The 13th Great Lakes Glia Meeting

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

Report

The 13th BiAnnual Great Lakes Glial Meeting was held in Traverse City, Michigan, USA from October 9th to 11th, 2022, in the newly renovated Park Plaza Hotel. Traverse City is located in the upper part of Michigan, with its' sea coast bounding on Lake Michigan; providing a scenic and relaxing venue for a scientific meeting.

The boat Marina just across the street from the Park Plaza Hotel

The GLG meeting is a small conference that focuses on current research in glial cell biology. The array of functions that glial cells (astrocytes, microglia, oligodendrocytes, Schwann cells) play in health and disease is constantly increasing. GLG meetings bring together scientists with common interests, leading to a better understanding of these cells. This year's meeting included 1 keynote speaker (Keith Murai) who presented a talk on the ultrastructure of astrocytes. Twenty other talks were presented along with one poster session that was up for the entire meeting. Sessions covered recent findings in the areas of oligodendrocytes in health and disease; astrocyte diversity, metabolism, and reactivity; microglial activation in AD, demyelination, and alcohol abuse; glial:neuronal interactions; and glial cells responses to demyelinating conditions.

ISN Support

The ISN has supported the GLG meetings for several years since 2015. This year ISN support was acknowledged in the opening remarks made by Doug Feinstein, and in the Program Book. This support allowed us to reduce the registration fee for students from $175 to $100, and provide a full waiver of the registration fees for 5 student speakers. In addition, all student participants received a $100 rebate on their hotel costs.

Organization

The scientific organizing committee for the 13th biannual GLG meeting included Douglas Feinstein (University Illinois, Chicago, IL, USA); Pamela Knapp (Virginia Commonwealth University, Richmond, VA, USA), and Robert Skoff (Wayne State University, Detroit, MI, USA). The scientific organizers were greatly assisted by Denise Bessert and Lisa Pope, both from Wayne State University. The meeting website was designed and is maintained by Minetta Gardinier (University of Iowa, Iowa City, USA).

Attendance and Program

Approximately 63 attendees were present, of which 23 were students (undergraduate, graduate, and post doctoral). The majority of attendees were from the USA, with a few from Canada. Although most participants came from the Great Lakes region, this year we had attendees who drove from New York City. The meeting began with an opening reception on Sunday afternoon, followed by the first symposium Sunday evening. The session was opened by Doug Feinstein who made introductory remarks, which included presenting an overview of ISN with a slide deck provided by ISN.

The program for the entire meeting went mostly as planned, with 5 sessions, 1 keynote speaker and posters that were available for viewing for most of the meeting. Unfortunately the 2nd key note speaker (Dr Doug Fields) was unable to attend or present a virtual talk. The first keynote speaker, Dr Keith Murai, was also unable to attend but was able to give a virtual talk which went without any problem.

The 5 sessions consisted of 4 30 minute presentations:

Session 1 Oligodendrocyte Development and Myelination:
Molecular Mechanisms in Health and Disease
Chair: Pablo Paez, University of Buffalo

Session 2 Astrocyte Heterogeneity in Health and Disease
Chairs: Keith Murai, McGill University;
Doug Feinstein, University of Illinois, Chicago

Session 3 Microglia in Health and Disease
Chair: Tyler Ulland, University of Wisconsin-Madison

Session 4 Contribution of Glia-Associated Mechanisms to Pathological Endophenotypes of Complex Behaviors
Chair: Sinead O'Donovan, The University of Toledo

Session 5 Glial Responses to Disease and Therapy in Demyelinating Conditions
Chair: Ernesto Bongarzone, University Illinois, Chicago

Highlights of the meeting

In lieu of Dr Fields presentation (he was not able to present), a poster blitz session was organized to take place at the beginning of the meeting on Monday morning. In this session, all students presenting their work on posters were invited to give a short (up to 3 minute) presentation and allowed one slide, to describe their work and encourage people to view it. Both the students and audience thought this was an excellent addition to the meeting, and several people in the exit survey recommended we continue to do this.

Posters

The posters were put up on the first day of the meeting, and kept up till the last day. There were a total of 16 posters (2 were not included in the program due to late submission), the majority coming from students. Poster viewing time was formally scheduled for one evening. Posters were judged by 8 judges for a variety of qualities (layout, novelty, presentation), each judge taking care of 2 posters. Prizes were awarded to 2 graduate students ($125 each) and one post-doctoral student ($250).

