speak of the ongoing studies in the identification of predictive transcriptomic and proteomic biomarkers for clinical outcomes. These biomarkers are also potential pharmacological targets for adjuvant treatments. Dr. Ann Stowe, PhD will present the immune cellular response in patients during ELVO and the identification of immune cell types that predict infarct volume and edema. Dr. Justin Fraser, a neurointerventionalist, will present the clinical view of this project to improve patient management and outcomes. Amanda Trout, PhD will present her current study examining the presence of extracellular vesicles in the intracranial and systemic blood of these patients and how these vesicles can provide insight into novel therapeutic approaches to stroke.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S10 - Contributions of Protein Kinases to Axonal Pathology and Neurodegenerative Disease Pathogenesis** Supported by the Basic Neurochemistry text **Regency Ballroom** **Session Chairs:** Gerardo Morfini Douglas Feinstein S10.01 - p38 MAPK Contributions to the Pathogenesis of Amyotrophic Lateral Sclerosis Gerardo Morfini S10.02 - Gender-Specific Beneficial Effects of Genetic JNK3 Deletion in the Eae Mouse Model of Multiple Sclerosis Douglas Feinstein S10.03 - Regulation of Neuronal Connectivity by Axonal Stress Signaling Catherine Collins S10.04 - Tau Misfolding and Aberrant Kinase Signaling in Alzheimer's Disease (Ad) and ADRDs Yuyu Song	Aberrant protein phosphorylation, abnormal activation of protein kinases and axonal pathology represent well-established pathogenic hallmarks of a wide variety of human neurodegenerative conditions, including Alzheimer's disease, multiple sclerosis, and most motor neuron diseases. Despite this knowledge, mechanisms linking neuropathogenic proteins associated with these diseases to activation of selected kinases remains unknown, as is the identity and relevance of specific substrates targeted. Further, the overall contribution of such kinases to axonal pathology has only recently started to be addressed. This symposium will feature work aiming to provide information that fills these gaps in our knowledge of neurodegenerative disease pathogenesis.
1:15pm - 3:15pm	**S11 - Inflammatory Mechanisms of Neurodegeneration** **Thoroughbred 1** **Session Chair:** Tara Desilva Nikhil Panicker S11.01 - Microglia-Mediated Mechanisms of Degeneration and Regeneration Tara DeSilva S11.02 - Microglial Asc Contributes to Neuronal Alpha-Synuclein Aggregation in Dementia With Lewy Bodies Nikhil Panicker S11.03 - Syk Is a Pivotal Regulator of Neuroprotective Microglial Responses in Neurodegenerative Disease John Lukens S11.04 - Inflammasome Regulation and Novel Therapeutic Targeting in Alzheimer's Disease Bradlee Heckmann	Dr. Tara M. DeSilva, associate professor, Cleveland Clinic will chair this session along with co-chair, Dr. Nikhil Panicker, a new assistant professor at the Cleveland Clinic. This session will also include seminars from Dr. John Lukens, associate professor at the University of Virginia and Dr. Bradlee Heckmann, assistant professor at the University of South Florida. The goal of this symposium is to explore how microglial inflammatory signaling in the settings of adaptive and innate immune responses contribute to neuronal dysfunction in Alzheimer's, Dementia with Lewy Bodies, and autoimmune demyelinating diseases. The central theme will encompass pivotal roles that microglia serve in both protective and degenerative responses. This will include microglia-mediated responses to axonal injury and synaptic dysfunction, progressive protein aggregation in Dementia with Lewy bodies, neuroprotective signaling marshalled by the tyrosine kinase SYK, and LC3-Associated Endocytosis (LANDO) signaling in inflammasome assembly. These studies will explore the spatio-temporal roles of microglia in the pathophysiology of diverse neurodegenerative diseases.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S12 - Glial Mechanisms and Neurodegeneration** **Thoroughbred 2** **Session Chair:** Leonor Pérez-Martínez S12.01 - Modulation of Tissue Injury and Repair by Microglia During Autoimmunity Astrid Cardona S12.02 - Aging Affects Myeloid Cell Responses That Mediate Repair After Spinal Cord Injury Jasmin Herz S12.03 - Rare Astrocyte Subtypes in Neurodegeneration and Neuroinflammation Philip Hasel S12.04 - Glial Interactions Regulating White Matter Health Veronique Miron	In recent years, neuroinflammation has been suggested as an important contributor for neurodegeneration. Glial cells play a pivotal role in the homeostasis of the CNS. However, under pathological conditions, both microglia- and astrocyte-derived proinflammatory mediators contribute to the progression of a number of neurodegenerative diseases. This point out the importance to understand the biology of non-neuronal cells under disease conditions. The proposed symposium will assemble the leaders in the field to discuss the contribution of glial cells to neurodegeneration. This symposium may open new avenues to identify new targets to address neurodegenerative diseases.
4:00pm - 5:30pm	**C04 - What Models of Restless Legs Syndrome Have Taught Us About Interactions of Genetics, Iron Metabolism and Neurochemistry in the Brain** **Regency Ballroom** **Session Chairs:** James Connor Christopher Earley C04.01 - Validation of the Dietary Iron-Deficiency Rodent Model as a Model for Rls Pathology Christopher Earley C04.02 - Bxd Ri Mouse Model – Exploring Factors That Define Brain Iron Homeostasis and Their Relevance of Rls Byron Jones C04.03 - Blood-Brain-Barrier – How We Learned About Iron Regulation From Rls James Connor C04.04 - Dietary Iron-Deficiency Rodent Model – Exploring the Consequence on Non-Dopaminergic Systems Sergi Ferre	Brain iron insufficiency is considered one of the primary underlying pathological elements in restless leg syndrome (RLS). Therefore, understanding the consequences of brain iron insufficiency may provide insight into the system-dynamics of neurotransmitter states in RLS. The present symposium will review the science behind the dietary-iron-deficiency rodent model that provides validity of this model for exploring the consequences of iron insufficiency as it pertains to RLS. Data will be presented utilizing this model to provide insight into the dynamic relationship between neurotransmitters that occurs as a consequence of brain iron insufficiency and provide insight into potential non-dopaminergic pathways that may underlie RLS. The BXD recombinant inbred mice provides a rodent model in which one can explore diverse and dynamic genetic factors that regulate iron homeostasis under normal and iron deprived conditions. Data will be presented on genetic factors that tie in to known RLS risk genes and their relation to brain iron homeostasis as well as providing insight into new systems that may be involved. Finally, one of the most important influencers of brain iron regulation is the blood-brain-barrier. So blood-brain-barrier models can provide cellular details of what is behind brain iron homeostasis that may be pertinent to understanding iron dysregulation in RLS.

TIME	DETAILS	DESCRIPTION
4:00pm - 5:30pm	**C05 - Novel Astrocyte Mechanisms of Epileptogenesis** **Thoroughbred 1** **Session Chairs:** Devin Binder Seema Tiwari-Woodruff C05.01 - Mechanisms of Demyelination-Associated Seizures Seema Tiwari-Woodruff C05.02 - Mechanisms of Demyelination-Associated Seizures Devin Binder C05.03 - Altered Astrocytic Transport in Tumor-Associated and Posttraumatic Epilepsy Stefanie Robel C05.04 - Impairment of Astrocyte Glutamine Synthetase Activity in Epilepsy Tore Eid	Astrocytes play an established role in removal of glutamate at synapses and the sequestration and redistribution of potassium and water during neural activity. It is becoming increasingly clear that changes in astrocyte channels, transporters, and metabolism play a direct role in seizure susceptibility and the development of epilepsy. This emerging concept of epilepsy as an "astrocytopathy" will be explored in this session. Each of the proposed speakers has contributed unique recent publications to the field identifying astrocytic changes in animal models of epilepsy. Seema Tiwari-Woodruff has identified novel astrocyte mechanisms in demyelination-associated seizures. Devin Binder has identified reduced astrocyte glutamate uptake as a contributor to epilepsy and that increasing astrocyte glutamate uptake may ameliorate seizures and neurotoxicity in epilepsy. Stefanie Robel has identified novel mechanisms of astrocyte changes in tumor-associated epilepsy and posttraumatic epilepsy, suggesting new therapeutic targets. Tore Eid has identified alterations in the astrocyte enzyme glutamine synthetase in both animal models of epilepsy and in human epilepsy tissue samples. Altogether, this session will provide the ASN community with an update on the role of astrocyte mechanisms in diverse conditions associated with seizures and epilepsy.
4:00pm - 5:30pm	**C06 - Remyelination Therapies: What Is Holding Us Back and Can We Fix It?** **Thoroughbred 2** **Session Chairs:** Karen Chandross Stefanie Giera C06.01 - Mechanisms of Remyelination in Mouse and Human, Where Do We Stand? Gonçalo Castelo-Branco C06.02 - Animal Models of Myelin Repair: What Can They Teach Us About Ms? Jeffrey Huang C06.03 - Finding the Human in Mice Stefanie Giera C06.04 - Imaging Biomarkers of Myelin Repair: Challenges and Opportunities Richard Dortch	Myelin loss is a hallmark of Multiple Sclerosis (MS) pathophysiology that leads to irreversible neuronal loss and functional deficits. It has been well demonstrated that remyelination, or myelin repair, can occur in MS lesions and animal models and this process can be enhanced by pharmacological means. Early drug discovery technologies and assays have been used successfully to identify new therapeutic candidates that modulate signaling pathways involved in driving oligodendrocyte progenitor (OPC) maturation and remyelination. Inducers of OPC maturation and myelination have been identified from both large-scale OPC phenotypic screens and target-based approaches, which have led to promising clinical development candidates. However, to date there are no successful myelin repair drugs on the market. Major hurdles include major translational challenges when going from mouse to human. For example, researchers rely heavily on rodent cells and models to achieve proof of concept, including experimental autoimmune encephalomyelitis, cuprizone and lysolecithin. However, these models do not fully recapitulate the complex human disease and the field lacks definitive non-invasive biomarkers of drug effects on myelin repair and the link to improved neuronal function. Recent advances in human stem cell biology, imaging endpoints and AI tools hold promise towards developing more meaningful therapeutic targets and pathways. This panel session will summarize the key drug discovery challenges to advancing myelin repair therapies and then facilitate coming together as a cross-disciplinary community to prioritize solutions through a dynamic panel discussion that includes audience participation.

