RUTGERS UNIVERSITY
RUTGERS HEALTH

7th Annual Neurosurgery Research Symposium

Poster Abstracts

Hosted by:
Dr. Detlev Boison
& Dr. Hai Sun

Poster #1

Protein Phosphatase Inhibition Blocks Stress Granule Assembly and Promotes Axon Regeneration

Manasi Agrawal, Meghal Desai, Shruti Ghumra, Pabitra Sahoo

PI: Dr. Pabitra Sahoo

Abstract:
Axonal regeneration following injury remains a major challenge due to intrinsic molecular barriers that limit protein synthesis, an essential process for neuronal repair. Our previous work demonstrated that Ras GTPase-activating protein-binding protein 1 (G3BP1), a core stress granule (SG) protein, sequesters specific axonal mRNAs away from translation, inhibits axonal protein synthesis, and suppresses axon regeneration. We have further shown that S149 phosphorylation of G3BP1 promotes axonal protein synthesis and enhances axon regeneration. However, the phosphatases regulating this pathway remain elusive. Here, we performed a comprehensive screen of various phosphatase families using small molecule inhibitors that block SG assembly. Our screening identified a phosphatase family known to regulate the cellular stress response as a key candidate that regulates SG assembly. Advanced pharmacological screening revealed that inhibition of this phosphatase family enhances axon growth of cultured adult rat dorsal root ganglion (DRG) neurons *in vitro* and accelerates sciatic nerve regeneration *in vivo*. Furthermore, we extended these findings to human models, where we found that inhibition of this phosphatase family in human induced pluripotent stem cell (iPSC)-derived glutamatergic neurons promotes neurite outgrowth, further supporting its potential as a therapeutic target for enhancing neuronal regeneration. Overall, these findings suggest that targeting phosphatase-mediated SG assembly could serve as a novel strategy for promoting axon regeneration after neuronal injury. We further aim to elucidate the molecular processes linking the phosphatase, G3BP1 phosphorylation, and translational control in axonal repair. This work provides a promising avenue for developing therapeutic approaches that enhance neural repair via protein synthesis pathways.

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Disclosures:
None

Poster #2

Targeting Poly (ADP-Ribose) Polymerase 1 (PARP1) as a Therapeutic Strategy for Chemobrain

Mohammad Abdur Rashid, Alfredo Oliveros, Faheem Ullah, Sang Hoon Kim, Sang Hoon Lee, Hyung Goo Kim, Peter Cole, Mi-Hyeon Jang

PI: Dr. Mi-Hyeon Jang

Abstract:
Chemotherapy-induced cognitive impairment (CICI), also known as chemobrain, is frequently reported as a neurotoxic side effect of chemotherapy, affecting approximately 14 million cancer survivors. Notably, CICI can persist long after therapy has ended, significantly diminishing patients' quality of life. Currently, there are no effective treatments to alleviate this condition, and thus the identification of novel therapeutic strategies is urgently needed. Using cisplatin, a platinum-based chemotherapy, as a model for CICI, our previous studies have shown that cisplatin induces mitochondrial damage, increased gliosis, impaired neurogenesis, and synaptic dysfunction—all of which contribute to cognitive decline. We further show that cisplatin induces neuronal DNA damage, leading to increased activation of Poly (ADP-ribose) polymerase 1 (PARP1) and the synthesis of its product, PAR, both in the adult mouse hippocampus *in vivo* and in human excitatory cortical neurons *in vitro*. Remarkably, inhibiting PARP1 with the anti-cancer drug veliparib (a potent PARP1/2 inhibitor) effectively prevents cisplatin-induced neuronal and cognitive deficits by blocking the cyclooxygenase-2-mediated prostaglandin E2 inflammatory signaling pathway. These results highlight a causative role of PARP1 upregulation in cisplatin-induced brain injury. Collectively, our study is the first to demonstrate the critical contribution of PARP1-dependent NAD+ metabolic dysregulation in chemobrain. Given that several PARP1 inhibitors are FDA-approved for cancer treatment, and veliparib is currently in clinical trials for pediatric and other cancers, our findings could rapidly translate into clinical trials targeting chemobrain, offering a promising strategy to improve the quality of life for cancer survivors.

