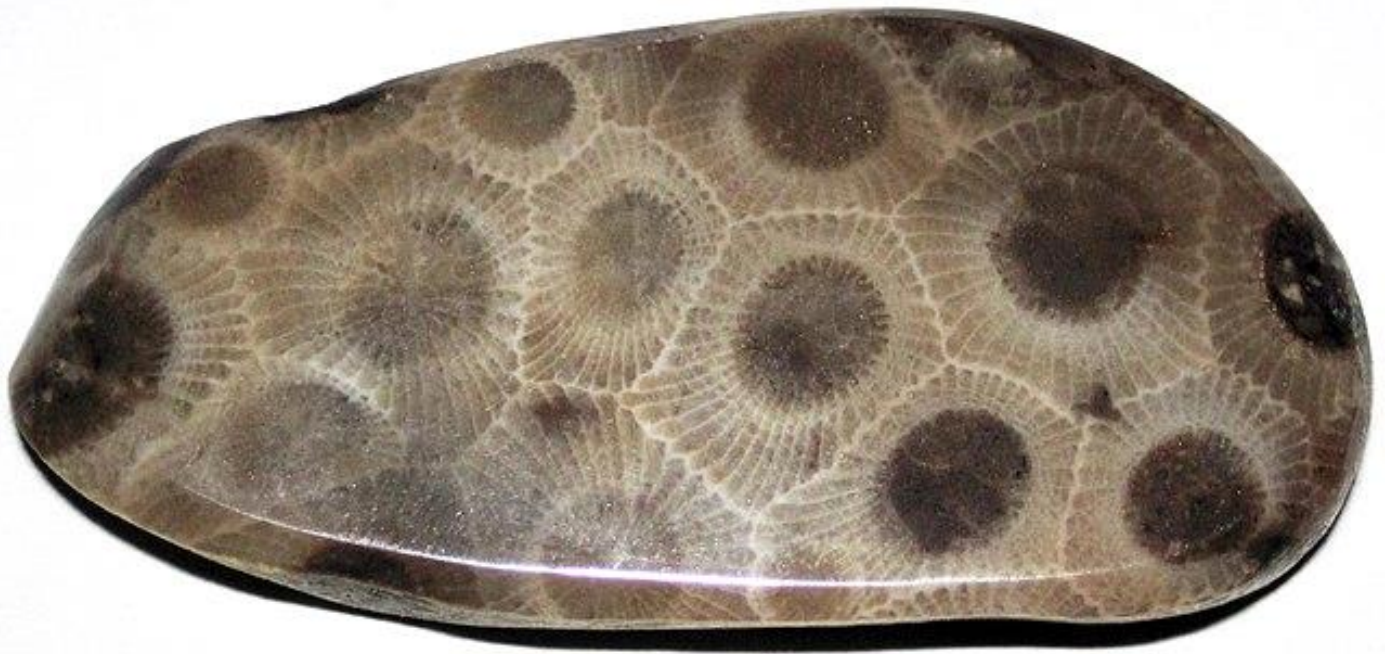


The 13th Great Lakes Glia Meeting



PARK PLACE HOTEL & CONFERENCE CENTER
Traverse City, MI
October 9th – 11th, 2022

Great Lakes Glia 2022

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Table of Contents

Great Lakes Glia 2022 Committees	3
Cover Photo Description	3
Acknowledgement of Support	4 - 7
Program	8 - 10
Abstracts	11 - 24
Registrants	25 - 30
Notes	31 - 32

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Petoskey stones, Michigan's official state stone, from the great lakes region. Wikimedia Commons

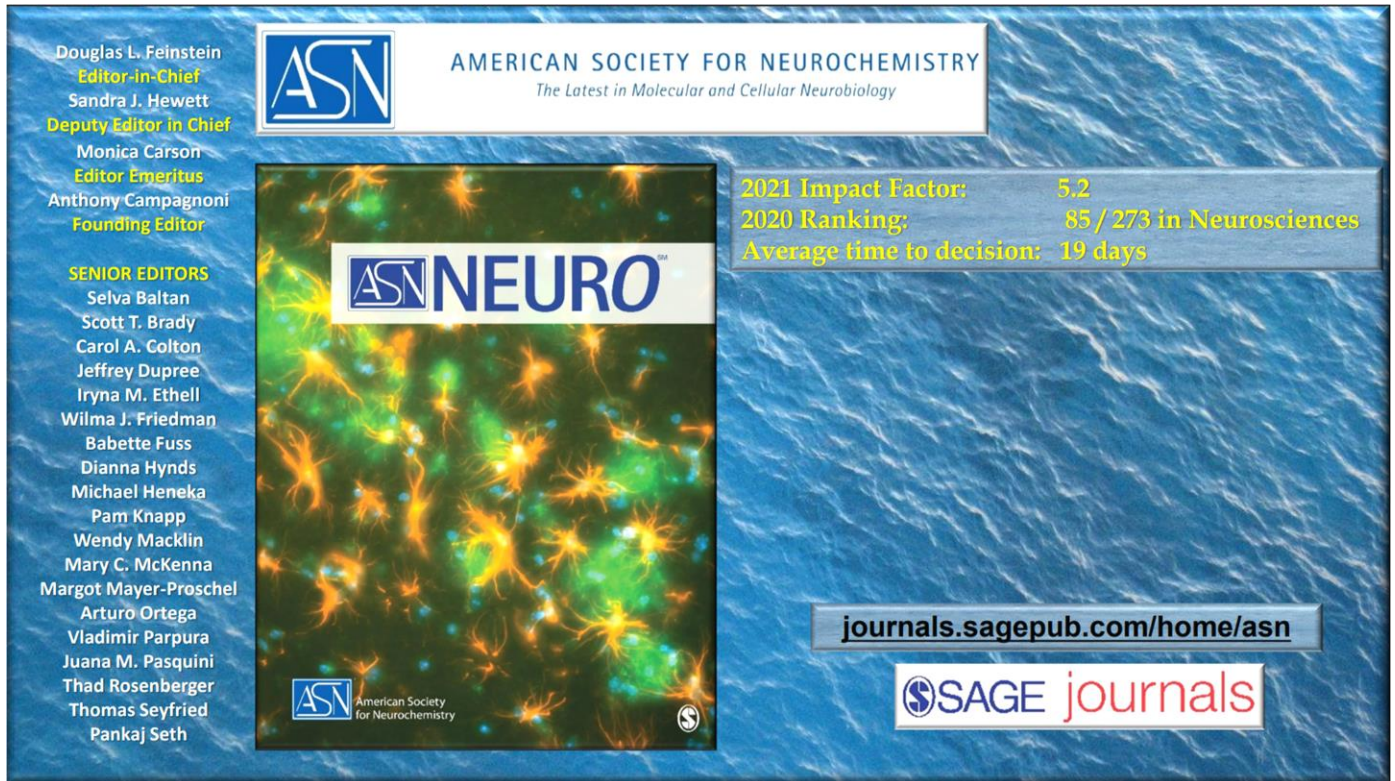
The Petoskey stone is fossilized pre-historic coral fossilized rugose coral, *Hexagonaria percarinata*. Distinguishable by its unique exoskeleton structure, a Petoskey stone consists of tightly packed, six-sided corallites, which are the skeletons of the once-living coral polyps. The center of each polyp was the mouth and contained tentacles that reached out for food. The hexagon shape of each cell and thin lines radiating out from the dark "eye" in the center are distinguishing features unique to this fossil. Source: Pure Michigan