Follow up survey and selected comments:

A post meeting survey was sent out to gather comments for designing the next meeting in 2 years and 12 reponses were received (attached). Several speakers indicated they would be willing to put together a mini-review article based on the session; or organize a session at the next meeting in 2024. Selected comments included:

“Thank you again for the opportunity to present some of our research at the recent Great Lakes Glia Meeting! It was so refreshing to attend a meeting in person and I enjoyed all the scientific exchange. It was a truly wonderful meeting”

“Also, thanks a bunch for organizing the GLG and giving me the opportunity to share our work. I enjoyed meeting everyone a lot and think the conference came at the perfect time, considering that I just started to establish my lab. Very much looking forward to the next GLG meeting.”

“It was a really fun meeting and it was an honor to be asked to speak.”

Other Sponsors

In addition to the generous support of ISN, the 13th GLG meeting also received support from the National Multiple Sclerosis Society, CARES, Chicago Association for Research and Education in Science (a non-profit arm of the Department of Veterans Affairs), and the ASN journal ASN NEURO. Each sponsor was acknowledged at the opening session and in the GLG program book.

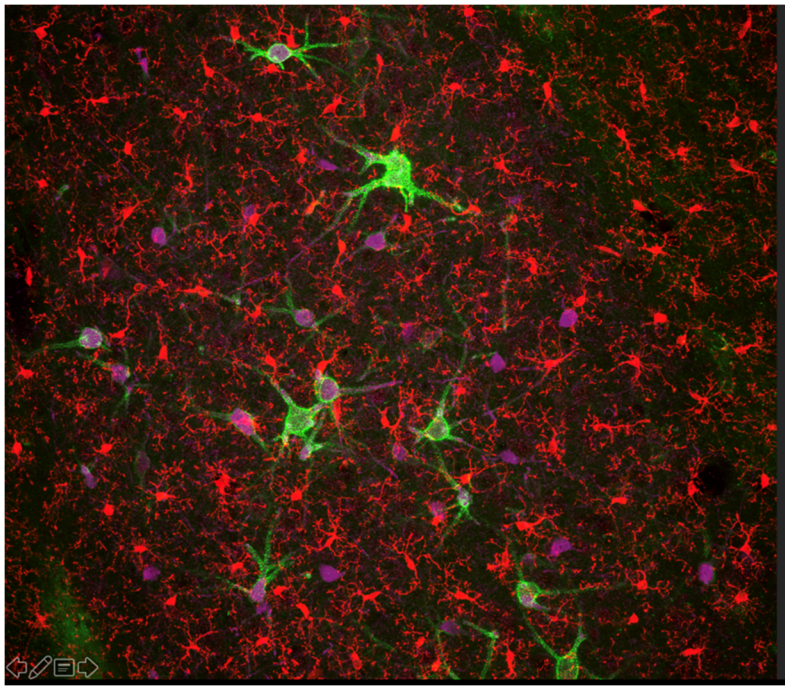
Use of ISN support:

ISN funds ($10,000 total) were used as follows.

<u>For Young Investigators:</u>		
18 students had the registration fee reduced from $175 to $100		$1,350
5 student speakers had the registration fee fully waived		$ 875
23 Students received a $100 reduction in room costs		$2,300
	Total to YI:	**$4,525**
<u>The Remaining $5,475 ISN funds contributed to the following costs:</u>		
16 non-student speakers had their registration fee fully waived (these fees would normally have helped pay for the venue, food, etc..)		$ 4,400
<u>Meeting costs:</u>		
Administration fees:		$ 4,500
Program printing:		$ 725
Office supplies (poster boards/name badges):		$ 580
Poster awards:		$ 500
Honararium (Murai) $750		$ 750
Venue Costs (meals, coffee breaks, room rental)		$15,000
	Total costs:	**$22,045**
<u>Other income sources:</u>		
ISN (not for YIs)		$ 5,475
National Multiple Sclerosis society		$ 5,000
Jesse Brown VA Medical Center / CARES		$ 1,500
ASN NEURO		$ 500
Registration		
18 x $100		$ 1,800
18 x $275		$ 4,950
	Total other income:	**$ 19,225**

Student Attendees who received ISN support: (G, graduate; P, post doctoral)

		Registration	Hotel
1	Jacen Emerson G	$75	$100
2	Jacques Gonzales P	$75	$100
3	Hongjoo An G	$75	$100
4	Marie Hanscom P	$75	$100
5	Ilana Deyneko G	$75	$100
6	Dylann Cordova G	$75	$100
7	Sonu Dobariya G	$75	$100
8	Beatriz Thomasi P	$75	$100
9	Carrie Lynn P	$75	$100
10	Elizabeth Dustin G	$75	$100
11	Edna Suarez Pozos P	$75	$100
12	Wilmarie Morales-Soto G	$75	$100
13	Lorena Yuliana Noriega Gonzalez G	$75	$100
14	Destiny Ogbu G	$75	$100
15	Elizabeth Clawson G	$75	$100
16	Tabitha Peterson P	$75	$100
17	Natalya Pashkova P	$75	$100
18	Chris Ptak P	$75	$100
1	Zila Martinez P (speaker)	$175	$100
2	Sebastian Werneburg P (speaker)	$175	$100
3	Sarah Elzinga P (speaker)	$175	$100
4	Marissa Smail P (speaker)	$175	$100
5	Leandro Marziali P (speaker)	$175	$100
		$2,225	**$2,300**