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Disclosures:
The authors have declared that no conflict of interest exists.

Poster #3

Monoclonal Antibodies Rescue Epsilon Toxin-Induced Damages in Human Oligodendrocytes

Irva Patel, Hiroko Nobuta, Timothy Vartanian

PI: Dr. Hiroko Nobuta

Abstract:
Multiple sclerosis (MS) is a chronic demyelinating disorder of the central nervous system (CNS) characterized by neuroinflammation, demyelination, and axonal loss. MS damages oligodendrocytes, the myelin-producing cells in the CNS. The exact cause of MS remains unknown, with studies highlighting genetic and environmental factors. One potential trigger is *Clostridium (C.) perfringens*'s epsilon toxin (ETX). C. perfringens is a gram-positive bacterium which releases ETX, a pore-forming toxin, known to specifically target oligodendrocytes in livestock and murine samples. It causes blood-brain barrier disruption and dose- and time-dependent demyelination in mouse cerebellar slice cultures. However, the direct effects of ETX on human oligodendrocytes remain unknown. Hence, we treated human oligodendrocyte samples with ETX (0–100nM) for 2 or 6 hours, and immunofluorescently stained with oligodendrocyte marker myelin basic protein. Images were captured and morphological analysis was conducted by tracing cellular processes. We determined that ETX reduces the total path length of cellular processes in oligodendrocytes, shifting the individual path length distribution by increasing short processes and decreasing long ones. ETX also increased the percentage of pyknotic cells in a dose- and time-dependent manner, suggesting cell death. To test the potential therapeutic effects of neutralizing antibodies to ETX, monoclonal antibodies JL4 or JL8 were premixed with ETX and added to the oligodendrocyte culture. This treatment effectively rescued oligodendrocyte processes and pyknotic cell percentage toward control levels. Together, these findings demonstrate ETX-induced injury in human oligodendrocytes and show that antibody neutralization reduces ETX-mediated damage.

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None

Poster #4

An Epileptogenic Network for Determining Chokepoints for Targets in Mesial Temporal Lobe Epilepsy

Rudra Joshi, Fabio Tescarollo, Spencer Chen

PI: Dr. Hai Sun

Abstract:

Introduction: Resection/neurostimulation treatments of seizure onset zone carry a 20-40% failure rate for patients, suggesting that other regions of the brain outside of the seizure onset zone may be contributing to ictogenesis. As such, we developed an analytical approach to reveal regions of the brain, “chokepoints,” that can be targeted to terminate seizure onset.

Methodology: We used optogenetics in PV-Cre mice (n=4) to excite the CA1 of the hippocampus to induce seizures (n=62 seizures). Simultaneously, we captured EEG recordings from 8-10 regions of the brain outside of the CA1. Our novel analytical approach involved developing “causalgrams” by applying Multivariate Granger Causality Analysis to determine the direction of signaling between brain regions during ictogenesis based on each optogenetic stimulation event.

Results: Our causalgrams indicate a net outflow of signal from the stimulated hippocampus (CA1) to all other regions of the brain during the first 10ms following each stimulation pulse. This was followed by a secondary discharge phase (15-40 ms after stimulation) with net signal inflow back to CA1. During the secondary discharge phase, the bilateral thalamus channeled the most signals in 3/4 mice, while in the remaining mouse, the entorhinal cortex was the predominant region.

Conclusion: Based on our causalgram analysis, recurrent feedback signaling from the entorhinal cortex and the thalamus, may be the potential driver of ictogenesis for hippocampal stimulated mice, suggesting these two regions are potential chokepoint targets in the brain network for seizure termination.