TUESDAY MARCH 21, 2023

TIME	DETAILS	DESCRIPTION
8:30am - 9:30am	**Plenary Session 3 - How and Why We Sleep: Insights From a Small Animal Model** **Regency Ballroom** Amita Sehgal	
10:15am - 12:15pm	**S13 - Biofluid and Neuroimaging Biomarkers As Translational Tools for Animal Models of Traumatic Brain Injury and Therapeutic Monitoring** **Regency Ballroom** **Session Chairs:** Kevin Wang Kobaissy Firas S13.01 - Brain Milieu Based Biofluid-Biomarkers for Traumatic Brain Injury and Their Theranostic Utilities Kevin Wang S13.02 - Structural and Functional Neuroimaging in Different Models of Traumatic Brain Injuries Marcelo Febo S13.03 - Combining MRI and MRS Biomarker and Blood-Based Biomarkers for Mouse Model of Traumatic Brain Injury and Post-Traumatic Epilepsy Elisa Zanier S13.04 - Biofluid–And Neuroimaging Biomarkers in the Study of Gyrencephalic Model of TBI and Their Theragnostic Potential Todd Kilbaugh	In recent years, there are increasing momentums of incorporating blood-based and neuroimaging biomarkers for both observational and interventional clinical traumatic brain injury (TBI) studies. This includes the recent FDA clearance of a rapid blood test that measures neuronal UCH-L1 protein and astroglial GFAP protein to aid in the diagnosis of TBI severity in the emergency medicine setting. Biofluid and MRI biomarkers for neurotrauma can provide advance insights in underlying pathophysiological mechanisms as well as disease progress, recovery and response to therapeutic intervention. On the other hand, animal models of TBI can be quite heterogenous. For example, there are a wide range of models (such as controlled cortical impact/CCI, fluid percussion injury/FPI, accelerated weight drop/AWD) , animal species (rat, mouse, pig, and ferret) as well as variability of injury level and assessment methods across research centers and laboratories. Although neurobehavioral and neuropathology endpoints will remain important, we should take advantage of our increasing knowledge and capabilities of measuring biofluid-based biomarkers that can interrogate neuronal injury, astroglia damage or changes, axonal injury and neurodegeneration. Similarly advances in neuroimaging in animals with high power magnetic (7-11Tesla) are allowing one to examine contusion, diffuse axonal injury and fiber tract changes, microhemorrhage as well as functional connectivity with high resolution. Thus, the adoption and validation of key biofluid and MRI biomarkers can allow them serve as novel endpoints that can translated from rodent and large gyrencephalic animals to human studies. Furthermore, these biomarker tools can also help guide the much-needed new therapeutic development for TBI.

TIME	DETAILS	DESCRIPTION
10:15am - 12:15pm	**S14 - Novel Therapeutics for Stroke** **Thoroughbred 1** **Session Chairs:** Bharath Chelluboina Selva Baltan S14.01 - Fasudil, Mifepristone and Interleukin-1 Alpha As Novel Stroke Therapies Gregory Bix S14.02 - Contribution of System Xc- to Cerebral Ischemic Injury Sandra Hewett S14.03 - Multipronged Approaches to Targeting Neuroinflammation and Blood-Brain Barrier Damage in Ischemic Stroke Eduardo Candelario-Jalil S14.04 - Tenascin-C as a New Therapeutic Target for Stroke Bharath Chelluboina	Despite the immeasurable burden of stroke, no effective therapeutics are yet available to protect the brain. As stroke risk increases with age and in the presence of comorbidities, therapeutic target screening needs a rigorous assessment for pharmacokinetic and dynamic aspects in the presence of biological variables such as age, sex, and comorbidities. Therefore, in this session, Drs. Chelluboina (Chair) and Baltan (Co-chair) will introduce the highlights of the recent advances in new therapeutic targets to treat stroke. Speaker 1 (Dr. Bix) will discuss the new neuroprotective targets of the extracellular matrix. Speaker 2 (Dr. Hewett) will discuss the significance of targeting cystine/glutamate antiporters to protect the brain after a stroke. Speaker 3 (Dr. Candelario-Jalil) will discuss the recent advances in bromodomain-containing protein 4, a member of the bromodomain and extra-terminal domain protein as a potential stroke therapeutic target. Speaker 4 (Dr. Chelluboina) will discuss the significance of targeting tenascin-c to protect the post-stroke brain. Overall, we present and discuss 4 potential new therapeutic avenues for stroke.
10:15am - 12:15pm	**S15 - Cerebrovascular Continuum From Brain Development to Neurological Disorders** **Thoroughbred 2** **Session Chairs:** Vanessa Coelho-Santos Baptiste Lacoste S15.01 - Does a Recapitulation of Developmental Mechanisms Impair Neurovascular Coupling in Disease? Anusha Mishra S15.02 - Neutrophils Contributing to Brain Blood Flow Reductions in Dementia Oliver Bracko S15.03 - Neurovascular and Metabolic Deficiencies in Autism Baptiste Lacoste S15.04 - A Window With a View to Cerebrovascular Development Vanessa Coelho-Santos	Cerebrovascular health is critical for a properly developing and functioning nervous system. All aspects of the neurovasculature are tightly regulated in the healthy brain, including developmental processes as angiogenesis and pruning, structures like the blood-brain barrier, and coupling between blood flow and neural activity. The brain vasculature has indeed been strongly associated with the onset of conditions associated with aging, and more recently with neurodevelopmental disorders. Where mechanisms governing cerebrovascular structure and function can become dysfunctional, with profound implications on cognitive abilities. Can the early vascular impairments have impact on the expression of neurodegenerative diseases? This symposium brings together a diverse group of early/mid-career scientists covering topics ranging from neurovascular development to its potential link to neurological disease in later life. Dr.Coelho-Santos will portray the cerebrovasculature development using advanced imaging techniques in mouse models. Dr.Lacoste will describe how neurovascular dysfunction could be involved in neurodevelopment disorders namely autism. Dr.Bracko will describe how immune cells in the vasculature contributes to pathology in neurodegenerative diseases using in vivo microscopy. Finally, Dr.Mishra will talk about how recruitment of developmental signaling pathways impairs neurovascular coupling dynamics in neurodegenerative disease states like stroke and dementia.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S16 - CNS Precursor Cells: New Players in Neuroinflammation?** **Regency Ballroom** **Session Chairs:** Anastassia Voronova Alex Nicaise S16.01 - Oligodendroglia in Development and Multiple Sclerosis: Insights From Single Cell and Spatial Omics Gonçalo Castelo-Branco S16.02 - Neural Stem Cells As Novel Drivers of Smouldering Brain Disease Alex Nicaise S16.03 - A Key Role for Proliferating NG2+ Cells in Scar Formation After Spinal Cord Injury Christina Marion S16.04 - Chemokine Regulation of CNS Progenitors in the Developing and Remyelinating Brain Anastassia Voronova	CNS precursor cells, like neural stem and precursor cells (NPCs) and oligodendrocyte progenitor cells (OPCs), build and regenerate the brain and spinal cord by differentiating into specialized CNS cell types. However, recent studies have revealed that the role of NPCs and OPCs extends beyond CNS cell replacement. Specifically, a growing number of reports demonstrate that CNS precursor cells directly regulate neuroinflammation in mouse models of neurodevelopmental and neurodegenerative disorders (Marteyn et al. 2016 Stem Cells; Falcão et al. 2018 Nat Med; Kirby et al. 2019 Nat Comm; Willis et al. 2022 Exp Neurol). This symposium will cover this novel and emerging role of CNS precursor cells. Presentations will focus on neurochemical mechanisms of 1) epigenomic priming of immune genes in OPCs and their contribution to multiple sclerosis (Dr. Castelo-Branco); 2) regulation of microglia and macrophage function via extracellular vesicles secreted by NPCs (Dr. Pluchino); 3) contribution of OPCs to glial scar formation in spinal cord injury (Dr. McTigue); and 4) chemokine-mediated regulation of OPCs and NPCs in the developing and demyelinated brain (Dr. Voronova). We believe this symposium will be of great interest to neurochemists in the fields of neuroinflammation, demyelination, MS, spinal cord injury, neurodevelopmental disorders, as well as neural stem cell and OPC biology and therapeutics.
1:15pm - 3:15pm	**S17 - Astrocytes and Microglia in Neural Repair: New Insights Across Model Organisms and Injury Types** **Thoroughbred 1** **Session Chairs:** Meifan Chen Ukpong Eyo S17.01 - Capacity of Astrocytes to Support Axon Growth in an Injured CNS Meifan Chen S17.02 - Pro-regenerative Glial Functions During Natural Spinal Cord Repair Mayssa Mokalled S17.03 - Dissecting Reactive Astrocyte Roles in Neural Repair Joshua Burda S17.04 - Reprogramming Microglia in CNS Remyelination Jeffrey Huang	This proposed session showcases innovative research by four Assistant Professors, who are emerging leaders in the field of glial mechanisms of injury. They include Dr. Meifan Chen (University of Kentucky), Mayssa Mokalled (University of Washington), Joshua Burda (Cedars-Sinai Medical Center), and Dr. Jeffrey Huang (Georgetown University). This session was put together with diversity in mind, in terms of research topics, speakers' gender, race, and institutional affiliation. All speakers have been confirmed. Talks will provide new insights into the beneficial roles of astrocytes and microglia in CNS repair, based on research spanning model organisms from mouse to zebra fish; methodologies from classical genetic screening, novel genetic models, to single cell RNA sequencing; and injury types from spinal cord injury to epilepsy. The breadth, depth, and conceptual/technological innovations of the work by these rising investigators is expected to have a wide appeal to the attendees of ASN meeting.