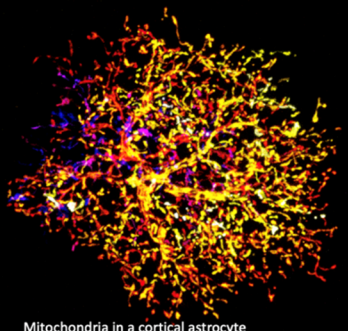


Animal Model of Loss Effects on Microglia and the Extracellular Matrix

Great Lakes Glia 2022
Marissa Smail
10/11/22

University of CINCINNATI

Generation of Astrocyte Diversity: Lessons from Transcriptional Regulation of the Glutamate Transporter 1 (GLT-1)



Mitochondria in a cortical astrocyte
Unpublished, Krizman E. 2021

Zila Martinez-Lozada, Ph.D.

Research Associate
Laboratory of Michael B. Robinson

The Children's Hospital of Philadelphia
University of Pennsylvania

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Committees

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Bob Skoff, Wayne State University, Detroit
Douglas Feinstein, University of Illinois at Chicago
Pamela E. Knapp, Virginia Commonwealth University, Richmond



Fundraising Committee

Bob Skoff, Wayne State University, Detroit
Lisa Pope, Wayne State University, Detroit
Doug Feinstein, University of Illinois at Chicago

Website Committee

Minnetta Gardinier, University of Iowa, Iowa City (webmaster)

Cover Illustrations

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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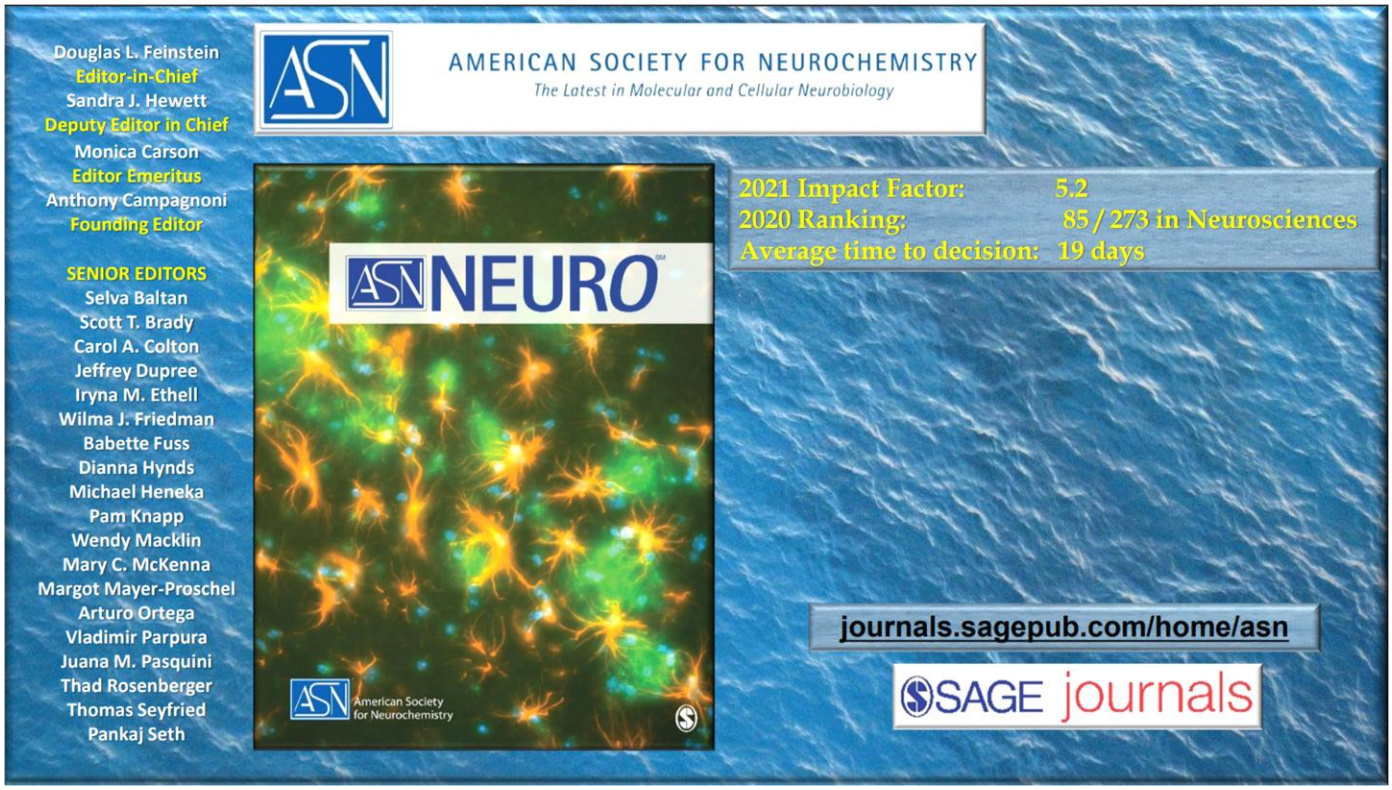