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None

Poster #5

The Effects of Rehabilitation Training on Corticospinal Tract Rewiring Following Spinal Cord Injury

Jaclyn T. Eisdorfer, Hannah D. Nacht, Tess Kowalski, Joshua T. Thackray, Alana M. Martinez, Lance Zymoro, Megan V. Phu, Ridhi R. Hirpara, Burhanuddin S. Danish, Riley Wang, Adyan Khondker, Adam B. Eisdorfer, Max Tischfield, Victoria E. Abraira

PI: Dr. Victoria Abraira

Abstract:
Spinal cord injury (SCI) often presents as a contusion, a bruising of the spinal cord that impairs motor functions but preserves some pathways. Contusions, therefore, have potential for recovery as preserved neurons can rewire to take over for lost pathways. Recovery is enhanced by activity-based rehabilitation therapies that stimulate motor pathways like the corticospinal tract (CST). Treadmill rehabilitation training has been shown to improve locomotor function following SCI, but the specific neuronal-level changes remain unclear. We assessed these physiological changes along the CST following treadmill training, and as a side study, we explored the motivational component behind recovery. A custom Emx1Cre;LSL-SynGFP mouse line helped quantify rewiring in mice given moderate contusion SCI, who either received or did not receive treadmill training. Mice were stratified by varying motivational levels using the Progressive Ratio Assay. We evaluated locomotor recovery using the Basso Mouse Scale (BMS), joint/limb kinematics, and Motion Sequencing (MoSeq) analysis. Our data indicated increased synaptic density in the ventral horn and revealed improved BMS scores, especially in highly motivated animals. Of note, behavioral differences in stance were correlated more with motivation levels, while speed was correlated with training levels, suggesting that rehabilitation offers generalized recovery while individual motivation can fine-tune recovered behaviors. These findings suggest that treadmill training enhances CST rewiring following SCI, with a significant motivational dependence. This study may provide insights for further research on activity or motivation-based neuronal rewiring and may help optimize current rehabilitation strategies.

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Poster #6

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

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PI: Dr. Renping Zhou

Abstract:
Amyotrophic Lateral Sclerosis (ALS) is a fatal neurodegenerative disease, with limited treatments. Gene therapy offers an alternative strategy for treating a large portion of ALS patients, however, the disparate genetic alterations in ALS complicate the development of gene therapies. Tyrosine receptor kinase B (TRKB) and Tyro3 receptors are highly expressed in mouse spinal cord motor neurons, suggesting that their ligands, brain-derived neurotrophic factor (BDNF) and growth arrest-specific 6 (GAS6), respectively, are crucial for neuronal survival. In this study, we tested whether genetically induced and muscle tissue-specific expression of such survival-enhancing ligands would ameliorate symptom development in the SOD1G93A ALS mouse model. The therapeutic vectors (AAV-Pmus7-HuBDNF-teLuc or AAV-Pmus7-HuGAS6), or a control vector (AAV-Pmus7-teLuc) were injected intravenously via the retro-orbital route and intramuscularly into the hindlimb skeletal muscle of six-week-old mice. Treatment with the therapeutic vectors delayed disease onset and slowed progression in both male and female mice. Interestingly, a sex-specific response was observed, with female mice benefiting more from the treatments than males. Lumbar motor neuron survival was more sustained in the therapeutic vector-treated group compared to control vector group. No statistically significant extension of lifespan was observed in the treated groups.

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Poster #7

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

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

PI: Dr. Mi-Hyeon Jang

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

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Disclosures:
The authors have declared that no conflict of interest exists.