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The image shows a promotional banner for the American Society for Neurochemistry (ASN) journal, ASN Neuro. The background is a blue, wavy texture. On the left, there is a list of editors. In the center, there is a thumbnail of the journal cover featuring a colorful, abstract image of neurons. On the right, there is a box with journal metrics and a URL. At the bottom right, there is the SAGE journals logo.

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Schedule

Sunday, October 9th

4:30pm	Opening Reception	Top of the Park
6:00pm	Dinner	Top of the Park
7:30pm	Session 1 Oligodendrocyte Development and Myelination: Molecular Mechanisms in Health and Disease	Grandview I

Chair: Pablo Paez, University of Buffalo

Leandro Marziali, University of Buffalo

A Novel Stress-Activated Inhibitor of Myelination

Yannick Poitelon, Albany Medical College

Role of YAP and TAZ in Oligodendrocytes

Yungki Park, University of Buffalo

Regulatory Mechanisms Governing Plp1 Expression for Central Nervous System Myelination

Pablo Paez, University of Buffalo

Modulation of Oligodendrocyte Development and Myelination by Voltage-Gated Calcium Channels

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Monday, October 10th

8:00am	Breakfast	Top of the Park
8:45am	Keynote Presentation Keith Murai, McGill University <i><u>Astrocyte Heterogeneity and Nanoarchitecture in the CNS</u></i>	Grandview I
9:45am	Break	
10:00am	Session 2 Astrocyte Heterogeneity in Health and Disease Co-Chairs: Keith Murai, McGill University Doug Feinstein, University of Illinois, Chicago Zila Martinez, The Children's Hospital of Philadelphia <i><u>Generation of Astrocyte Diversity: Lessons from Transcriptional Regulation of the Glutamate Transporter 1(GLT1)</u></i> José Otero, The Ohio State University College of Medicine <i><u>Brainstem Astrocytes and Their Regulation of Autonomic Homeostasis</u></i> Ryan Gilbert, Rensselaer Polytechnic Institute <i><u>Biomaterial Approaches to Augment Astrocyte Reactivity</u></i> Isobel Scarisbrick, Mayo Clinic <i><u>Modulating Astrocyte Metabolism to Promote CNS Regeneration</u></i>	Grandview I
Noon	Free time	
3:30pm	Poster Session	Grandview II
4:30pm	Session 3 Microglia in Health and Disease Chair: Tyler Ulland, University of Wisconsin-Madison Kathryn Lenz, The Ohio State University <i><u>Prenatal Allergic Inflammation, Mast Cell-Microglia Interactions, and Sex-Specific Programming of Motivated Behavior</u></i> Tyler Ulland, University of Wisconsin-Madison <i><u>Inhibition of the Nlrp3 Inflammasome by 6-Hydroxybutyrate Decreases Alzheimer's Disease Pathology</u></i> Sebastian Werneburg, University of Michigan <i><u>Microglia and the Elimination and Recovery of Synapses in Demyelinating Disease</u></i> Subhash Pandey, University of Illinois <i><u>Neuroinflammation Signatures in the Pathophysiology of Alcohol Use Disorder</u></i>	Grandview I
7:00pm	Dinner	Top of the Park
8:30pm	Poster Session & Refreshments	Grandview II

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Tuesday, October 11th

7:30am Breakfast Top of the Park
8:15am **Keynote Presentation** Grandview I

R. Douglas Fields, NICHD

Regulation of Myelin and Conduction Velocity by Action Potentials

9:15am Break

9:30am Session 4 Contribution of Glia-Associated Mechanisms to Pathological Endophenotypes of Complex Behaviors Grandview I