TIME	DETAILS	DESCRIPTION
1:15pm - 3:15pm	**S18 - Innovative Cellular Models and Experimental Approaches to Probe Mechanisms and Therapies for Gulf War Illness** **Thoroughbred 2** **Session Chairs:** Liang Oscar Qiang Kimberly Sullivan S18.01 - Axonal Transport-Based Therapeutic Strategies for Gulf War Illness Alvin Terry S18.02 - Improving Brain Function in Gulf War Illness Through Modulation of Leukotriene Signaling Ashok Shetty S18.03 - Cytoskeletal Mechanisms of Cognitive Deficits in Gulf War Illness Peter Baas S18.04 - Advanced Machine Learning for Constructing and Comparing Single-Cell Gene Regulatory Networks in Neural Organoids of Gulf War Illness James Cai	Gulf War Illness (GWI) is a chronic multisystem disorder best understood in the context of neurochemistry. GWI is suffered by at least 25% of the nearly 700,000 U.S. veterans who fought in the 1990-1991 Gulf War. Central nervous system CNS symptoms include chronic fatigue, reduced information processing speeds, memory deficits, chronic headaches and impaired mood and sleep. Evidence suggests that GWI is caused by exposure to a cluster of neurochemical toxicants, including organophosphates, reversible and irreversible acetylcholinesterase inhibitors, insecticides and burning oil smoke. A constellation of cellular and neurochemical changes in the CNS contribute to the long-lasting symptoms of GWI. No effective therapies are available to treat the afflicted veterans, due in part to insufficient knowledge of the neurochemical pathways affected in the disease. To address the gaps in knowledge, our panel speakers have performed high throughput cellular analyses, screened potential therapies that can be rushed to suffering veterans and investigated potential susceptibility. Our four speakers will present results on deficits in neuronal microtubules, axonal transport, aberrant phosphorylation of tau, neuroinflammation and transcriptome analyses at the single-cell level. Model systems include veteran-derived forebrain organoids.
4:00pm - 5:30pm	**C07 - Developing Therapeutic Strategies for Injury or Disease of the Nervous System: The Role of TNFR2** **Regency Ballroom** **Session Chair:** Haritha Desu Kayla Nguyen C07.01 - TNFR2 Regulates the Immunomodulatory Properties of Oligodendrocytes in Neuroimmune Disease Haritha Desu C07.02 - TNFR2 Activation and Sex-Specific Functional Recovery in a Model of Multiple Sclerosis Kayla Nguyen C07.03 - TNFR2 Activation as a Therapy for Alzheimer's Disease Uli Eisel C07.04 - TNFR2 Activation as a Therapy for Chronic Neuropathic Pain John Bethea	The session will explore new advances in understanding mechanisms and developing therapies for chronic neurodegenerative disorders such as multiple sclerosis, Alzheimer's disease, and chronic neuropathic pain. The collective focus of our work ism on developing a more thorough understanding of how TNFR2 signaling is therapeutic in different neurodegenerative disorders. Our session chairs, Drs. Brambilla and Nguyen will discuss recent findings on TNFR2 signaling in oligodendrocytes and EAE, respectively. Whereas Drs. Eisel and Bethea will discuss recent advance in Alzheimer's disease and chronic pain, respectively.

TIME	DETAILS	DESCRIPTION
4:00pm - 5:30pm	**OR1 - Oral Presentation Session 1** **Thoroughbred 1** **Session Chairs:** Farida Sohrabji OR01.01 - Functional Characterization of DNA Methylation in the Transcriptional Regulation of Glutamate Glial Transporters in Excitotoxicity Conditions Jaqueline Loaeza OR01.02 - The Lysosomal Cation TRPML1 Regulates the Oligodendrocyte Actin Cytoskeleton Via Rac1 Activation Lindsay Festa OR01.03 - Age-Related Decrease in Brain Endothelial Cell Response to WNT/β-Catenin Signaling Unnderlies Vulnerability to NeuroCOVID KaReisha Robinson OR01.04 - Vulnerability of the Blood-Brain Barrier to Focal Stroke in a Mouse Model of Autism Pavel Kotchetkov OR01.05 - Microglial Ablation During Recovery From MS-like Injury is Associated with Deficits in Oligodendrocyte Differentiation and Quality of Remyelination Ex Vivo Andrew Lapato OR01.06 - A Novel Method for Isolation of Extracellular Vesicles (EVs) From Brain Tissues Using Gentle Proteolytic Dissociation and Membrane Affinity Capture Ahmed Elsherbini	
4:00pm - 5:30pm	**OR2 - Oral Presentation Session 2** **Thoroughbred 2** **Session Chairs:** Anusha Mishra OR02.01 - Traumatic Brain Injury Induces Transient Intestinal Permeability, Increased Colon Hypoxia, and Subacute Microbiome Changes in Mice Anthony DeSana OR02.02 - Post-ischemic Protection of Subcortical White Matter Following a Selective Ischemic Attack Hung Nguyen	

TIME	DETAILS	DESCRIPTION
	OR02.03 - Micro-RNA-22 Acts as an Inhibitor of Remyelination Adya Sapra OR02.04 - Cognitive Impairment After Stroke is Linked to Gut Repair: Divergent Effects of Central and Systemic IGF-1 Treatment Yumna El-Hakim OR02.05 - Alterations in the Transcriptome and DNA Methylome of the Magnocellular Neurosecretory System May Underlie the Age-dependent Loss of Neuronal Plasticity Derick Thompson OR02.06 - Alzheimer's Disease like Neuropathology in Down Syndrome Cortical Organoids Helen Zhao	
5:30pm - 7:00pm	**ASN 2023 Business Meeting** Supported by sanofi **Regency Ballroom**	

WEDNESDAY MARCH 22, 2023

TIME	DETAILS	DESCRIPTION
8:30am - 9:30am	**Plenary Session 4 - PL.04 - Replacing Dying Oligodendrocytes One Cell at a Time** **Regency Ballroom** Robert Hill	
10:00am - 12:00pm	**S19 - Multifaceted Roles of Astrocyte Signaling in Alzheimer's Disease (AD) and Related Dementias (ADRDs)** **Regency Ballroom** **Session Chairs:** Chris Norris Erica M. Weekman S19.01 - Brain Insulin Sensitivity, Role of Astrocytes in Health and Disease Olivier Thibault S19.02 - Thyroid Hormone, Astrocytes, and Adrd in Mouse Models and Humans Dana Niedowicz S19.03 - MMP9 and Astrocyte End-Feet Degeneration in Vcid Donna Wilcock S19.04 - Reactive Astrocytes: A Critical Contribution to Synaptic and Neurovascular Dysfunction in Ad and ADRDs Pradoldej Sompol	Astrocytes can exhibit profound molecular, biochemical, and morphologic changes in diseased brain. Collectively, these changes are widely thought to reflect a "reactive" phenotype. Though conceptually complex, astrocyte reactivity is most commonly studied as a critical partner of microglia in the generation and maintenance of neuroinflammation. However, astrocytes also play essential roles in the regulation of cerebral blood flow, brain metabolism, and synaptic maintenance and/or function— all things that are compromised in Alzheimer's disease (AD) and AD-related dementias (ADRD)s. Each of our speakers are part of a collaborative effort at the University of Kentucky Sanders-Brown Center on Aging to move beyond the conceptual constraints of neuroinflammation to elucidate the multifaceted role(s) of astrocytes in AD and ADRDs. Astrocyte molecular pathways including those associated with glutamate transport, insulin signaling, K-ATP channels, and endfeet integrity are under investigation using multiple mouse models of AD/ADRDs, as well as human source material. Astrocyte signaling, cerebrovascular function and neural activity are evaluated (often in awake, behaving mice) using two photon imaging and other physiologic approaches. The goal of this symposium is to raise awareness of astrocyte signaling (or dysfunction) as a cellular mechanism for pathophysiology and as a possible drug target for treating multiple forms of dementia.