Poster #8

Uncovering the Mechanism of Electrical Stimulation in Schwann Cells for Nerve Injury Repair

Felicia Giordano

PI: Dr. Hongjun Wang

Abstract:
Peripheral nerve regeneration relies critically on Schwann cells (SCs), which undergo a transient repair-state transition following injury to support axonal regrowth. Electrical stimulation (ES) has emerged as a promising therapeutic strategy to enhance nerve regeneration, yet the direct cellular and molecular mechanisms by which ES modulates SC behavior remain poorly defined. Here, we developed a custom-engineered, sterile, high-throughput electrical stimulation platform compatible with standard 96-well plates to deliver uniform, reproducible direct current (DC) electric fields under controlled in vitro conditions. Using this system, we investigated how ES influences calcium signaling, gene expression, and migratory behavior. Live-cell calcium imaging revealed that DC ES elicited rapid, voltage-dependent intracellular calcium influx in primary rat SCs, with stronger and more sustained responses observed at higher field strengths. RNA sequencing identified ES-induced transcriptional programs involving cytoskeletal regulation, stress response pathways, metabolic remodeling, and injury-associated signaling. Notably, Map1b and other cytoskeleton-related genes were differentially regulated, motivating further investigation. Targeted qPCR revealed dynamic, time-dependent regulation of injury-responsive genes, including Atf3, Sox2, Thbs2, and Map1b, consistent with transient repair-state activation. Functionally, ES significantly enhanced SC migration in vitro, an effect inhibited by microtubule inhibitors but preserved under mitotic blockade, indicating a migration-driven rather than proliferation-driven response. Together, these findings demonstrate that ES directly activates calcium-dependent, cytoskeleton-mediated signaling pathways in SCs, driving transcriptional and functional changes consistent with a pro-regenerative repair phenotype. This work establishes a mechanistically grounded in vitro framework for studying ES-driven nerve repair and highlights its therapeutic potential for enhancing regeneration, including in chronically denervated states.

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Disclosures:
None

Poster #9

Convergent Genetic and Functional Evidence Supports AHNAK as an Autism Gene

Yu Young Jeong, Raul Aramburo, Jung Hee Jin, Bonsu Ku, Yongkyu Park, Hyung Goo Kim, Mi-hyeon Jang and Yong Kim

PI: Dr. Yong Kim

Abstract:
The rising prevalence of autism spectrum disorder (ASD) represents a major public health concern. Despite advances in genomic technologies, 60% of individuals with ASD remain without a molecular diagnosis, underscoring the pressing need to identify novel autism genes and convergent biological pathways. AHNAK is a large, evolutionarily conserved scaffold protein involved in calcium-dependent signaling. Our prior studies demonstrated that AHNAK regulates L-type voltage-gated calcium channel (VGCC) activity through N-terminal interaction with Cav1.2 or Cav1.3 pore-forming subunits and C-terminal interaction with VGCC β auxiliary subunits, positioning AHNAK as a key regulator of neuronal development and plasticity. Importantly, L-type VGCC subunit genes, including CACNA1C, CACNA1D, CACNA1S, and CACNB2, are established neurodevelopmental disorder (NDD) genes implicated in ASD, providing genetic evidence that disruption of the AHNAK signaling complex may contribute to the pathogenesis of ASD. Although AHNAK has not been designated as an autism gene in prior literature, a comprehensive review of large independent NDD cohorts uncovered 52 rare AHNAK variants, including missense, nonsense, splice-site, and frameshift alterations, across reported datasets. In silico analyses, including protein modeling of missense variants, predict deleterious, likely loss-of-function effects that may impair channel scaffolding and synaptic signaling. Consistent with the human genetic findings, Ahnak knockout mice exhibit ASD-relevant behavioral abnormalities, including impaired social interaction, deficits in sensorimotor gating, reduced cognitive flexibility, spatial memory impairment, and depression-like behaviors. Together, convergent evidence from binding partner genetics, rare-variant burden, and functional mouse phenotypes supports AHNAK as a previously unrecognized autism gene and implicates dysregulated calcium-signaling complexes in ASD pathogenesis.