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Chicago Association for Research and Education in Science

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www.CARES-Research.org

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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** **Grandview I**

Keith Murai, McGill University
Astrocyte Heterogeneity and Nanoarchitecture in the CNS

9:45am **Break**

10:00am **Session 2** **Astrocyte Heterogeneity in Health and Disease** **Grandview I**

Co-Chairs: Keith Murai, McGill University
Doug Feinstein, University of Illinois, Chicago

Zila Martinez, The Children's Hospital of Philadelphia
Generation of Astrocyte Diversity: Lessons from Transcriptional Regulation of the Glutamate Transporter 1(GLT1)

José Otero, The Ohio State University College of Medicine
Brainstem Astrocytes and Their Regulation of Autonomic Homeostasis

Ryan Gilbert, Rensselaer Polytechnic Institute
Biomaterial Approaches to Augment Astrocyte Reactivity

Isobel Scarisbrick, Mayo Clinic
Modulating Astrocyte Metabolism to Promote CNS Regeneration

Noon **Free time**

3:30pm **Poster Session** **Grandview II**

4:30pm **Session 3** **Microglia in Health and Disease** **Grandview I**

Chair: Tyler Ulland, University of Wisconsin-Madison

Kathryn Lenz, The Ohio State University
Prenatal Allergic Inflammation, Mast Cell-Microglia Interactions, and Sex-Specific Programming of Motivated Behavior

Tyler Ulland, University of Wisconsin-Madison
Inhibition of the Nlrp3 Inflammasome by 6-Hydroxybutyrate Decreases Alzheimer's Disease Pathology

Sebastian Werneburg, University of Michigan
Microglia and the Elimination and Recovery of Synapses in Demyelinating Disease

Subhash Pandey, University of Illinois
Neuroinflammation Signatures in the Pathophysiology of Alcohol Use Disorder

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**

1. Role of Myrf enhancers in CNS myelination

Author: Hongjoo An [1], Dongkyeong Kim[1], Chuandong Fan[1], and Yungki Park[1],

[1]Institute 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).

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]

[1]University of Wisconsin-Madison, [2]School of Veterinary Medicine, [3]Department of Comparative Biosciences, [4]Stem Cell and Regenerative Medicine Center, [5]Molecular 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.

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

Dylann Cordova-Martinez[1], Chang Hoon Cho[2], Bryen A. Jordan[1]

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

4. The Role of Sulfatide in Oligodendrocytes

Dustin E[1,2,3], Flounlacker K[1,3], Palavicini J[4], Han X[4], McQuiston R[1], Dupree JL[1,2]

[1]Department of Anatomy and Neurobiology, VCU Medical Center [2]Hunter Holmes McGuire Veterans Affairs Medical Center [3]Virginia Commonwealth University, Neuroscience Curriculum, Richmond [4]UT 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.

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

Ilana Vasilisa Deyneko[1], Chang Hoon Cho[1], Juan Vazquez1, Dylann Cordova-Martinez[1], Abigail U. Carbonell[1], Jaafar O. Tindi[1], Min-Hui Cui[2], Roman Fleysher[2], Craig A. Branch[2], Bryen A. Jordan[1]
[1]Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, NY, USA. [2]Department 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.

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 inTG2 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.

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

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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.

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

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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-1β showed a 5-fold increase ($n = 4$-5, $p<0.05$). Enteric glia contribute to the production of IL-1β 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-1β immunolabeling data at peak colitis ($n = 3$, $p<0.05$). IL-1β increased glial dye uptake under basal conditions and potentiated ADP-stimulated dye uptake by 24% ($n = 10$-15 ganglia, form 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-1β 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 synthetizing enzyme, cycloexogenasae-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-1β. 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-1β as a candidate glial mediator that has the capacity to affect nociceptors by influencing glial Cx43-dependent PGE2 release.

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

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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.

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.

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

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 is 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.

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 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.