Chair: Sinead O'Donovan, The University of Toledo

Sarah Elzinga, University of Michigan

Metabolism and Obesity Effects on Microglia

Marissa Smail, The University of Toledo

Animal Model Characterizing the Consequences of Microglial Loss

Sinead O'Donovan, The University of Toledo

Effects of Psychotropic Medications on Astrocytes using a Bioinformatics Approach

Hayley McLoughlin, University of Michigan

Non-neuronal Contributions to Neurodegeneration: A Role for Oligodendrocytes in Spinocerebellar Ataxia

11:30am Break Out Grandview I

12:00pm Session 5 Glial Responses to Disease and Therapy in Demyelinating Conditions Grandview I

Chair: Ernesto Bongarzone, University Illinois, Chicago

Sarah Lutz, University Illinois, Chicago

Glial Response to Respiratory SARS-CoV-2 Infection is Modified by Age

Stephen Crocker, University of Connecticut

Impact of Cellular Aging on Glial Responses and CNS Remyelination

Anne Boullerne, University Illinois, Chicago

Rediscovery of Pio del Rio Hortega Myelinic Channel System using Fluorescent Markers

Ernesto Bongarzone, University Illinois, Chicago

Adult-Onset Focal Demyelination after Neonatal AAV-Gene Therapy in Leukodystrophies

2:00pm End of Meeting

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1. Role of Myrf enhancers in CNS myelination

Author: Hongjoo An¹, Dongkyeong Kim¹, Chuandong Fan¹, and Yungki Park¹,

¹Institute for Myelin and Glia Exploration, Department of Biochemistry, Jacobs School of Medicine and Biomedical Sciences, State University of New York at Buffalo, Buffalo, NY 14203, USA

Abstract: Myelination of the central nervous system (CNS) by oligodendrocytes (OLs) is indispensable for the function and the development of the CNS. Oligodendrocyte progenitor cells (OPCs) differentiate into OLs that generate myelin in the CNS, highlighting OL differentiation is a key event in CNS myelination. Myrf (myelin regulatory factor, previously known as Mrf or Gm98) is a master regulator of OL differentiation. A distinctive property of Myrf, unparalleled to other OL transcription factors, is that it is expressed in an all-or-nothing pattern in OL lineage cells; its expression is stalled in OPCs, but its expression increases dramatically as OPCs differentiate into OLs. This binary expression pattern suggests that Myrf expression is a critical moment prompting OL differentiation. Therefore, artificial exogenous expression of Myrf in OPCs force their differentiation even in the proliferation condition. Additionally, gene expression analysis of multiple sclerosis lesions revealed that OLs stuck in their differentiation are those that couldn't upregulate Myrf expression. In light of the significance of accurate Myrf expression for OL differentiation, we have explored how Myrf expression is triggered in OL lineage cells. Our interdisciplinary research has found two OL enhancers for Myrf (referred as Myrf-E1 and Myrf-E2).

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2. The effect of G protein-coupled receptor Gpr62 on adult neural stem cells

Elizabeth D. Clawson^{1,2,3,4,5}, Daniel Z. Radecki, PhD^{1,2,3,4}, Jayshree Samanta., MBBS, PhD^{1,2,3,4}

¹University of Wisconsin-Madison, ²School of Veterinary Medicine, ³Department of Comparative Biosciences, ⁴Stem Cell and Regenerative Medicine Center, ⁵Molecular and Cellular Pharmacology Program

G protein-coupled receptors (GPCRs) are the largest group of transmembrane receptors and are involved in many cell signal transduction pathways, including oligodendrocyte development. There are still dozens of GPCRs whose biological roles are unknown. Gaining an understanding of how they may function in oligodendrocyte differentiation and remyelination could open avenues for new treatments for patients of neurodegenerative diseases. In an RNAseq screen for genes regulating remyelination by neural stem cells in the mouse brain, we found differential expression of one GPCR in particular, Gpr62, when the transcription factor Gli1 is knocked out. Additionally, we found Gpr62 to be enriched in the white matter of the mouse brain. In this study, we have characterized the effect of Gpr62 knockdown in primary adult neural stem cells.

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3. Clemastine rescues social deficits and promotes myelination in oligodendrocyte-specific *Anks1b* knockout mice

Dylann Cordova-Martinez¹, Chang Hoon Cho², Bryen A. Jordan¹

¹Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, NY, USA.

²Departments of OMNI Biomarker Discovery and Neuroscience, Genentech, Inc, 1 DNA Way, South San Francisco, CA, 94080, USA

Once considered a quiescent structure, evidence now suggests that myelin sheath formation remains dynamic throughout the lifespan and that this process can play an essential role in regulating behavior. Pioneering research in oligodendrocytes has recently linked dysmyelination to neurodevelopmental disorders. Therefore, understanding the mechanisms that underlie oligodendrocyte function may significantly impact our understanding of disease etiology. Recent work from our lab has reported that monogenic deletions in the *ANKS1B* gene lead to a neurodevelopmental syndrome characterized by autism spectrum disorder, attention deficit hyperactivity disorder, speech impairment, and motor deficits. The *ANKS1B* gene encodes for AIDA-1, a protein highly involved in synaptic and NMDA receptor function. However, loss of AIDA-1 expression from forebrain excitatory neurons or inhibitory Purkinje cells, two areas that highly express the *ANKS1B* gene, does not have an impact on social behavior. To assess social deficits, we performed the often used three-chamber social preference test. Interestingly, we find that loss of *Anks1b* expression from oligodendrocyte-lineage cells in mice (*Anks1b*-OPC-KO) results in social deficits and myelination abnormalities, similar to what we find in a CNS-wide *Anks1b* knockout and other autism mouse models. To test the role of myelination in the behavioral phenotypes found in the *Anks1b*-OPC-KO, we stimulated myelin formation using clemastine, an FDA-approved drug previously shown to promote oligodendrocyte differentiation and myelination. To measure the number of myelinated axons and the morphology of the myelin sheath, we performed transmission electron microscopy. Clemastine treatment of adult *Anks1b*-OPC-KO mice rescued the social preference deficits and improved myelin morphology. Together these results support the role of myelination in the pathophysiology of the *ANKS1B* syndrome and open new avenues for potential therapeutic interventions.