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None

Poster #10

Parvalbumin-positive Interneurons Promote Seizure Intensification and Inhibitory-Excitatory Phase Locking in an Optogenetic Kindling Model of Mesial Temporal Lobe Epilepsy

Rishi Jai Patel, Koray Ercan, Giselle Korn, Rogério Gerbatin, Spencer Chen, Hai Sun

PI: Dr. Hai Sun

Abstract:
Mesial temporal lobe epilepsy (mTLE), characterized by focal seizures originating from hippocampal structures, accounts for ~35% of epilepsies. Although parvalbumin-positive (PV) interneurons classically suppress excitatory pyramidal networks, emerging evidence suggests a more nuanced role in the context of epilepsy. To investigate this, we used PV-Cre mice transduced to express ChR2 under a CaMKIIα promoter to enable optogenetic seizure induction and Cre-dependent hM3Dq to permit selective chemogenetic activation of PV interneurons. A fiber optic implant targeted the left ventral hippocampus and sEEG probes were placed bilaterally. Mice underwent repeated 465 nm light stimulation (kindling) to model mTLE.

PV activation exhibited a pro-ictal effect resolvable both at the single-seizure level and across kindling progression. Following optogenetic stimulation, PV activation reduced latency to seizure onset and produced more intense seizures behaviorally, as measured by a modified Racine scale. Upon seizure onset, PV-activated mice demonstrated elevated theta-band activity, consistent with increased pyramidal cell firing. At the network level, phase–amplitude coupling revealed sustained theta–gamma phase locking throughout seizures in PV-activated animals, indicating persistent hypersynchrony between PV interneurons and pyramidal cells otherwise absent in controls. Additionally, aperiodic component slope regression demonstrated a prolonged shift toward a more excitation-dominant population state after seizure onset in PV-activated mice. Consistent with these acute pro-ictal effects, PV-activated mice also progressed through the kindling paradigm more rapidly, reaching three consecutive Racine 6 seizures in fewer stimulations than controls. Collectively, these results suggest PV interneuron activation may lower seizure threshold, drive increased seizure intensity, and accelerate epileptic network remodeling in mTLE.

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Poster #11

EEG-based Connectivity in Secondary Motor Areas are Related to Increased Functional Recovery Four Months Post-Stroke

Michael Glassen, Jigna Patel, Qinyin Qiu, Gerard Fluet, Alma Merians, Soha Saleh, Sergei Adamovich

PI: Dr. Soha Saleh, Dr. Sergei Adamovich

Abstract:
This study examined how alterations in brain connectivity metrics during the first four months after a stroke relate to recovery of upper limb motor function in individuals post-stroke. Using a data-driven approach, we explored the relationship between changes in resting-state EEG-derived brain connectivity and improvements in motor function, as assessed by the Fugl-Meyer Assessment(UEFMA). We also explored how baseline connectivity relates to changes in UEFMA.

Resting-state EEG data were collected within the first month after stroke and again at four months post-stroke for thirty-seven individuals. Connectivity metrics were analyzed across the Theta, Alpha, Beta1, Beta2 and Gamma frequency bands. Correlation analyses and modeling were employed to identify the brain networks most strongly associated with recovery.

Results show a relationship between changes in brain connectivity among specific regions and improvements in UEFMA scores. Specifically, greater increase in connectivity between the precuneus and middle frontal gyrus correlate with higher recovery. Baseline IPL-MFG connectivity in the affected hemisphere correlates with increased UEFMA, while the affected precuneus and dorsal premotor areas show similar relationships. Comparison of node strengths for each studied ROI at 4 months versus baseline revealed that dorsal premotor node density in the unaffected hemisphere increases across all frequency bands. These results reinforce the value of exploring EEG-based metrics to inform individualized treatment approaches, underscoring the critical role of engaging the affected hemisphere in post-stroke recovery.