TIME	DETAILS	DESCRIPTION
10:00am - 12:00pm	**S20 - Regulation of Neuroinflammation by Glial Cell Death Signaling** **Thoroughbred 1** **Session Chairs:** Brian Daniels Jessica Williams S20.01 - RIPK3 Signaling Promotes Neurotoxic Astrocyte Activation and Parkinsonian Neurodegeneration Brian Daniels S20.02 - Astrocyte Interferon-Gamma Dampens Inflammation During CNS Autoimmunity via Pd-1/Pd-L1 Signaling Jessica Williams S20.03 - AIM2 Inflammasome Activation in Astrocytes During Eae Mari Shinohara S20.04 - Microglia Iron Overload Causes Ferroptosis and Neurodegeneration Tim Hammond	Our session will focus on exciting new work defining specialized roles for programmed cell death (PCD) signaling in the CNS, with a particular emphasis on PCD signaling in glial cells. PCD in the CNS presents unique risks compared to other tissues. Structural and biochemical features of the CNS limit its capacity for repair; thus, the immunologic benefits of PCD must be weighed against the cost of losing irreplaceable cells and critical cytoarchitecture. This set of talks will explore the unique ways in which glial cells employ cell death signaling to regulate neuroinflammation, resulting in both protective and pathologic outcomes. These outcomes are elicited both by traditional cell death modalities, as well as by cell-death independent adaptations to traditional PCD signaling pathways that are unique to the CNS. Our laboratories are pursuing these studies using a variety of models of neurologic disease, including Parkinson's disease, Alzheimer's disease, and multiple sclerosis.
10:00am - 12:00pm	**S21 - Cell Swelling and Changes in Extracellular Space as Drivers of Brain Pathologies** **Thoroughbred 2** **Session Chairs:** Alexander Mongin Sergei A. Kirov S21.01 - Non-excitatory Amino Acid Transporters Drive Astroglial Swelling and Amplify Hypoxic and Ischemic Neuronal Injury Sergei Kirov S21.02 - Neuronal Edema and Control of Network Excitability in Traumatic Brain Injury Punam Sawant-pokam S21.03 - Cell Volume-Sensitive Cation-Chloride Transporters and WNK-SPAK/OSR1 Protein Kinases As Therapeutic Targets in Stroke Dandan Sun S21.04 - Seizures and Adolescent Mortality in Animals With Deletion of the Volume-Regulated Anion Channel Protein LRRC8A Alexander Mongin	Within the brain, the dynamic regulation of cellular volume and extracellular space represents a critical, yet underappreciated aspect of brain physiology and pathophysiology. Several major neural disorders—such as stroke, traumatic brain injury, and epilepsy—are associated with profound cellular swelling and reciprocal reductions in the extracellular space. Even minute changes in these compartments strongly impact transmembrane ionic gradients, concentrations of extracellular signaling molecules, and neuronal excitability. Cellular swelling is thought to promote neuronal excitability through at least two independent mechanisms: (1) release of excitatory neurotransmitters via volume-regulated anion channels (VRACs), and (2) reductions in the volume and diffusion parameters of the extracellular space. The causal relationship between cellular swelling and neural pathologies is now being actively explored by many groups in the field. The proposed symposium includes a diverse group of speakers who will highlight emerging findings and perspectives in this rapidly developing area of neuroscience and neurochemistry.

TIME	DETAILS	DESCRIPTION
1:00pm - 3:00pm	**S22 - Extracellular Vesicles in Stroke and Neurodegeneration** **Regency Ballroom** **Session Chairs:** Julie Saugstad Ursula Sandau S22.01 - Small Extracellular Vesicles Harvested From Cerebral Endothelial Cells for the Treatment of Stroke and Diabetes-Associated Dementia Li Zhang S22.02 - Identification of Neuron-Specific Extracellular Vesicle Markers Dima Ter-Ovanesyan S22.03 - Identification of Neural Cell Type-Specific Molecules in the Extracellular Vesicles Enriched From Human Cerebrospinal Fluid and Their Application to Alzheimer's Disease Tsuneya Ikezu S22.04 - Effects of Apoe Genotype and Sex on Human Cerebrospinal Fluid Extracellular Vesicle Cargo Ursula Sandau	Extracellular vesicles are membrane-bound spheres that carry complex cargos, including lipids, proteins, and nucleic acids. Release of extracellular vesicles from cells is a universally conserved process that occurs in all eukaryotes and prokaryotes, and in every biofluid examined, thus extracellular vesicles serve as mediators of specific cell-to-cell communication. Cells produce different classes of extracellular vesicles based on their approximate size distribution, as well as their mode of release from cells, cargo, and extracellular vesicle surface markers that indicate their respective biogenesis routes. For example, CD9, CD63, and CD81 are canonical tetraspanins associated with exosomes. extracellular vesicles are of great interest for their potential as biomarkers of and therapeutics for central nervous system disorders, including stroke and Alzheimer's disease. The speakers in this session will provide an overview of the current state of extracellular vesicle studies in stroke and neurodegeneration. Dr. Chopp will discuss exosome-based approaches to improve recovery after stroke in human patients, Dr. Ter-Ovanesyan will discuss the quest for specific surface markers for neuronal extracellular vesicles, Dr. Ikezu will discuss markers of human brain cell derived extracellular vesicles in Alzheimer's disease, and Dr. Sandau will discuss differential effects of sex and APOE-e4 genotype on Alzheimer's disease extracellular vesicles miRNA cargo that may account for increased prevalence of Alzheimer's disease in females and those that carry the APOE-e4 genotype.
1:00pm - 3:00pm	**S23 - Quantitative Neuroimaging of Degeneration and Repair: The Brain and Beyond** **Thoroughbred 1** **Session Chairs:** Richard Dortch Jun Li S23.01 - Clinical Imaging of Neurodegeneration Francesca Bagnato S23.02 - Application of Stage Imaging in Neurodegenerative Diseases Ewart Mark Haacke S23.03 - Investigating the Relationship Between Perfusion and White Matter Damage/Repair Ashley Stokes S23.04 - Imaging Neurodegeneration in Peripheral Neuropathies Yongsheng Chen	Conventional MRI techniques are exquisitely sensitive to neurodegenerative pathologies, including de/remyelination, inflammation/edema, gliosis, and axonal loss; however, they lack specificity. As a result, numerous quantitative MRI methods have been developed to derive tissue-specific indices with improved pathological specificity, with the goal of improving diagnostic quality and/or our ability to monitor disease progression and treatment response. These quantitative MRI techniques are increasingly pursued as non-invasive biomarkers of neurodegeneration and repair, which has been made possible by several recent advancements. First, diffusion MRI methods have been combined with biophysical models to probe microanatomical tissue features (e.g., axonal packing) at sub-voxel scales, providing information that was previously only available via invasive histopathology. Second, modern hardware and acquisition methods have significantly improved efficiency, allowing for bench-to-bedside translation of quantitative MRI methods throughout the human central and peripheral nervous system. Finally, multi-parametric approaches have been pursued to evaluate the complex pathological substrates (e.g., inflammation, perfusion, and iron) that accompany (and often drive) microanatomical changes. In this session, we will review recent advances in quantitative MRI methods and discuss their ability to provide biomarkers of neurodegeneration and repair. Methods from across the development spectrum (from advanced preclinical tools to practical clinical tools) will be discussed. In addition, the emergence of methods for neurodegenerative disorders beyond the brain (e.g., peripheral neuropathies) will be highlighted. Attendees will gain a broad understanding of the current and emerging quantitative MRI tools available for neurodegenerative disease and how these might be applied in both preclinical and clinical settings.

TIME	DETAILS	DESCRIPTION
1:00pm - 3:00pm	**S24 - New Mechanisms and Therapies for Ischemia-Related Brain Injuries** **Thoroughbred 2** **Session Chairs:** Raghu Vemuganti Ann Stowe S24.01 - The Role of Astrocyte-Secreted Chordin-Like 1 in Synaptic Plasticity After Stroke Elena Blanco-Suarez S24.02 - Hemoglobin-Mediated Brain Injury in Neonatal Intraventricular Hemorrhage Brandon Miller S24.03 - Circulating Estrogen Promotes Recovery of Hippocampal Synaptic Plasticity Following Cardiac Arrest in Juveniles Nidia Quillinan S24.04 - Kinase Regulation in Cerebral Ischemia Reggie Lee	Ischemia-related brain injuries are major contributors to both developmental delays in children, as well as vascular dementia in adults. Several of these injury mechanisms are conserved across the lifespan and have potential as therapeutic targets, as outlined in this session. First, the Blanco-Suarez lab is interested in targeting astrocytes to improve post-stroke plasticity and functional recovery. Dr. Blanco-Suarez will be presenting their latest research on the role of the astrocyte-secreted protein Chordin-like 1, and its potential injurious role limiting post-stroke plasticity and motor recovery. Second, the Miller lab investigates injury mechanisms that contribute to neurodevelopmental delays secondary to intraventricular hemorrhage (IVH) and hydrocephalus. Dr. Miller will discuss current drugs that improve mitochondrial bioenergetics to reduce inflammation and brain injury after neonatal IVH. Third, the Quillinan lab studies excitability and plasticity changes in the brain following cerebral ischemia. Dr. Quillinan will discuss hippocampal plasticity impairments following ischemic brain injury that contribute to long-term memory deficits. This includes the contribution of endogenous sex hormones to spontaneous recovery of hippocampal plasticity that is unique to the developing brain. Finally, the Lee lab research focus is to understand the mechanisms of novel kinases that cause brain injury after cerebral ischemia. Since the FDA has approved over 46 kinase-related drugs for the treatment of various diseases, Dr. Lee will discuss translation into human clinical trials for the patients suffering from ischemic brain injury. These presentations will highlight several novel mechanisms of ischemia-related brain injury that could be developed into neurotherapeutics to address long-term brain pathology.
3:30pm - 5:00pm	**C08 - Astrocyte-neuron Interactions** **Regency Ballroom** **Session Chair:** Anna Yu-Szu Huang C08.01 - Striatal Astrocyte-Neuron Mechanisms and Signaling Drive OCD Behaviors Baljit Khakh C08.02 - Perineuronal Nets and the Tripartite Synapse Harald Sontheimer C08.03 - Astrocyte Cell-Cell Communication in Health and Disease Francisco Quintana	Dr. Baljit S. Khakh is one of the pioneers who established the functional roles of astrocyte Ca2+ activity in the regulation of neuronal circuits and has established an array of tools to probe astrocyte functionality that have benefited the astrocyte community to this day. Dr. Harald Sontheimer is the expert in astrocytes' involvement in epilepsy and their interaction with perineuronal net. Dr. Francisco J. Quintana specializes in neuroinflammation and has discovered fundamental mechanisms by which astrocytes regulate neuronal function during inflammation. The talks of the three speakers will complement each other and encompass astrocyte-neurons interactions in physiological state, during development, and in pathological conditions.