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4. The Role of Sulfatide in Oligodendrocytes

Dustin E^{1,2,3}, Flounlacker K^{1,3}, Palavicini J⁴, Han X⁴, McQuiston R¹, Dupree JL^{1,2}

¹Department of Anatomy and Neurobiology, VCU Medical Center ²Hunter Holmes McGuire Veterans Affairs Medical Center ³Virginia Commonwealth University, Neuroscience Curriculum, Richmond ⁴UT Health San Antonio, Medicine, San Antonio, USA

The myelin sheath in oligodendrocytes acts to maintain rapid conduction velocity, provide trophic support to the neuron, and protect the axon from degeneration. The myelin sheath largely consists of lipids and 3-O-Sulfogalactosylceramide (sulfatide) is a sphingolipid that constitutes up to 4% of total myelin lipids in the central nervous system. Sulfatide has been reported to be dramatically reduced in regions of normal appearing white matter (NAWM) of MS patients. Reduction of this lipid in regions of NAWM suggests that sulfatide may contribute as a driving force of disease pathology and not merely a consequence of disease progression. Previously, our lab and others characterized a mouse lacking sulfatide's synthesizing enzyme, cerebroside sulfotransferase (CST) through constitutive gene disruption. Using this mouse, our lab has shown that sulfatide is required for proper establishment and maintenance of myelin and the axoglial junctions that attach the myelin sheath to the axon and that provide stability to the nodal domains. In addition, we reported that sulfatide is involved in oligodendrocyte differentiation, proliferation and that sulfatide may play a role in protein compartmentalization within the oligodendrocyte and myelin sheath. Interestingly, some of the ultrastructural pathologies that we reported in the CST KO mice are consistent with structural abnormalities observed in Multiple Sclerosis (MS). However, since MS is typically diagnosed in young adults, the constitutive CST KO mouse has limited clinical relevance since these mice lack sulfatide embryonically. To generate a more clinically relevant model, our lab has generated a "floxed" CST mouse, which provides both temporal and cell specific ablation of the CST gene. Using this mouse, with the PLP-cre^{ERT} driver, we demonstrate that there is no change in g-ratios or myelin abnormalities. However ion channels and neuronal proteins become mis-localized. Proper spatial distribution of ion channels is required to propagate a proper signal down the axon, and our electrophysiology data shows the myelinated axons are behaving as unmyelinated axons. Therefore, our studies show adult onset sulfatide depletion is sufficient to drive axonal pathology while maintaining myelin integrity. These findings have significant implications in understanding the clinical presentation of MS indicating that loss of CNS function may precede demyelination. Although our previous and current findings strongly implicate the loss of sulfatide as an early disease driving event, how sulfatide loss contributes to CNS dysfunction remains unknown. To further explore sulfatide-dependent mechanisms that are essential for maintaining CNS structure and function, we are currently culturing mouse oligodendrocytes that lack sulfatide. Using this in vitro model system, we are currently analyzing the role that sulfatide plays in regulating OL development, morphology and myelin protein compartmentalization. Our hypothesis is that sulfatide is essential for proper myelin protein trafficking, and in disease, impaired transport results in compromised myelin repair, disruption in myelin-axon communication and ultimately loss of proper CNS function. Using both in vivo and in vitro, we are understanding the role sulfatide plays during myelin maintenance as well as during myelin repair.

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5. ANKS1B encoded AIDA-1 regulates oligodendrocyte function and myelination through interaction with Rac1 GTPase

Ilana Vasilisa Deyneko¹, Chang Hoon Cho¹, Juan Vazquez¹, Dylann Cordova-Martinez¹, Abigail U. Carbonell¹, Jaafar O. Tindi¹, Min-Hui Cui², Roman Fleysher², Craig A. Branch², Bryen A. Jordan¹

¹Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, NY, USA. ²Department of Radiology, Albert Einstein College of Medicine, Bronx, NY, USA.