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Disclosures:
None

Poster #12

Mechanistic Pathways Linking Pediatric Temporal Lobe Seizures to Cortical Neuronal Loss

Faith U. Chukwudinma

PI: Dr. Todd Mowery

Abstract:

Objectives: Investigating how hippocampal kindling and focal motor seizures contribute to apoptotic signaling and progressive gray matter degeneration within para-hippocampal cortical networks.

Background: Gray matter supports higher-order cognition, including attention, memory, and decision-making, while white matter enables rapid signal transmission via myelinated axons. Focal motor seizures, often arising from epileptogenic lesions in the contralateral frontal lobe, are characterized by neuronal hyperexcitability and hypersynchronous network activity. Hippocampal kindling, repeated subthreshold stimulation, sensitizes neurons, increasing seizure susceptibility and altering temporal-frontal circuitry. Chronic imbalance between programmed cell death (apoptosis) and necrosis contributes to degenerative processes and structural remodeling within affected regions.

Methods: A cross-sectional analysis focused on hippocampal subfields vulnerable to kindling-induced degeneration. Attention was given to dentate gyrus and CA3–CA1 circuitry, examining how necrotic damage, impaired neurogenesis, and glial scarring propagate apoptotic cascades.

Results: Kindling within the dentate gyrus and CA3 enhances synaptic excitability, producing hypersynchronous bursts (200–600 Hz) that propagate through hippocampal networks. Excess glutamate release drives NMDA and AMPA receptor overactivation, leading to calcium influx, reactive oxygen species (ROS) generation, and caspase-dependent apoptosis. Chronic overstimulation promotes maladaptive synaptic pruning and neurofilament light chain release, indicating neuroaxonal injury. Progressive neuronal loss in CA3 and dentate regions triggers astrocytic and microglial proliferation, resulting in hippocampal sclerosis. These structural changes contribute to gray matter reduction, cognitive decline, and increased vulnerability to disorders such as temporal lobe epilepsy and schizophrenia-like neuropathology.

Conclusion: Hippocampal kindling initiates excitotoxic and apoptotic cascades that culminate in sclerosis, gray matter atrophy, and long-term neurodegeneration within temporal lobe networks.

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Disclosures:

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Poster #13

Sex Differences in Brain Tumor Diagnosis and Outcomes: Are Women Being Diagnosed Later?

Gloria Bachmann, Akshita Prakash, Marisa Syed

PI: Dr. Gloria Bachmann

Abstract:
Brain tumors remain a major cause of neurological morbidity and mortality, yet potential sex-based disparities in diagnosis and outcomes remain understudied. Emerging evidence suggests that women may experience longer diagnostic delays compared to men, potentially due to the misattribution of neurological symptoms to less serious conditions such as stress, hormonal changes, or migraines. These delays may contribute to more advanced disease at diagnosis and poorer clinical outcomes. Understanding how sex-based differences influence clinical recognition and diagnosis is essential for improving early detection, reducing disparities and improving patient care.

Method: Peer-reviewed studies will be analyzed to assess diagnostic timelines and misdiagnosis rates among patients with brain tumors. Data sources will include PubMed, Google Scholar, and the National Institutes of Health (NIH). Keyword searches such as “gender disparities in neurological diagnosis” and “brain tumor misdiagnosis” were used to identify relevant studies. Articles conducted in the United States and published between 2014 and 2025 will be prioritized, with a focus on research examining gender differences in diagnostic experiences, and outcomes.

Hypothesis: We hypothesize that women experience longer diagnostic delays for brain tumors compared to men due to differences in symptom interpretation and potential gender bias in clinical evaluation, leading to later-stage diagnosis, and poorer outcomes.

Conclusion: By synthesizing current evidence, our review aims to highlight gaps in the literature and identify potential mechanisms contributing to sex-based disparities in brain tumor diagnosis. Findings may inform future clinical research, improve diagnostic awareness, and guide strategies to reduce gender based diagnostic bias in neurological care for women.