TIME	DETAILS	DESCRIPTION
3:30pm - 5:00pm	**C09 - Emerging Roles for Glial and Neuronal Dysmaturation in Developmental Brain Injury** **Thoroughbred 1** **Session Chairs:** Stephen Back Taasin Srivastava C09.01 - Beyond Cell Death: Hypoxemia-Mediated Neuronal Dysmaturation Stephen Back C09.02 - Hyaluronan-Mediated Opc Dysmaturation in White Matter Injury Taasin Srivastava C09.03 - Inflammation-Mediated Glial and Neuronal Dysmaturation Justin Dean	Preterm infants acquire life-long neurodevelopmental disabilities that involve various forms of injury to cerebral white and gray matter that result in life-long motor and cognitive disabilities. This session will explore the emerging role of dysmaturation of neurons and oligodendrocyte progenitors (OPCs) in persistent disturbances in brain development. The session will introduce key features of dysmaturation and the conditions that trigger it. Dr. Back will discuss recent findings from studies in preterm fetal sheep and a new neonatal murine hypoxia model that found that a brief mild exposure to hypoxia alone persistently disrupts fetal or neonatal cortical and hippocampal neuronal complexity, synaptic activity, gene expression and behavior without triggering cell death or inflammation. OPC dysmaturation is characterized by arrested maturation of late OPCs, as an injury response to white matter injury (e.g. GLIA 2014;62:1790–1815). Dr. Srivastava will discuss mechanisms of OPC dysmaturation that involve a novel hyaluronan-dependent immune tolerance-like pathway that disrupts OPC lineage progression and myelination via TLR4/AKT/FoxO3-dependent signaling. Dr. Dean will discuss unexpected roles for mild prolonged inflammatory insults in disruption of oligodendrocyte maturation, myelination, neuronal dendritogenesis and microstructural developmental of cerebral white matter and neocortex as defined by advanced high field ex vivo imaging in neonatal rats. This session will thus provide novel insights into the unique susceptibility of the developing brain to milder insults at critical windows in preterm brain development. These insults do not cause pronounced cell death, but result in diffuse persistent disturbances in glial and neuronal maturation.
3:30pm - 5:00pm	**C10 - Glial Influence in Neurodegeneration: A Focus on Non-cell Autonomous Toxicity** **Thoroughbred 2** **Session Chairs:** Brigid Jensen Elena Blanco-Suarez C10.01 - Modeling Non-cell Autonomous Toxicity in Als: Targeting Astrocyte-Secreted TNFα as a Potential Therapeutic Candidate Brigid Jensen C10.02 - Protective Functions of Astrocytic GDF15, Impacts for Parkinson's Disease Jingli Cai C10.03 - The Role of profilin1 (PFN1) in Altering Microglial Responses and Neuronal Viability in Als/Ftd Daryl Bosco C10.04 - HIV-Induced Neuroinflammation Causes Deficits in Oligodendrocyte Maturation Through Glutamate Signaling and the Integrated Stress Response Judy Grinspan	In many neurodegenerative conditions, research has been initially focused on the cell autonomous effects of mutant proteins in neurons and consequential toxicity that results. However, in recent years it has also come to light that many conditions also display features of non-cell autonomous toxicity, where mutant or aberrant protein expression in glial cell populations also independently contribute to disease through various mechanisms which ultimately culminate in neuronal damage and death. This has been shown in many disease states and in injury. This session will highlight the contributions of astrocytes, microglia, macrophages and oligodendrocytes in driving neurodegeneration, and consider several models by which we can better understand glial roles in disease and potentially test new therapeutic pathways. Our speakers represent a range of backgrounds and academic levels and are all successful and dynamic women in science. Dr. Jensen will describe a novel model of non-cell autonomous mechanisms in vivo, identifying TNF-alpha as a toxic astrocyte-secreted molecule in FUS-ALS. Next, Dr. Blanco-Suarez will discuss how astrocytes via Chrdl1 secretion may worsen stroke outcomes. Dr. Bosco will then explain how microglial deficits in lipid processing impacts phagocytic function in ALS and frontotemporal dementia. Finally, Dr. Grinspan will describe how activation of monocyte-derived macrophages infected with HIV impairs oligodendrocyte maturation, through the integrated stress response.

PROGRAM
POSTER LISTING

P01 - BUILDING THE NERVOUS SYSTEM

P01.01 Endothelial Cells Regulate Astrocyte to Neural Progenitor Cell Trans-Differentiation in Stroke
Wenlu Li

P01.02 Iron Deficiency Alters the Development of Human Ventral Forebrain Organoids
Garrick Salois

P01.03 Late-Term Maternal ZIKV Infection Impacts Fetal Brain Development
Tsui-Wen Chou

P01.04 Mecp2-Null Astrocytic Factors Lead to Formation of Precocious and Biochemically Distinct Perineuronal Nets on Cortical Neurons
Jessica MacDonald

P01.05 Use Themogenetic Technologies to Manipulate the Electrical Properties of OPCs During the Postnatal Development of the Mouse Brain
Veronica Cheli

P01.06 Combined DMH1 and Neural Stem Cells Treatment Improved Ischemic Stroke Outcome
Lei Chen

P01.07 Meningeal Lymphatics Developmental Dynamics and its Involvement in Neurodevelopmental Disorders
Gabriel Tavares

P01.08 Precursors for a Subpopulation of Cortical Astrocytes Migrate from the Subventricular Zone Postnatally
Zila Martinez-Lozada

P02 - GLIAL MECHANISMS AND INJURY

P02.01 A Hystological and Immunochemical Comparison of Intact and Injured Human Nerve Tissues in Vitro and in Vivo
Gabriela Aparicio

P02.02 A Simplified In Vitro Platform to Study Myelin Gene Expression in Human Peripheral Glial Cells
Gabriela Aparicio

P02.03 Roles of Calpains In Peripheral Nerve Development and Injury
John Miller

P02.04 Conserved Astrogliosis During Chronic Toxoplasma Infection and Methamphetamine Exposure Reveal Reactive Astrocyte Function
Zoe Figueroa

P02.05 Defining the Role of Motor and B-Raf Signaling in Oligodendrocytes of the Brain and Spinal Cord
Divyangi Kantak

P02.06 Lipocalin Prostaglandin D Synthase Modulates Central Nervous System Myelination and Mitochondrial Oxidative Stress
Takese Mckenzie

P02.07 The Sphingosine-1-Phosphate Receptor 1 Antagonist Ponesimod Reduces Tlr4-Induced Neuroinflammation and Increases Aβ Clearance
Zhihui Zhu

P02.08 Calcineurin-Mediated Dephosphorylation of Connexin 43 In Astrocytes: Implications for Hemichannel Function in the Progression of Alzheimer's Disease
Susan Kraner

P02.09 Microglial P2ry12-Dependent Regulation of Astrocytic Features
Aida Lopez

P02.10 Changes in the Transcriptome of Hypothalamic Astrocytes may be Associated with Maturational Loss of Regeneration in the Magnocellular Neurosecretory System
Abiodun Odufuwa

P02.11 Serotonin Supplementation and Respiratory Motor-Based Optimization of Intermittent Hypoxia Procedure Improve Expression of Long-Term Facilitation in Adult Rats
Aaron Silverstein

P02.12 Mild Traumatic Brain Injury Results in Acute Microhemorrhage and Modifications in Cerebral Artery Territories in Mice
Dominic Nthenge

P02.13 Deletion of Microglial Na/H Exchanger 1 Increases Microglia Subgroup with Elevated Creb Signaling Phenotypes and Post-Stroke Myelination
Shanshan Song

P02.14 Loss of Ataxia-Telangiectasia Mutated Exacerbates Motor Coordination Defects in Atm Knockout Mice Following Oxidative Insult
Maleelo Shamambo

P02.15 Insights into the Function of Interleukin-1 In Regulating Neuroinflammatory Gene Expression Following a Closed-Head Traumatic Brain Injury in Mice
Jonathan Vincent

P02.16 Early Loss of Astrocyte P38alpha Mapk has Sexually Dimorphic Impacts on Measures of Neuronal Excitability and Cytokines in Aged Mice
John Gant

P02.17 Does a Closed Head Injury Early in Life Have Lasting Effect on Microglia in an Alzheimer's Disease-Relevant Mouse?
Elika Moallem

P02.18 Interferon-Gamma Regulates Expression of the Transcription Factor Batf2 in Astrocytes to Modulate Ap-1 Induced Neuroinflammation
Rachel Tinkey

P02.19 Astrocyte-Interneuron Interactions in the Striatum
Anna Yu-Szu Huang

P02.20 Changes to Astrocyte Gap Junction Channel and Channel-Independent Functions in Response to Inflammation Using Super-Resolution Microscopy
Randy Stout

P02.21 P2ry12 Regulates Microglial Density Independent of Sex and Transcriptional Profile in a Sex-Specific Manner
Akhabue Kenneth Okojie

P02.22 Overexpression of Astrocytic Glutamate Transporters Alleviates Hyperexcitability in Mouse Model of Alzheimer's Disease
Jenna Gollihue

P02.23 Leucine Zipper-Bearing Kinase (Lzk) Modulates Dynamicity of Cytoskeleton and Migration in Astrocytes
Matin Hemati-Gourabi

P02.24 Defining the Role of Il-1r1 in Endothelial to Microglia Interactions After a TBI
Lydia Sanders

P02.25 Targeting Lrp1 in a Rodent Cervical Injury Model to Promote Functional Recovery
Michael Sunshine

P02.26 Astrocyte Calcium Signaling Alterations and Neurovascular Coupling Deficits in a Diet Based Model of Small Cerebral Vessel Disease
Blaine Weiss

P02.27 Sex Differences Exist in the Dendritic Complexity of Adult Born Hippocampal Neurons
Ashley Glover

P02.28 Endothelin-1 Signaling in Subventricular Zone Regeneration After Perinatal Brain Injury
Lauren Rosko

P02.29 Therapeutic Implications of the Gut-Cns-Axis in Promoting Recovery After Cervical Spinal Cord Injury
Jessica Newton Wilson

P02.30 Transcriptome Analysis in Lumbosacral Dorsal Root Ganglia Reveals Molecular Changes Underlying Nociceptive Sensitization in Animal Models of Chronic Pelvic Pain
Sathish Kumar Yesupatham