Haploinsufficiency in the gene ANKS1B underlies a rare genetic disorder that presents as a neurodevelopmental syndrome. This gene encodes AIDA-1, a brain-specific protein that is highly enriched at neuronal synapses where it regulates NMDA receptor function and synaptic plasticity. However, these functions alone are insufficient to explain the full scope of the syndrome. Individuals with mutations in the ANKS1B gene present with a cluster of symptoms including Autism Spectrum Disorder (ASD), Attention Deficit/Hyperactivity Disorder (ADHD), and speech and motor deficits. Furthermore, MRI in many of these patients reveal abnormalities in white matter, including dysgenesis, partial agenesis, T2 hyperintensities, or thin body of the corpus callosum. Quantitative morphometric analyses showed microstructural differences in patient white matter overall compared to the unaffected controls. Together, these pathological phenotypes suggest deletions in this gene may disrupt myelination. To explore the molecular mechanisms that underlie disease etiology, we used our previously published ANKS1B mouse model. These Nestin-Cre driven transgenic mice demonstrate both behavioral analogues and abnormalities in white matter that are reminiscent of clinical findings in patients. Immunohistological analysis of the mouse model supported evidence for deficits in myelination and revealed that oligodendrocytes were also affected, suggesting an interesting new role for AIDA-1 in oligodendrocyte function. Results showed fewer oligodendrocytes, delayed maturation, and impaired migration of oligodendrocyte lineage cells in our mouse model. Previous interactome analyses revealed specific interactions of AIDA-1 with regulators of Rho family GTPases. The small GTPase Rac1 is essential for oligodendrocyte maturation, myelination and overall function. Therefore, our interactome analyses suggested that GTPase disruption may underlie some phenotypes seen in patients and our mouse model. FRET and GLISA experiments reveal that indeed AIDA-1 strongly regulates Rac1 function in cortical tissue and in oligodendrocyte cell culture, suggesting that disrupted interaction between AIDA-1 and Rho-family GTPases in oligodendrocytes may lead to impaired myelination and thereby act as a potential mechanism underlying ANKS1B Syndrome.

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6. Defining the Molecular Mechanism by Which Transglutaminase 2 Modulates the Astrocytic Response to Injury

Jacen Emerson, Joel Rodwell-Bullock, Peter Girardi, and Gail V.W. Johnson

Department of Anesthesiology and Perioperative Medicine, University of Rochester, Rochester, NY 14620, USA

Astrocytes play a crucial role in promoting neuronal recovery after CNS injury. Previously, our lab has shown that Transglutaminase 2 (TG2) is an important participant in the astrocytic response to injury, however the molecular mechanisms by which it acts are unknown. These studies begin to deduce the mechanism by which TG2 mediates gene expression in astrocytes after injury. Prior work suggested that Zinc Finger and BTB Domain Containing 7a (Zbtb7a), a transcription factor-like protein with a DNA binding domain, was a TG2 interactor. We hypothesize that this interaction between TG2 and Zbtb7a is a key component in the in mediating the response of astrocytes after CNS injury. Co-immunoprecipitation studies confirmed the interaction of TG2 and Zbtb7a, and immunocytochemical analyses showed the interaction localized to the nucleus of astrocytes. Further work investigated whether TG2 may mediate the function of a Zbtb7a- HDAC1 repressor complex by determining how acetylated histone levels were affected by Zbtb7a in the presence or absence of TG2. Immunoblot analysis revealed that basal histone acetylation is higher in TG2 knockout (TG2^{-/-}) astrocytes compared to wild type (WT) astrocytes, and that Zbtb7a overexpression further increased histone acetylation levels. Interestingly, these differences in histone acetylation correlated with the effects of astrocytes on neurite outgrowth. Just as Zbtb7a overexpression increased histone acetylation levels, it increased the ability of both WT and TG2^{-/-} astrocytes to support neurite outgrowth. Further, Zbtb7a overexpression increased the ability of WT astrocytes to support neurite outgrowth significantly more than it did in TG2^{-/-} astrocytes. Considering these findings, and prior RNAseq analyses of injured spinal cords from wild type and astrocytic specific TG2 knockout mice, we hypothesized the TG2-Zbtb7a interaction could be controlling the expression of genes that regulate lipid metabolism in the astrocytes. Promoter activity assays, immunoblotting, and qPCR analyses of WT and TG2^{-/-} astrocytes with modified levels of Zbtb7a indicate alterations in a key enzyme involved in lipid metabolism: fatty acid synthase (FASN), supporting this hypothesis. These studies suggest TG2 may negatively affect the astrocytic response to injury by repressing the expression of key genes involved in fatty acid metabolism in general, and more specifically FASN. Further studies are underway to delineate the interplay of Zbtb7a and TG2 in the regulation of gene expression in astrocytes.

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7. Early life adversity feminizes the molecular architecture of enteric glia

Jacques Gonzales, Christine Dharshika, Wilmarie Morales Soto, Brian D. Gulbransen
Department of Physiology and Neuroscience Program, Michigan State University, East Lansing MI, USA

Irritable bowel syndrome (IBS) affects roughly 12% of humans and is characterized by a remodeling of the nervous system leading to abdominal pain and altered intestinal function. Early life stress is an important risk factor for the development of IBS. How it contributes to disorders of the gut-brain axis through effects on the nervous system is still unclear. Here, we tested the hypothesis that early life stress causes genomic changes in enteric glia, which could influence enteric nervous system function. We used the neonatal-maternal separation (NMS) model as a psychological stressor, and studied glial-specific transcriptional signatures using *Sox10^{CreERT2};RiboTag* mice. Distal colons were collected at 16-20 weeks of age and RNA-seq of myenteric glial mRNA or *in situ* RNA localization were performed. Then, we tested the physiological consequences on visceral hypersensitivity, *in vivo* motility and glial calcium communication on wild-type or *Wnt1^{Cre};GCaMP5g-tdT/Gfap^{hM3Dq}* mice, which expressed chemogenetic hM3Dq DREADD receptors in GFAP⁺ cells and the calcium indicator GCaMP5g in neurons and glia. Male animals that underwent NMS exhibited large shifts in glial genomic profiles with many genes upregulated such as immune-related signaling genes (*Oasl2* ($p < 0.001$), *Ifit1* ($p < 0.05$)), while subtler effects were observed in females such as downregulation of GPCR signaling genes (*RGS5* ($p < 0.001$)). Interestingly, male glia become more like those from females following NMS. These results were confirmed by spatial RNA localization for selected genes such as *Oasl2* ($p < 0.05$) and *Ifit1* ($p < 0.01$). The molecular modification observed after NMS are associated with a change in RNA spatial distribution. This remodeling in molecular architecture is associated with an increased frequency of calcium discharge after specific glial stimulation. Finally, we observed an increased visceral sensitivity in male after NMS ($p < 0.01$), and a slower intestinal transit after glial specific activation in both sex ($p < 0.05$), which may be linked to the hyperexcitability of glia. Together, these data show that early life adversity shifts the molecular architecture of enteric glia in a sex-specific manner, with a most prominent effect in males where maternal separation promotes a 'feminization' of genomic signatures. This remodeling following NMS is associated with physiological consequences on intestinal motility and visceral hypersensitivity, however the mechanisms involved in these changes remain to be identified.