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Disclosures:
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Poster #14

Classical Complement Pathway Activation May Regulate Macrophage-Mediated Cochlear Synaptic Repair Following Noise-Induced Hearing Loss

Shriti S. Thakur, Sreevarshini Murali, Dinesh Y. Gawande, Emma J. Nicolaisen, Kanika Sharma, Vikas Kumar,Tejbeer Kaur

PI: Dr. Tejbeer Kaur

Abstract:
Noise-induced cochlear synaptopathy (NICS) refers to the rapid and primary loss of inner hair cell (IHC) ribbon synapses following exposure to mild to moderate noise. Such synaptic loss contributes to deficits in auditory acuity, particularly in noisy environments. We have shown critical role of macrophages, in facilitating the restoration of IHC ribbon synapses following NICS. To determine the factors that regulate macrophage-dependent synaptic repair, proteomics was performed, which revealed an upregulation of classical complement protein, C1qa implicated in CNS synapse remodeling. Our goal is to determine the expression, activation, source, and localization of complement proteins after noise-induced synaptopathy.

C57BL/6J (WT) mice were exposed to a synaptopathic noise of 93 dB SPL for 2 hours at an octave band of 8-16 kHz. Complement proteins were examined by immunoblotting, ELISA, and fluorescent immunohistochemistry at different days post-noise exposure (DPNE). Cochlear function was assessed in C3 wildtype and knockout mice by auditory brainstem responses (ABR) and peak I amplitudes

C1q, C3 and C5 activity increased in the cochlea by 1 DPNE, peaked at 3 DPNE and started to subside by 7 and 15 DPNE as measured by ELISA. At 1 DPNE, C3 immunolabeling showed increased fluorescent intensity in the middle region of the cochlea. We saw accumulated C3 opsonizes the noise-damaged IHC ribbon synapses. Also, macrophages displayed higher immunoreactivity for the complement receptor CD11b, phagocytic markers CD68 and GAL3 and engulfment of synaptic proteins. The ABR thresholds were comparable between NE C3 WT and KO mice, the peak I amplitude and synapse density were significantly reduced in the C3 KO.

These preliminary findings suggest that the activation of the classical complement pathway may regulate cochlear ribbon synaptic repair following noise-induced hearing loss.

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None

Poster #15

Investigating Regulatory Mechanisms in Helminth-mediated Neuroinflammation

Arman Sawhney, Pravanvel Bala, Jianya Peng, Marissa N. Schroeter, John J. Ponessa, Krupa Chavan, Mark C. Siracusa

PI: Dr. Mark C. Siracusa

Abstract:
Protective immunity to helminth parasites is associated with the development of immune responses that promote worm clearance and simultaneously initiate wound healing responses required to mitigate tissue damage. While these tightly regulated responses have been extensively studied, how they affect the central nervous system (CNS) remains poorly defined. Given that helminths can have tolerance-promoting effects, investigating infection-induced changes occurring in the CNS may provide insight into mechanisms which restrict neuroinflammatory damage. Here we report that *Trichinella spiralis* infection causes acute damage to the blood-brain barrier and is associated with the infiltration of immune cells into the CNS. Moreover, we report increases in regulatory markers such as *F10* and *Chil3* that are distinct from the proinflammatory signatures seen with more established models of neuroinflammation. Importantly, these immunologic changes correlate with a rapid healing response and a return to CNS homeostasis within 3-4 weeks of initial infection. Depleting basophils using transgenic mice is associated with diminished transcriptional signals of repair-associated molecules in the brain. These studies demonstrate that *T. spiralis* infection promotes a unique and understudied form of neuroinflammation that exhibits features of classical wound healing responses which have not yet been described in the CNS. Taken together, this demonstrates that peripheral helminth infection can induce substantial changes in the CNS immune environment that have the potential to inform our understanding of regulatory and restorative mechanisms in the compartment. Future work will investigate how these helminth-specific responses regulate unrelated forms of neurological damage.

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