P02.31 Long-Term Fate of Posttrauma-Born Hippocampal Neurons in the Context of TBI
Hannah Williams

P02.32 Astrocytes Promote Acute Survival of CNS Macrophages and Motor Recovery After SCI
Tuoxin Cao

P02.33 Tppp Forms Liquid Condensates and Aggregates in Multiple System Atrophy
Shahrnaz Kemal

P02.34 The Bone Transcription Factor Osterix Controls Extracellular Matrix and Node of Ranvier Related Gene Expression in Oligodendrocytes
Benayahu Elbaz

P02.35 Hiv Pre-Exposure Prophylaxis (Prep) Inhibits Oligodendrocyte Differentiation Through Lysosome Deacidification
Caela Long

P02.36 Hiv Antiretroviral Drugs Trigger Stress Granule Formation via the Integrated Stress Response in Differentiating Oligodendrocytes
Eliana Von Krusenstiern

P02.37 Mitochondria-Targeted Peptide, SS-31, Provides Neuroprotection Within In-Vitro and In-Vivo Rodent Models of Spinal Cord Injury
Baylen Ravenscraft

P02.38 Role of Sox2 in Astrocyte Maturation
Yan Wang

P02.39 The Effect of T9 Contusion Spinal Cord Injury on Colon Pathology in Mice
Ellie Sams

P02.40 Apoe4 Drives Maladaptive Heterogeneity and Immunometabolic Responses of Astrocytes
Josh Morganti

P02.41 The Role Of Astrocytes in Perineuronal Net Development
Courtney Prim

P02.42 Probiotic Treatment in Vivo After Spinal Cord Injury Enhances in Vitro Outgrowth Abilities of Neurons
Sydney Speed

P02.43 Concurrent Microrna and Flow-Cytometry Umap Analysis Identify Cell-Type Specific Correlations in the Cerebrospinal Fluid of Patients with Aneurysmal Subarachnoid Hemorrhage
Thomas Ujas

P02.44 Mechanisms of Experiential Intervention in the Recovery from Neonatal Brain Injury
Evan Goldstein

P02.45 Role of Innate and Adaptive Immune System in Neurodegeneration
Xiaoying Chen

P02.46 Aav-Mediated Gene Expression in Oligodendrocytes with Improved Cell Specificity
Nathan Zubin

P02.47 The Trauma Degradome of Gfap and Fragment Citrullination Relate to Astrocyte Pathobiology and Distinguish TBI Patients
Ina Wanner

P02.48 Multiparity Silences Microglial Response to the Demyelinating Injury of the White Matter
Abdeslam Mouihate

P02.49 Astrocyte Interferon-Gamma Dampens Inflammation During Chronic CNS Autoimmunity via Pd-1/Pd-L1 Signaling
Brandon Smith

P02.50 Inflammation as a Mediator of Dim Light at Night Deleterious Effects in a Mouse Model of Autism
Cristina Ghiani

P02.51 Sphingolipid Regulation by Ormdl3 in Oligodendrocytes the Myelinating Glial Cells of the Central Nervous System
Usha Mahawar

P02.52 Deletion of LRP1 Imparts Resilience to Oxidative Stress-Induced Mitochondrial Dysfunction in Traumatic Brain Injury
Velmurugan Gopal Viswanathan

P02.53 Chemogenetic Manipulation of Astrocyte Reactivity: Implications for Demyelinating Diseases
Christina Angeliu

P02.54 Genetic Ablation of the Glial Brain-Type Fatty Acid Binding Protein, Fabp7, Increases Seizure Threshold and Alters Activity-Dependent Gene Expression
Jason Gerstner

P02.55 Defects in Oligodendrocyte Development and ECM Homeostasis in the Movement Disorder Dystonia
Dhananjay Yellajoshyula

P02.56 Spatial and Temporal Specific Mitochondrial Dynamic Changes in Severe Controlled Cortical Impact Mouse Model of Traumatic Brain Injury
Hemendra J. Vekaria

P02.57 Effect of 6-Pentadecyl Salicylic Acid in the Uptake of Glutamine In U373mg Astrocytoma Cells
Laura Mendez

P02.58 Enolase Activation Triggers Gliosis and Neuronal Death After Spinal Cord Injury
Azizul Haque

P02.59 Microglia Dysregulation Increases the Anti-Inflammatory Astrocyte Response In Hypoxia-Induced Retinopathy
Colin Rorex

P02.60 Role of Choroid Plexus Autotaxin Secretion in Hemorrhage-Induced White Matter Injury
Alexandra Hochstetler

P02.61 Longitudinal Analysis of Social, Sensory and Motor Behaviors in System Xc-Null Mice
Carla Frare

P02.62 Tamoxifen, a Selective Estrogen Receptor Modulator, Activates Microglia and Astrocytes and Exacerbates Mitochondrial Stress After Oxygen Glucose Deprivation
Macy Zardeneta

P02.63 Models of Microglia Depletion and Replenishment Elicit Protective Effects to Alleviate Vascular and Neuronal Damage in the Diabetic Murine Retina
Kaira Church

P02.64 Molecular and Functional Dissection of Lesion-Remote Astrocyte Reactivity States Linked to Regenerative Plasticity, Neural Repair and Inflammation After CNS Injury
Sarah Mccallum

P02.65 Bdnf-Derivatized Nanospheres Carrying C3 Transferase Readily Cross a Bbb Model And Selectively Increase Neurite Outgrowth from Corticospinal Tract Neurons
Dianna Hynds

P02.66 Role of the Adaptive Immune System in Peripheral Nerve Tumor
Jay Pundavela

P02.67 Microglia in the Hippocampus of Female But Not Male Mice Exposed to Fungal Allergens Increase in Numbers and Morphologic Complexity
Taher Bhaijee

P02.68 Role of Choline Transporters Like Protein-1 in Schwann Cells
Nisha Gautam

P02.69 Microglia Enhance Neuronal Activity Through Inhibitory Shielding
Koichiro Haruwaka

P02.70 The Impact of Palmitic Acid and DHA on Cellular Stress and Metabolism in Microglia and Neurons
Michael Butler

P02.71 Temporal Relationship Between Astrocytic Volume and Seizures in an In-Vivo Generalized Seizure Model
Manolia Ghouli

P02.72 The Role of Microglia in Mediating Basal Capillary Tone in Health and Alzheimer's Disease
William Mills Iii

P02.73 Sex Differences in Cortical Astrocyte Density: A Role for Microglia?
Abigayle Hughes

P03 - METABOLISM, CELLULAR AND MOLECULAR NEUROBIOLOGY

P03.01 The Effects of Isoflurane on Cerebrovascular Dynamic During Whisker Stimulation
Pradoldej Sompol

P03.02 Sphingolipid Dysmetabolism is a Candidate Driver of Demyelination and Axonal Degeneration in Both Quaking Depleted and Injured Nerves
Joseph Barnes-Vélez

P03.03 Mitochondrial Dysfunction Following Repeated Mild Blast Traumatic Brain Injury is Attenuated by a Mild Mitochondrial Uncoupling Prodrug
Brad Hubbard

P03.04 Secreted Semaphorins are Regulators of Aging-Associated Dendritic Modifications and Cognitive Function
Jiyeon Baek

P03.05 The Effect of Postsynaptic Receptor Desensitization During Repetitive Synaptic Activation
Kaitlyn Brock

P03.06 Characterizing the Effect of 17β-Estradiol on Mitochondrial Dysfunction Before and After Severe Controlled Cortical Impact in Mice
Olivia Kalimon

P03.07 Epitranscriptomic RNA Modification M6a as a Stroke Therapeutic Target
Raghu Vemuganti

P03.08 Intranasal Hb-Egf Treatment After Neonatal Hypoxia Increases Oxidative Metabolism in the Hippocampus
Joseph Scafidi

P03.09 Neuron-Secreted Molecules Regulate SVZ Neural Stem Cell Fates
Nicole Dittmann

P03.10 Translational Control of Interleukin 6 During Endoplasmic Reticulum Stress
Claire Kisamore

P03.11 Patients Residing in Coal Producing Counties Differ in Stroke Severity and Proteomic Response Following Large Vessel Ischemic Stroke
Jacqueline Frank

P03.12 Brain Metabolic Phenotyping in Honey Bees Provides New Insights into Traumatic Brain Injury
Elisabeth Rintamaa

P03.13 Cytokine Receptor Heterogeneity of Astrocytes and Mechanisms of Transcriptional Regulation
Sarah Milne

P03.14 Acute Postnatal Increase in Interleukin-6 Modifies Synaptogenesis in a Mouse Model of ASD-Like Behavioral Phenotypes
Fernando Janczur Velloso

P03.15 Effects of Aripiprazole On Nicotine Withdrawal-Like Behaviors and Murine Neuregulin 3-Erbb4 Signaling
Emily Prantzalos

P03.16 Nicotine Withdrawal During Contextual Fear Extinction Causes Sex Specific Impacts In The Murine Dorsal and Ventral Hippocampus
Jack Keady

P03.17 Behavioral Characterization of Nhb-6012, A Functionally Selective and Potent Synthetic Mu Agonist
Kai Johnson

P03.18 Altered Pro-Opiomelanocortin (Pomc) Neuron Function Characterizes Energy Expenditure Dysfunction and Increased Body Weight Linked to Fragile X Gene Mutation
Rebecca Ruggiero-Ruff

P03.19 Astrocytic P53 Signaling Promotes An Immunometabolic-Dependent Pro-Viral Effect During Flavivirus Infection of the Central Nervous System
Juan P. Angel

P03.20 Neuroinflammation Promotes Glucose Metabolism in Regional Astrocytes
Maria Habean