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8. Enteric glia modulate visceral hypersensitivity through the connexin-43 dependent sensitization of Trpv1 nociceptors during inflammation

Morales-Soto W.¹, McClain J.², Gulbransen B.^{1,2}

¹Neuroscience Program, ²Department of Physiology, Michigan State University East Lansing MI

Abdominal pain is a predominant symptom associated with inflammatory bowel disease. Nociceptors innervate the myenteric plexus where they are known to influence enteric glia and control inflammation. The sensitization of nociceptors occurs during the acute phase of colitis, however how enteric glia contribute to nociceptor sensitization is not known. Here, we tested the hypothesis that alterations to intercellular signaling between glia and nociceptors contribute to the sensitization of nociceptors during gut inflammation. We used the dinitrobenzene sulfonic acid (DNBS) model of acute colitis and tested the effects on enteric glia using immunohistochemistry, multiplex immunoassays, and ethidium bromide dye uptake to measure glial Cx43 channel activity. Specific interactions between glia and nociceptors were assessed using *GFAP-HM3Dq;TRPV1Cre;GCaMP5g-TdT* to specifically modulate glial cells using chemogenetics and directly record calcium (Ca^{2+}) activity in nociceptors. Finally, we used the glial specific Cx43 knock out model *Cx43::Sox10Cre^{ERT2}* in combination with visceromotor reflex recordings to disrupt glial intracellular signaling and study its impact on visceral hypersensitivity. Peak DNBS colitis drove an increase in proinflammatory cytokines at the whole tissue level including IL-6, TNF- α , and IL-17, and IL-1 β showed a 5-fold increase ($n = 4-5$, $p < 0.05$). Enteric glia contribute to the production of IL-1 β during colitis which can be observed as a 6-fold increase by RNAseq ($n = 3$, p -adjusted < 0.1) and a 40% increase in glial IL-1 β immunolabeling data at peak colitis ($n = 3$, $p < 0.05$). IL-1 β increased glial dye uptake under basal conditions and potentiated ADP-stimulated dye uptake by 24% ($n = 10-15$ ganglia, from at least 3 mice; $p < 0.0001$) in a Cx43 dependent manner. The selective activation of glial cells with clozapine-N-oxide (CNO) in *GFAP-HM3Dq;TRPV1Cre;GCaMP5g-TdT* induced changes in nerve fiber responses to capsaicin in the presence of IL-1 β and the inhibition of Cx43 with the mimetic peptide 43Gap26 ameliorated this response ($n = 5-7$, $p < 0.05$). RNAseq data showed increases in the PGE2 synthesizing enzyme, cyclooxygenase-2 (COX-2) in enteric glia during DNBS ($n = 3$, $p < 0.05$). Hence, blocking prostaglandin EP4 receptors on nociceptors resulted in a reduction of Ca^{2+} responses on nociceptors in the presence of CNO and IL-1 β . Finally, visceromotor responses to colorectal distensions during inflammation were reduced in glial Cx43 knock out mice ($n = 8-10$, $p < 0.05$). Interestingly, this effect was only observed in females and not males. Together our data identify IL-1 β as a candidate glial mediator that has the capacity to affect nociceptors by influencing glial Cx43-dependent PGE2 release.

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9. NOVEL EXPRESSION OF NEUROFASCIN IN MICROGLIA: A CANDIDATE FOR MICROGLIA-AIS INTERACTION AND MORPHOLOGY REGULATOR

Suárez-Pozos E¹, Benusa S¹, Giordano E², Izabel S⁴, Clark K⁵, Manzoor B⁶, Dupree JL^{1,3}

¹Department of Anatomy and Neurobiology, Virginia Commonwealth University, ²Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institute of Health, ³Research Service, Hunter Holmes McGuire Veterans Affairs Medical Center, ⁴Neuroscience Program, Stanford University, ⁵Virginia Tech Carilion Research Institute, Department of Cellular and Integrative Physiology San Antonio Health.