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 Ca^{2+} imaging using colonic myenteric plexus of *Wnt1Cre*$^{GCaMP5g\text{-}tdTom}$ 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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Notes

Notes

Please check the column you think is best , and add any comments or suggestions
Please return to Doug Feinstein at dlfeins@uic.edu

General Questions:	Great	Good	Bad	Comments
I. Registration				
1. Cost				
2. Ease				
3. Other				
II. Venue				
1. Traverse City				
2. Hotel rooms				
3. Hotel price / value				
4. Auditorium				
5. Other				
III. Food				
1. Breakfast				
2. Coffee breaks				
3. Other				
IV. Program				
1. Keynotes				
2. Sessions				
3. Poster Sessions				
4. Poster Blitz				
5. Length				
6. Zoom talks				
7. Other				

Specifc Questions:	
1. If we had to change city, where would you suggest?	
1a. If yes, suggestions for a venue in or near that location?	
2. Should Tuesday sessions have only 3 speakers ?	
3. Suggestions for Keynote speakers for next meeting?	
4. Would you be interested to organize a session for next meeting?	

ID	Source.Name	Subject	Sub Subject	Great	Good	Bad	Comments
1	Copy of GLG 2022 exit survey_1021221104a.x	Program	1. Keynotes				I didn't actually attend keynote because of the Zoom format
2	GLG 2022 exit survey_ZML.xlsx	Program	1. Keynotes	X			It was a shame that the keynote speakers couldn't be there but it was great
3	GLG 2022 exit surve3y.xlsx	Program	1. Keynotes	x			it was unfortunatel tha tDoug Fields coul not attend but the student intros were a great idea
4	GLG 2022 exit survey.xlsx	Program	1. Keynotes	x			One key note may be sufficient and allows trainees more room for presentation
5	GLG 2022 exit survey 103122 ll15A.xlsx	Program	1. Keynotes	x			Sad to miss the other keynote - could it be a future webinar?
6	Gulbarsen GLG 2022 exit survey.xlsx	Program	1. Keynotes		X		Would have been better in person but oh well.
7	AIB GLG 2022 exit survey.xlsx	Program	1. Keynotes	X			
8	Gow GLG 2022 exit survey.xlsx	Program	1. Keynotes	X			
9	GLG 2022 exit survey2.xlsx	Program	1. Keynotes	X			
10	Pablo GLG 2022 exit survey1.xlsx	Program	1. Keynotes	XXX			
11	GLG 2022 exit survey_102122845a.xlsx	Program	1. Keynotes		X		
12	GLG 2022 exit survey.xlsx	Program	2. Sessions	x			I would encourage PIs more clearly to bring their trainees to the meeting when advertising it.
13	GLG 2022 exit survey_ZML.xlsx	Program	2. Sessions	X			that you accomodated Dr. Murai talk as a virtual presentation
14	Gulbarsen GLG 2022 exit survey.xlsx	Program	2. Sessions			X	The science wasn't necessarily bad but it would have been nice to see a bit more diversity in terms of the topics and institutions reporesented. Some sessions were overrepresented by individuals from a single institution. I think we should be striving for a bit more diversity in terms of thought.
15	AIB GLG 2022 exit survey.xlsx	Program	2. Sessions	X			
16	Gow GLG 2022 exit survey.xlsx	Program	2. Sessions	X			
17	GLG 2022 exit survey2.xlsx	Program	2. Sessions	X			
18	Copy of GLG 2022 exit survey_1021221104a.x	Program	2. Sessions	x			
19	GLG 2022 exit surve3y.xlsx	Program	2. Sessions	x			
20	GLG 2022 exit survey 103122 ll15A.xlsx	Program	2. Sessions	x			
21	Pablo GLG 2022 exit survey1.xlsx	Program	2. Sessions	XXX			
22	GLG 2022 exit survey_102122845a.xlsx	Program	2. Sessions		X		
23	Pablo GLG 2022 exit survey1.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				4 spaeakers is OK
24	Gow GLG 2022 exit survey.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				current is good. One more session on Sunday, little extra cost
25	GLG 2022 exit survey_ZML.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				I think that was good as it was, however chair of the sessions need to do a better job at keeping the schedule, let know speakers if they run out of time and if they do, then there is no time for questions.
26	GLG 2022 exit survey_1021221254p.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				maybe; it did not really matter much
27	GLG 2022 exit survey.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				N/A