P03.21 Age-Dependent Alterations in Primary Somatosensory Neuronal Calcium Network Dynamics: Relationship to Gait
Sami Case

P03.22 Alterations in Glucose Oxidative Metabolism After Traumatic Brain Injury in the Developing Brain
Regina Fernandez Fernandez

P03.23 Blocking Perception of Cigarette Odors to Improve Smoking Cessation
Evan Meredith

P03.24 An Investigation into the Peripheral Organ Metabolome Following Acute Spinal Cord Injury and Identification of Phospho-Mtor as a Treatment Target
Chase Taylor

P03.25 Stroke-Induced CCR3 Expression Associated with Delayed Cerebrovascular Microbleeds
Sydney Claypoole

P03.26 Proteins Associated with Age in Stroke Patients Undergoing Thrombectomy are Related to Functional and Cognitive Outcomes
Evan Hall

P03.27 Regional Differences in Oligodendroglial Cholesterol Acquisition and Myelin Lipid Composition
Marie Mather

P03.28 Oxidative Stress-Induced Extracellular Vesicles are Enriched with Ceramide and Neurotoxic
Zainuddin Quadri

P03.29 Tet3-Interacting Long Noncoding RNA Ak020504 Inhibition Exacerbates Ischemic Brain Injury
Vijay Arruri

P03.30 Neuronal ATP Sensitive Potassium (Katp) Channel Activity Couples Metabolism and Excitability to Affect Sleep
Nicholas Constantino

P03.31 Cellular Targets of Gestational Iron Deficiency
Margot Mayer-Proschel

P03.32 Fatty Acid Synthase Regulates Fatty Acid Remodeling in the Brain and Liver
Drew Seeger

P03.33 Amino Acid Fermentation Drives ATP Synthesis in Mouse and Human Glioblastoma Cells
Derek Lee

P03.34 Understanding How Microglial Activation and Neuroinflammation Alter Cerebral Glucose and Lactate Metabolism in Alzheimer's Disease: A Pilot Study
Ryan Pettit-Mee

P03.35 The Bioenergetic Profiles of Intraspinal Macrophages Differ Between Young and Aged Mice After Spinal Cord Injury
Reena Kumari

P03.36 Glutamate and Functional NMDA Receptors Contribute to Hippocampal Axonal Elongation Through Rac1 Activity, Modulating Actin Dynamics And Nox2-Mediated H2o2 Production
Christian Gonzalez-Billault

P03.37 In Vitro and Ex Vivo Imaging of Atp:adp Ratio in Neurons and Astrocytes Using the Percevalhr Nanosensor
Sophiya Sims

P03.38 Allergic Lung Inflammation Affects Glutamatergic Inputs in NTS in Sex-Specific Manner
Paula Da Silva Frost

P03.39 Airborne Exposure to Fungal Allergens Elicits a Greater Response than Airborne Exposure to LPS in the Brainstem of Male Mice
Sirajan Kamara

P03.40 Determining the Function of Choline Transporter-Like 1 in Oligodendrocyte Development and Myelination
Adriana Torres

P03.41 Weak Base Drugs and Lysosome Stress Responses
Jonathan Geiger

P04 - NEURODEGENERATIVE DISEASES

P04.01 Assessment of White Matter Function in Alzheimer's Disease: A Tale of Two Alzheimer's Disease Mouse Models
Sarah Zerimech

P04.02 Drα1-Mog-35-55 Alleviates Axonal Injury In Two White Matter Tracts of EAE Mouse Model
Sarah Zerimech

P04.03 Role of 5-Lipoxygenase on Glial Populations During Cuprizone-Mediated Demyelination
Marilyn Wang

P04.04 CSF1R Signaling Modulates Myeloid Subsets and Dictates CNS Lesion Localization in Experimental Autoimmune Encephalomyelitis
Marilyn Wang

P04.05 Direct Interactions Between Amyloid-Beta and The NLRP3 Inflammasome
Michael Nichols

P04.06 A Multiomics Approach to Deciphering the Cellular Responses of the Brain in the Context of Aging, Inflammation, and Alzheimer's Disease
Jessica Gebhardt

P04.07 CCR5 Inhibition Improves Spatial Learning and Memory Performance in a Non-Infectious Model of HIV-Associated Neurocognitive Disorder: Sex Difference in Outcomes
Chloe Simons

P04.08 Shear Stress Induces the Secretion of Extracellular Vesicles from Primary Cilia in Astrocytes
Priyanka Tripathi

P04.09 RIPK3 Limits Langat Virus Neuropathogenesis in a Brain Region-Dependent Manner
Marissa Lindman

P04.10 Inhibition of Endothelial Notch Signaling Preserves Cognitive Function in a Model of Familial Alzheimer's Disease
Stephanie Villa-Niemczyk

P04.11 Ergothioneine via Attenuating Neuroinflammation Ameliorates Ischemic Brain Damage and Behavioral Deficits
Teng-Nan Lin

P04.12 Sex Differences in Cognitive Deficits Induced by HIV-1 TAT: Potential Roles of Creb and Activity-Regulated Cytoskeleton (ARC)
Yun-Kyung Hahn

P04.13 Improvement of Cognition in a Canine Model of Alzheimer Disease Using FDA-Approved and Experimental Small Chemical Inhibitors of Calcineurin/NFAT Pathway
Lorena Sordo

P04.14 Sex-Based Differences in Inflammatory Biomarkers in Subjects with Cardiovascular Diseases and at Risk for Alzheimer's Disease and Related Dementias
Jenny Lutshumba

P04.15 IGFBP3: A Possible Prognostic Marker for Large Vessel Occlusion Strokes in Males
Hunter Hazelwood

P04.16 Therapeutic Targeting of the Mitochondrial Pyruvate Carrier in a Mouse Model of Amyloidosis with MSDC-0160 Demonstrates Sexual Dimorphism
Rachel D. Hendrix

P04.17 Impact of High-Low Training as a Rehabilitative Therapy in Chronic Spinal Cord Injury
Daimen Britsch

P04.18 Age and TBI: Does Age Play a Synergetic Role in Injury Outcome?
Teresa Macheda

P04.19 Regulation of Adult Neural Stem Cells by G Protein-Coupled Receptor GPR62
Elizabeth D. Clawson

P04.20 RIPK3 Signaling Reduces Neuronal Sensitivity to Excitotoxic Cell Death Following Zika Virus Infection
Irving Estevez

P04.21 Alterations of Brain Bioactive Lipids After Microglial Elimination in Animal Models for Alzheimer's Disease: A Functional Lipidomics Study
Ziying Xu

P04.22 Stroke-Induced Persistent Transcription Changes of Bilateral Choroid Plexus in Mice
Yang Chen

P04.23 Changes of Ion Transporter Signaling Pathway in Choroid Plexus Blood-Cerebrospinal Fluid Barrier Damage And Neuroinflammation in Vascular Dementia Model
Lin Lin

P04.24 Alterations in Perineuronal Nets Coincides with Matrix Metalloproteinase Upregulation in the Alzheimer Mouse Models
Lata Chaunsali

P04.25 Immuno-Transmission Electron Microscopic Study of Ad-Related Pathological Proteins in Human Brains
Wang-Xia Wang

P04.26 AKT and C-Jun N-Terminal Kinase Drive Proliferation of Patient-Derived Glioblastoma Cells via an Additive Mechanism
Antonio Fidaleo

P04.27 UMAP and Histological Analyses Reveal Age-Associated B Cell Subsets in Aged Male and Female Mice Post-Stroke
Katie Malone

P04.28 Preliminary Investigation of the Impact of Exercise and Stroke on Common Carotid Artery Compliance in the Absence of Cathepsin B
Katherine Cotter

P04.29 Regenerative Therapy to Alleviate Cognitive Decline, Synaptic Loss, and Neuroinflammation Following Cranial Radiotherapy for Brain Cancers
Munjal Acharya

P04.30 Conditional Deletion of Lrrc8a in the Mouse Brain Protects Against Stroke Damage Independently of Swelling-Activated Glutamate Release
Mustafa Balkaya

P04.31 Intraindividual Longitudinal Stability of Human Cerebrospinal Fluid Mirna Biomarkers for Alzheimer's Disease in Plasma
Julie Saugstad

P04.32 Vagus Nerve Stimulation Restricts Immunocyte Infiltration and Reduces Disease Burden in a Rat Model of Multiple Sclerosis
Chandramohan Natarajan

P04.33 Estrogen and Sigma Receptors in the Mechanism of Long-Term Brain Injury in a Mice Model of Covid-19
Michael J Paidas

P04.34 Dysregulated Neuroimmune Response in Amylin-Induced Type-2 Diabetic Neurodegeneration
Edric Winford

P04.35 HIV-1 TAT and Opioid-Induced Inflammation is Mediated b M-Opioid Receptor-Expressing Astroglia
Sara Nass

P04.36 Gulf War Veteran Derived Cerebral Organoids Display Multifaceted Pathological Defects In Gulf War Illness
Kendra Case

P04.37 A Novel In Vivo Model System Of Ms Antibody-Mediated Demyelination
Lisa Golden

P04.38 Cannabidiol (Cbd) As A Therapy For Experimental Subarachnoid Hemorrhage
Jill Roberts

P04.39 Fragile X Gene Mutations Alter Gnrh Neuron And Ovarian Innervation With Consequences on Reproductive Function
Pedro Villa

P04.40 Asymptomatic Zika Virus Infection Induces Chronic Neuroinflammation and Neurodegeneration in Immunocompetent Adults
Benjamin Buckley

P04.41 Neurotoxicity of 1-Deoxysphingolipids
Susana Munoz

P04.42 Perineuronal Net Coverage is Associated with Alterations of CA1 Interneurons in a Pilocarpine Mouse Model of Epilepsy
Annalin Woo

P04.43 T Cell Subsets Altered in a Murine Alzheimer's Model Superimposed with Latent U94
Justin Youngyunpipatkul