Microglia, the innate immune cells of the CNS, influence the development and function of neuronal networks by actively interacting with neurons. Consistent with this role, our lab recently reported that microglia extend and establish intimate interaction with the specialized axonal domain known as the axon initial segment (AIS) during inflammatory events. Moreover, we showed that the AIS was disrupted both structurally and functionally during these inflammatory events and that microglial contact preceded this disruption. Based on these observations, we proposed that microglia are potential regulators of AIS function and may elicit a negative impact on the AIS during disease onset and progression. To test this hypothesis, we determined to inhibit this interaction and analyze AIS structure and function. To this end, we undertook a candidate approach to first identify a potential mediator of microglial-AIS interaction. Based on the literature, we identified neurofascin (Nfasc) as a strong candidate since the Nfasc gene has been postulated to have as many as 50 splice variants, generated cell-type specific isoforms that regulate process extension, axonal domain maintenance, and axo-glial interaction. Here, we have identified and characterized a novel isoform of Nfasc that is unique from previously described isoforms and is expressed by microglia. Sequencing experiments revealed that microglial Nfasc excludes the small alternatively spliced domains in the extracellular region (mini-exons 6, 11, and 20), the mucin, and 5th FNIII domains while including the 3rd FNIII domain. IHC analysis revealed that expression of this novel Nfasc isoform localized to microglial processes and was enriched at the microglial-AIS interface. To further characterize the role that the novel isoform of Nfasc plays in microglia we used constitutive Nfasc knockout mice under neuroinflammatory conditions to determine if Nfasc regulates microglia morphology; fractal analysis revealed that Nfasc may play a role in the regulation of microglia's morphology when transitioning from surveying to activated state. These findings are exciting since they identify a novel protein that regulates microglial response to inflammatory conditions and that may serve as a therapeutic target for immunomodulation.

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10. Comprehensive and comparative protein binding screen reveals new interactions of the MPZ(P0) cytosolic tail

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Myelin Protein Zero is the major myelin protein expressed by Schwann cells. Mutations in the cytosolic tail of MPZ can cause defects in myelin compaction or lead to axonal degeneration. However, the protein interaction(s) MPZ uses to mediate its functions are poorly characterized.

To identify interactors of the MPZ tail we used DEEPN, (Dynamic Enrichment for Evaluation of Protein Networks), a yeast 2-hybrid workflow that we developed that uses next-generation sequencing on a population of yeast undergoing selective pressure over time as 'bait' and 'prey' protein fusions interact. The custom 'prey' library we used was composed of fragments of the human ORFeome. Among the interactors are regulators of the actin cytoskeleton, which plays a critical role in the early differentiation of Schwann cells and their ability to wrap myelin. Specifically, the dynamics of actin assembly, and activation of myosin light chain kinase have been found pivotal for myelination. DEEPN recovered a myosin light chain kinase (MLCK3), FHL3 and FAM13B as interactors with MPZ tail. MLCK3, is homologous to MLCK that is required for myelin compaction and undergoes increased phosphorylation and relocation to the cell surface during Schwann cell differentiation. FHL3 binds actin, regulates assembly of actin stress fibers, and also binds SMADs, which can regulate a number of cellular differentiation events including Schwann cell development. FAM13B is a Rho GAP (GTPase accelerating protein), implicating it in the control of the cytoskeleton. The finding that MPZ may interact with a regulator of Rho is particularly relevant since Rho kinase (ROCK) can regulate myosin light chain phosphorylation during myelination and defects in the Rho guanine-nucleotide exchange protein FGD4 result in an autosomal recessive form of CMT. Some interactors were sensitive to disease-causing mutations in the MPZ tail, suggesting that loss of these interactions contribute to CMT pathogenesis.

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11. MPZ(P0) forms a specific complex with PMP22 that alters PMP22 localization and may play a role in peripheral neuropathies

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Myelin Protein Zero (MPZ) is one of the highly abundant components in the sheath of peripheral nerves. This adhesion protein is essential for proper structure and function of myelin. Multiple mutations in MPZ found to cause inherited peripheral neuropathies, including CMT (Charcot-Marie-Tooth) disease. Whereas one proposed mechanism for these disorders is that mutant MPZ proteins may induce ER (endoplasmic reticulum) stress, other mutations may affect additional functions of MPZ and delineating the different functions of MPZ is our long-term goal with these studies. Mutations in Peripheral Membrane Protein 22 (PMP22), including its gene duplication, account for most CMT cases. PMP22 is a tetraspan integral membrane protein expressed at high level in Schwann cells. Presently, it is unclear what function PMP22 provides or why an extra copy of wildtype PMP22 can cause dominant CMT1A. Interestingly, when highly expressed in non-Schwann cells, PMP22 accumulates in the ER and might potentially cause disease by eliciting ER stress as well. We found that PMP22 and MPZ form a strong and specific interaction. Using coimmunoprecipitation experiments, PMP22 forms a robust interaction with MPZ in both HEK293 and RT4 rat Schwannoma cells. PMP22 and MPZ each belong to larger protein families, which include the PMP22 homologs EMP1,2,3 and MPZ-like proteins and NaV beta subunits, respectively. Our studies show that among different family members, only PMP22 forms a complex with MPZ and only MPZ forms a complex with PMP22. Using a chimeric approach with different PMP22 or MPZ family members, we found that the determinant for complex formation is the transmembrane domain of MPZ. Finally, we found that co-expression of MPZ with PMP22-GFP shifted its localization from predominantly ER compartments to the cell surface, potentially by accelerating the degradation of the ER-pool of PMP22. These studies provide a physical and mechanistic link to the two major proteins involved in the pathogenesis of CMT and imply that complex formation may play an important disease relevant role.