28	GLG 2022 exit survey_102122845a.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				No
29	GLG 2022 exit survey 103122 ll15A.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				No need to decrease - so many great talks
30	GLG 2022 exit surve3y.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				no, if the session is reduced fewer people will stay
31	Copy of GLG 2022 exit survey_1021221104a.x	Program	2. Should Tuesday sessions have only 3 speakers ?				the Tuesday session was fine as scheduled! It was good because the people who were interested in the final session could stay and enjoy it, and it is fine that some people chose to leave at lunch and skip the final session. Leave it up to the attendees.
32	Gulbarsen GLG 2022 exit survey.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				This would be a good idea. Wrap it up by noon.
33	GLG 2022 exit survey2.xlsx	Program	2. Should Tuesday sessions have only 3 speakers ?				Yes
34	Gulbarsen GLG 2022 exit survey.xlsx	Program	3. Poster Sessions		X		It would have been great to have a refreshment hour in the poster session room.
35	Copy of GLG 2022 exit survey_1021221104a.x	Program	3. Poster Sessions		x		not enough posters but that is because of pandemic and I'm sure will be better next time
36	AIB GLG 2022 exit survey.xlsx	Program	3. Poster Sessions	X			Wine was nice during posters
37	Gow GLG 2022 exit survey.xlsx	Program	3. Poster Sessions	X			
38	GLG 2022 exit survey2.xlsx	Program	3. Poster Sessions	X			
39	GLG 2022 exit surve3y.xlsx	Program	3. Poster Sessions	x			
40	GLG 2022 exit survey.xlsx	Program	3. Poster Sessions	x			
41	GLG 2022 exit survey_ZML.xlsx	Program	3. Poster Sessions	X			
42	GLG 2022 exit survey 103122 ll15A.xlsx	Program	3. Poster Sessions	x			
43	GLG 2022 exit survey_102122845a.xlsx	Program	3. Poster Sessions		X		
44	Pablo GLG 2022 exit survey1.xlsx	Program	3. Poster Sessions		XXX		
45	Gow GLG 2022 exit survey.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Chris Filley, MD, Colorado, Christopher.Filley@cuanschutz.edu
46	Pablo GLG 2022 exit survey1.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Dwight Edward Bergles, Laura Feltri
47	GLG 2022 exit survey2.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Ethan Hughes, Jeffrey Huang
48	GLG 2022 exit survey_102122845a.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Eva Feldman
49	GLG 2022 exit survey_1021221254p.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				french Constant; Richardson; Franklin
50	Gulbarsen GLG 2022 exit survey.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Jaideep Bains (Will be moving from Calgary to Ontario), Sarah Kucenas, Fievos Christofi
51	GLG 2022 exit survey_ZML.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				Michelle Monje
52	GLG 2022 exit survey.xlsx	Program	3. Suggestions for Keynote speakers for next meeting?				N/A
53	AIB GLG 2022 exit survey.xlsx	Program	4. Poster Blitz	X			Excellent!
54	GLG 2022 exit survey 103122 ll15A.xlsx	Program	4. Poster Blitz		x		Good this year with little prep - good to include in future too!
55	GLG 2022 exit survey.xlsx	Program	4. Poster Blitz		x		More previous notice (which was not possible this time) would have made this more informative. \
56	GLG 2022 exit survey_102122845a.xlsx	Program	4. Poster Blitz				N.A.
57	GLG 2022 exit survey2.xlsx	Program	4. Poster Blitz		X		This was a great idea.
58	Gulbarsen GLG 2022 exit survey.xlsx	Program	4. Poster Blitz		X		This was fun
59	Gow GLG 2022 exit survey.xlsx	Program	4. Poster Blitz	X			
60	Copy of GLG 2022 exit survey_1021221104a.x	Program	4. Poster Blitz	x			
61	GLG 2022 exit surve3y.xlsx	Program	4. Poster Blitz	x			
62	GLG 2022 exit survey_ZML.xlsx	Program	4. Poster Blitz	X			
63	Pablo GLG 2022 exit survey1.xlsx	Program	4. Poster Blitz	XXX			
64	Gow GLG 2022 exit survey.xlsx	Program	4. Would you be interested to organize a session for next meeting?				maybe
65	GLG 2022 exit survey.xlsx	Program	4. Would you be interested to organize a session for next meeting?				Maybe