P04.44 Brain-Specific Suppression of AMPKβ2 Impairs Cognitive Function and Synaptic Plasticity in Mice
Nathaniel Swift

P04.45 Loss of EIF2B5 Disrupts Astrocyte Differentiation in Vanishing White Matter Disease
Chris Proschel

P04.46 High-Volume Microglia Staining in the Presence of Disease Pathology
Ryan Shahidehpour

P04.47 Glucose Transporter 1 is Affected Differentially by Alzheimer's Disease Compared to Normal Aging and Type-2 Diabetes
Steven Barger

P04.48 White Matter Injury and Vasodilator Dysfunction of Cerebral Microvessels in Alzheimer's Disease
Zsolt Bagi

P04.49 Lanthionine Ketenamine (ESTER) Phosphonate (ESTER) Stimu-Lated Autophagy Protects Against CA2+ Overload-Induced Death in Neuron-Like Cells and Cardiomyocytes
Travis Denton

P04.50 Alterations of Lipid-Mediated Mitophagy Result in Aging-Dependent Sensorimotor Defects
Natalia Oleinik

P04.51 The Role of Advanced Age in Recovery from Neurotropic Coronavirus Infection
Kristen Funk

P04.52 Progression of White Matter Atrophy Depends on the Interplay of Traumatic Brain Injury and Neuronal TAU Pathology in Transgenic Mice
Regina Armstrong

P04.53 Voltage-Gated Calcium Entry in Astrocytes Promotes Astrocyte Reactivity, Spinal Cord Inflammation and Demyelination in the EAE Model of MS
Pablo Paez

P04.54 Early Loss of Microglial P38α Mapk in APPSWE/PS1DE9 Mice Increases Soluble Amyloid-Β and Reduces Microglia-Plaque Co-Localization Without Exacerbating Amyloid-Associated Pathology
Hilaree Frazier

P04.55 Glutamate Uptake and Release in Young and Aged Mice
Brittany Sulkowski

P04.56 The Roles of Astrocytic Nampt in Mediating Astrocyte Metabolism and Neuronal Survival After Ischemic Stroke
Zhe Zhang

P04.57 Impact of NMN Supplementation on Motor and NMJ Function In SOD1G93A ALS Mice
Samuel Lundt

P04.58 Chronic NSMASE2 Inhibition Suppresses Neuronal Exosome Spreading In-Vivo & Induces Sex-Specific Attenuation of AD Pathology in Appnl-F/Nl-F Knock-In Mice
Francesca Mowry

P04.59 Selectively Targeting TAU Through the Pathological PAD Domain in AD
Raghd Nowar

P04.60 The Effect of C9ORF72-Associated Dipeptide Repeat Proteins on Mammalian Motor Neurons
Sarah Martin

P04.61 Nutrient Sensing Receptor GPRC6a Hemizygous Deletion in PS19 Mice Mitigates TAU Neuropathology and Plays Regulatory Role in Autophagy
Kelsey Campbell

P04.62 Calpain Regulation of Degeneration of Substantia Nigra Neurons in the Rotenone Rat Model of Parkinson's Disease
Narendra Banik

P04.63 Dietary Interventions Improve Sleep/Wake Disturbances Aswell as Motor Symptoms in the Bachd Mouse Model of Huntington's Disease
Chris Colwell

P04.64 Microglial Micrornas are Essential for Successful Remyelination
Nagendra Rai

P04.65 Age-Related Decrease in Brain Endothelial Cell Response to WNT/B-Catenin Signaling Underlies Vulnerability to Neurocovid
Sarah Lutz

P04.66 Amyloid Precursor Protein Inhibits Ceramide-Mediated Mitophagy in Alzheimer's Disease
Chase Walton

P04.67 Novel Estrogen Receptor B Ligand Treatment Improves Visual Dysfunction in a Model of Chronic Experimental Autoimmune Encephalomyelitis
Micah Feri

P04.68 Mechanisms of Rna-Binding Protein TDP-43 Mediated Regulation of Neuronal DNA Mismatch Repair and its Implications in the Pathogenesis of ALS
Vincent Provasek

P04.69 Exploring Microglial Response to a Viral Infection-Induced Inflammation as a Process Underlying Cognitive Decline and Neurodegeneration in Aged C57BL6/J Mice
Eva Simoncicova

P04.70 Docosahexaenoic Acid-Induced Autophagy Protects Nerve Growth Factor Differentiated PC12 Cells from Hypoxia-Induced Cell Death
Manuel Montero

P04.71 Region-Specific Neuronal Activity in a Demyelinating Brain Could Lead to Hyperexcitability And Seizures
Moyinoluwa Ajayi

P04.72 Qualitative and Quantitative Analysis of Serum and Cerebro-Spinal Fluid Circular Cell Free DNA of Parkinson's Disease Patients
Małgorzata Wojtkowska

P04.73 Altered Hypothalamic Transcriptome in a Mouse Model of Multiple Sclerosis
Jonathan Carver

P04.74 The Effect of Dolutegravir on Oligodendrocyte Development
Melanie Cruz-Berrios

P04.75 Dysregulated Cholinergic Homeostasis Alters Oligodendrocyte Maturation Upon Demyelination
Farah Gadelkarim

P04.76 Novel, Interesting, Surprising and Useful Properties of a Panel of NF-L, NF-M and NF-H Monoclonal Antibodies
Gerry Shaw

P04.77 Trem2-Deficiency Hastens and Exacerbates Age-Related Myelin Degeneration via Galectin-3
Tyler Mccray

P04.78 Electronic Cigarette Exposure Exacerbates Ischemic Stroke Outcome via Altered Neurotransmitter Metabolism in Rats of Both Sexes
Hari Pradhyumnan

CONGRATULATIONS

ASN 2023 AWARDS

JORDI FOLCH-PI AWARD

The Jordi Folch-Pi Award is given to an outstanding young investigator who has demonstrated a high level of research competence and originality, who has significantly advanced our knowledge of neurochemistry and who shows a high degree of potential for future accomplishments.

Recipient: **Jaeda Countinho-Budd**

MARIAN KIES AWARD

The Marian Kies Memorial Award is given to a junior scientist for outstanding research conducted during graduate training.

Recipient: **Lauren Rosko**

YOUNG INVESTIGATOR EDUCATIONAL ENHANCEMENT (YIEE) TRAINEE AWARDS

Recipients:
Joseph Barnes-Velez,
Pedro Villa,
Stephanie Villa-Niemczyk,
Takese McKenzie,
Nicole Dittman,
Chloe Simons
Jiyeon Baek,
Brandon Smith,
Sara Nass
Rachel Hendrix

YOUNG LATIN AMERICAN SCHOLAR (YLAS) TRAINEE AWARD

Recipient: **Laura Mendez**

ASN NEURO TRAINEE AWARD

Recipient: **Alexandra Hochstetler**

SANOFI TRAVEL AWARD

Recipient: **Hung Nguyen**

ASN 2023
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EXHIBITS & POSTER HALL HOURS

Location: Patterson Ballroom

Saturday, March 18	5:30pm – 7:00pm **(Welcome Reception)**
Sunday, March 19	9:30am – 5:30pm
	5:30pm – 7:00pm **(Poster Reception)**
Monday, March 20	9:30am – 7:00pm
Tuesday, March 21	8:30am – 12:15pm
	12:15pm – 5:30pm **(Posters Only)**

REGISTRATION DESK HOURS

Location: Hyatt Regency Hotel Lobby

Saturday, March 18	2:00pm - 7:00pm
Sunday, March 19	7:00am - 5:00pm
Monday, March 20	8:00am - 4:30pm
Tuesday, March 21	8:00am - 4:30pm
Wednesday, March 22	8:00am - 4:00pm

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The official language of the ASN 2023 Meeting is English. All sessions will be conducted in English.

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REFRESHMENT BREAKS

MARCH 19 – MARCH 21

Location: Patterson Ballroom, Exhibit & Poster Hall

Morning Refreshment Break	9:30am – 10:15am
Lunch Break (on own)	12:15pm – 1:15pm
Afternoon Refreshment Break	3:15pm – 4:00pm

MARCH 22

Location: Regency Ballroom Foyer

Morning Refreshment Break	9:30am – 10:00am
Lunch Break (on own)	12:00pm – 1:00pm
Afternoon Refreshment Break	3:00pm – 3:30pm

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ASN 2023
GENERAL INFORMATION

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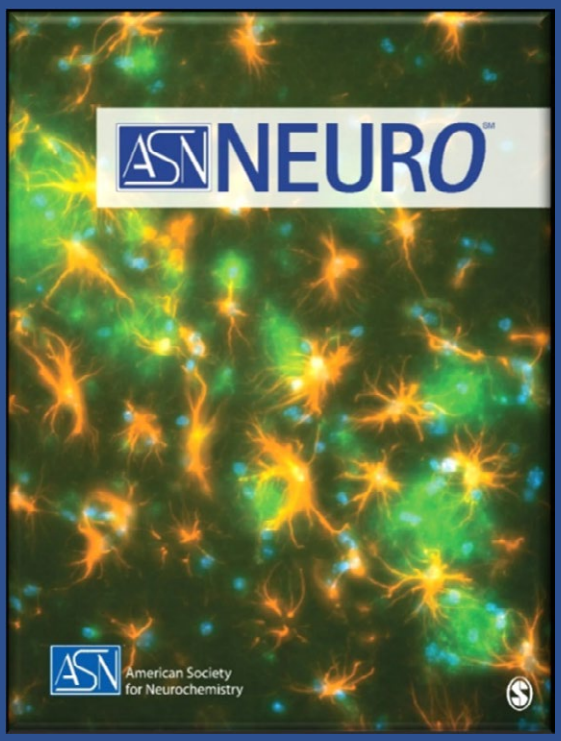

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