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12. Solution structure studies of the Ig domain of the MPZ(P0) myelin adhesion protein

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Mutations in Myelin Protein Zero (MPZ) account for 5% of CMT cases overall and can cause demyelinating or axonal phenotypes, reflecting the diverse roles of MPZ in Schwann cells. MPZ holds the apposing membranes of the myelin sheath together, with the adhesion role fulfilled by the extracellular Immunoglobulin-like domain (Ig^{MPZ}), which can oligomerize. Most of what we know for how the Ig^{MPZ} might form oligomeric assemblies has been extrapolated from a protein crystal structure in which individual rat Ig^{MPZ} subunits are packed together under artificial conditions. The current molecular model for how Ig^{MPZ} oligomerizes involves 3 potential weakly-interacting interfaces. These include an interface that organizes the Ig^{MPZ} into tetramers, a 'dimer' interface that could link tetramers together, and a third hydrophobic interface that could mediate binding to lipid bilayers or the same hydrophobic surface on another Ig^{MPZ} domain. Currently, there are no data confirming whether the proposed Ig^{MPZ} interfaces actually mediate oligomerization in solution, whether they are required for the adhesion activity of MPZ, whether they are important for myelination, and whether their loss results in disease. Analysis of the Ig^{MPZ} molecular structure reveals that axonal late-onset disease phenotypes (CMT2) mostly map to surface residues of Ig^{MPZ} whereas early-onset severe demyelinating mutations (CMT1) map to the Ig^{MPZ} interior core. The correlation of surface mutations to CMT2 suggests the dysfunction of Ig^{MPZ} interaction interfaces as a possible cause of this disease phenotype. To explore whether the crystal-packing cis-tetramer and *trans*-dimer interfaces identified in early studies mediate Ig^{MPZ} oligomerization in solution, we performed NMR and SAXS analysis of wildtype Ig^{MPZ} as well as mutant forms with amino-acid substitutions designed to interrupt its presumptive oligomerization interfaces. Here, we confirm the interface that mediates Ig^{MPZ} tetramerization, but find that dimerization is mediated by a distinct interface that has yet to be identified. Finally, we integrate a computational evaluation of disease-causing mutations on the Ig^{MPZ} surface with alternative hypothetical oligomerization interfaces and discuss the possibilities for how MPZ mediates adhesion of myelin layers and how its inability to do so might mediate different forms of CMT.

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13. Lanthionine ketimine ethyl ester accelerates remyelination in a mouse model of MS

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Although over 20 disease modifying therapies are currently approved to treat Multiple Sclerosis (MS), these target the adaptive immune system but do not increase remyelination of demyelinated axons. Previous studies have shown that LKE (lanthionine ketimine ethyl ester) reduces clinical signs in the EAE (experimental autoimmune encephalomyelitis) mouse model of MS and increased maturation of oligodendrocyte progenitor cells (OPCs) in vitro. In the current study we used the cuprizone (CPZ) model of demyelination to test if LKE could increase remyelination. Examination of the corpus callosum (CC) showed that after 5 weeks treatment with CPZ, the % of myelinated axons was reduced from 95% to 5%, and increased to 50% after recovery for 2 weeks on control chow. In contrast, recovery in the presence of LKE the % myelinated axons increased to 65%, which is close to the maximal recovery reported in other studies even after 8-12 weeks of recovery on normal chow. Average myelin thickness after recovery on control chow was the same (about 0.50 microns) across all axon calibers, suggesting that a similar and limited extent of wrapping had occurred after 2 weeks. The average myelin thickness was significantly increased after recovery on LKE, and increased linearly with axon size, reaching values similar to control (non CPZ) levels. Likewise LKE led to almost complete normalization of g-ratios. Immunohistochemical staining showed that LKE increased myelin basic protein and proteolipid expression in both the CC and cortex. LKE also increased the total number of Olig2+ oligodendrocytes and mature CC+ myelinating cells in the cortex, but did not increase the number of Olig2+ / Ki67+ proliferating oligodendrocyte progenitor cells (OPCs) suggesting that the effects of LKE were on OPC survival and maturation, but not proliferation. In initial studies to determine the mechanisms of action of LKE we tested if its main protein target, CRMP2 (collapsin response mediator protein 2) plays a role in OPC maturation. Using primary OPCs, we find that disruption of CRMP2 activity (using small peptides) increases basal Ca²⁺ levels and reduces depolarization-induced Ca²⁺ influx. This suggests that by regulating CRMP2 activity, LKE may increase basal Ca²⁺ needed for OPC maturation, and prevent excessive Ca²⁺ influx under pathological conditions thus reducing apoptosis. Together these findings suggest that LKE has potential to increase remyelination in MS and other neurological diseases associated with demyelination.

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14. Enteric glial activity and S100B secretion during gut inflammation

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Enteric glia are unique type of neuroglia that regulate enteric nervous system (ENS) homeostasis. Here, we tested the hypothesis that glial S100B secretion is altered by inflammation and that impairing S100B synthesis disrupts ENS function. Acute inflammation was studied using the 2,4-dinitrobenzene sulfonic acid (DNBS) mouse model of colitis (5mg/0.1ml). Longitudinal Muscle-Myenteric Plexus (LMMP) from control and DNBS C57Bl/6 mice were prepared and S100B content in their supernatants was measured. Glial activity was assessed through Ca²⁺ imaging using colonic myenteric plexus of *Wnt1Cre^{GCaMP5g-tdTom}* mice at 7 days post-DNBS challenged with ADP (100uM). S100B production was modulated by incubating LMMP with the S100B mRNA inhibitor arundic acid (AA; 50uM; 1h). Controls were incubated for an equal time in Krebs. LMMP supernatants from inflamed mice displayed less S100B compared to controls. After 1w of DNBS induction inflamed LMMP incubated with AA presented less percentage of responsive glial cells compared to DNBS incubated with Krebs solution and control groups. Besides, enteric glia from the DNBS group incubated with AA displayed decreased peak glial responses compared to controls. These data show that inflammation decreases S100B release from myenteric glia, impairs glia response to purines and that this effect is exacerbated when S100B synthesis is blocked by AA. Given the effects of AA on glial excitability in the inflammatory context, S100B may play role in regulating ENS excitability following inflammation.

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