66	GLG 2022 exit survey_ZML.xlsx	Program	4. Would you be interested to organize a session for next meeting?				Maybe, would need to check my other commitments at that point
67	Gulbarsen GLG 2022 exit survey.xlsx	Program	4. Would you be interested to organize a session for next meeting?				Sure. This would be fun.
68	Pablo GLG 2022 exit survey1.xlsx	Program	4. Would you be interested to organize a session for next meeting?				Yes
69	GLG 2022 exit survey2.xlsx	Program	4. Would you be interested to organize a session for next meeting?				Yes
70	GLG 2022 exit surve3y.xlsx	Program	4. Would you be interested to organize a session for next meeting?				yes
71	Gow GLG 2022 exit survey.xlsx	Program	5. Length		X		little too short
72	Gulbarsen GLG 2022 exit survey.xlsx	Program	5. Length	X			Perfect
73	AIB GLG 2022 exit survey.xlsx	Program	5. Length	X			
74	Copy of GLG 2022 exit survey_1021221104a.x	Program	5. Length	x			
75	GLG 2022 exit surve3y.xlsx	Program	5. Length	x			
76	GLG 2022 exit survey.xlsx	Program	5. Length	x			
77	GLG 2022 exit survey_ZML.xlsx	Program	5. Length	X			
78	GLG 2022 exit survey 103122 ll15A.xlsx	Program	5. Length	x			
79	Pablo GLG 2022 exit survey1.xlsx	Program	5. Length	XXX			
80	GLG 2022 exit survey_102122845a.xlsx	Program	5. Length		X		
81	GLG 2022 exit survey2.xlsx	Program	5. Length		X		
82	GLG 2022 exit survey_1021221254p.xlsx	Program	5. Other				with such cohort of ~60 people+/- 10, we can have better arrangements in a smaller, cozier, food-friendly inn rather that this place which looks halted in time (sorry but I had serious issues with food and cleanliness of bathrooms/rooms
83	Gulbarsen GLG 2022 exit survey.xlsx	Program	5. Other	X			
84	GLG 2022 exit survey2.xlsx	Program	6. Zoom talks			X	Hard to focus...
85	Copy of GLG 2022 exit survey_1021221104a.x	Program	6. Zoom talks			x	I don't like zoom talks
86	AIB GLG 2022 exit survey.xlsx	Program	6. Zoom talks		X		In-person talk always best
87	GLG 2022 exit survey.xlsx	Program	6. Zoom talks		x		In-person talks are preferred to allow for better interactions after the talk
88	GLG 2022 exit survey_102122845a.xlsx	Program	6. Zoom talks				N.A.
89	Gulbarsen GLG 2022 exit survey.xlsx	Program	6. Zoom talks		X		Zoom is ok when necessary but in person is always better
90	Gow GLG 2022 exit survey.xlsx	Program	6. Zoom talks	x			
91	GLG 2022 exit survey_ZML.xlsx	Program	6. Zoom talks	X			
92	GLG 2022 exit survey 103122 ll15A.xlsx	Program	6. Zoom talks	x			
93	Pablo GLG 2022 exit survey1.xlsx	Program	6. Zoom talks		XXX		
94	GLG 2022 exit survey_1021221254p.xlsx	Program	7. Other				Missed opportunity for the myelin plasticity keynote :(
95	GLG 2022 exit survey_102122845a.xlsx	Program	7. Other				The timing of the sessions was not ideal, and was compunded by not keeping to time.
96	GLG 2022 exit survey_ZML.xlsx	Registration	2. Ease		X		An online sistem might be better than through emails exchange
97	Gulbarsen GLG 2022 exit survey.xlsx	Registration	2. Ease		X		Sending the credit card on the form by email is not great. It would be better to have some kind of online payment platform.
98	AIB GLG 2022 exit survey.xlsx	Registration	2. Ease	X			
99	Gow GLG 2022 exit survey.xlsx	Registration	2. Ease	X			
100	GLG 2022 exit survey_102122845a.xlsx	Registration	2. Ease	X			
101	GLG 2022 exit survey2.xlsx	Registration	2. Ease	X			
102	GLG 2022 exit surve3y.xlsx	Registration	2. Ease	x			
103	GLG 2022 exit survey.xlsx	Registration	2. Ease	x			
104	GLG 2022 exit survey 103122 ll15A.xlsx	Registration	2. Ease	x			
105	Pablo GLG 2022 exit survey1.xlsx	Registration	2. Ease	XXX			
106	Copy of GLG 2022 exit survey_1021221104a.x	Registration	2. Ease		x		
107	Copy of GLG 2022 exit survey_1021221104a.x	Registration	3. Other			x	hotel registration challenge
111	Gulbarsen GLG 2022 exit survey.xlsx	Venue	1. Breakfast		X		Breakfast was good but for people who don't eat meat it would be nice to have more options.
112	GLG 2022 exit survey_1021221254p.xlsx	Venue	1. Breakfast				food was tasteless, cold or overheated, veggies over OVER cooked, without texture, eggs seemed plastiware and old
113	AIB GLG 2022 exit survey.xlsx	Venue	1. Breakfast		X		More fruit salads! Keep the pastries
114	GLG 2022 exit surve3y.xlsx	Venue	1. Breakfast	x			
115	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	1. Breakfast	x			
116	Pablo GLG 2022 exit survey1.xlsx	Venue	1. Breakfast	XXX			
117	Gow GLG 2022 exit survey.xlsx	Venue	1. Breakfast		X		
118	GLG 2022 exit survey_102122845a.xlsx	Venue	1. Breakfast		X		
119	GLG 2022 exit survey2.xlsx	Venue	1. Breakfast		X		
120	Copy of GLG 2022 exit survey_1021221104a.x	Venue	1. Breakfast		x		
121	GLG 2022 exit survey.xlsx	Venue	1. Breakfast		x		
122	GLG 2022 exit survey_ZML.xlsx	Venue	1. Breakfast		X		
123	GLG 2022 exit survey_ZML.xlsx	Venue	1. Cost	X			I was super impresed with the registration cost for trainnes, well done!
124	AIB GLG 2022 exit survey.xlsx	Venue	1. Cost	X			
125	Gow GLG 2022 exit survey.xlsx	Venue	1. Cost	X			
126	GLG 2022 exit survey_102122845a.xlsx	Venue	1. Cost	X			
127	GLG 2022 exit survey2.xlsx	Venue	1. Cost	X			
128	GLG 2022 exit surve3y.xlsx	Venue	1. Cost	x			
129	GLG 2022 exit survey.xlsx	Venue	1. Cost	x			
130	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	1. Cost	x			
131	Pablo GLG 2022 exit survey1.xlsx	Venue	1. Cost	XXX			
132	Gulbarsen GLG 2022 exit survey.xlsx	Venue	1. Cost		X		
133	Copy of GLG 2022 exit survey_1021221104a.x	Venue	1. Cost		x		
134	GLG 2022 exit survey_ZML.xlsx	Venue	1. If we had to change city, where would you suggest?				A city with a larger airport (Detroit, Chicago, ...)
135	Pablo GLG 2022 exit survey1.xlsx	Venue	1. If we had to change city, where would you suggest?				Ann Harbor
136	GLG 2022 exit surve3y.xlsx	Venue	1. If we had to change city, where would you suggest?				I like Traverse City. I don't have an alternative suggestion
137	GLG 2022 exit survey.xlsx	Venue	1. If we had to change city, where would you suggest?				I liked Traverse City
138	Gulbarsen GLG 2022 exit survey.xlsx	Venue	1. If we had to change city, where would you suggest?				I think Traverse City is a great location, but if we had to change Holland (MI) could be an option
139	Copy of GLG 2022 exit survey_1021221104a.x	Venue	1. If we had to change city, where would you suggest?				Lake Geneva
140	Gow GLG 2022 exit survey.xlsx	Venue	1. If we had to change city, where would you suggest?				Muskegon
141	GLG 2022 exit survey_1021221254p.xlsx	Venue	1. If we had to change city, where would you suggest?				TC is OK. But the venue for the meeting is NOT OK.
108	GLG 2022 exit survey2.xlsx	Venue	1a. If yes, suggestions for a venue in or near that location?				Buffalo, NY
109	Gow GLG 2022 exit survey.xlsx	Venue	1a. If yes, suggestions for a venue in or near that location?				Shoreline Inn & conference center
110	GLG 2022 exit survey_1021221254p.xlsx	Venue	1a. If yes, suggestions for a venue in or near that location?				There should be much much better places for similar price
142	GLG 2022 exit survey_ZML.xlsx	Venue	1. Traverse City		X		Difficult access, maybe a city with easier access would atract more participants
143	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	1. Traverse City	x			Love this location for fall!
144	AIB GLG 2022 exit survey.xlsx	Venue	1. Traverse City	X			
145	Gulbarsen GLG 2022 exit survey.xlsx	Venue	1. Traverse City	X			
146	Gow GLG 2022 exit survey.xlsx	Venue	1. Traverse City	X			
147	GLG 2022 exit survey_102122845a.xlsx	Venue	1. Traverse City	X			
148	Copy of GLG 2022 exit survey_1021221104a.x	Venue	1. Traverse City	x			
149	GLG 2022 exit surve3y.xlsx	Venue	1. Traverse City	x			
150	GLG 2022 exit survey.xlsx	Venue	1. Traverse City	x			
151	Pablo GLG 2022 exit survey1.xlsx	Venue	1. Traverse City	XXX			
152	GLG 2022 exit survey2.xlsx	Venue	1. Traverse City		X		
153	Gow GLG 2022 exit survey.xlsx	Venue	2. Coffee breaks	X			
154	GLG 2022 exit surve3y.xlsx	Venue	2. Coffee breaks	x			
155	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	2. Coffee breaks	x			
156	Pablo GLG 2022 exit survey1.xlsx	Venue	2. Coffee breaks	XXX			
157	AIB GLG 2022 exit survey.xlsx	Venue	2. Coffee breaks		X		
158	Gulbarsen GLG 2022 exit survey.xlsx	Venue	2. Coffee breaks		X		
159	GLG 2022 exit survey_102122845a.xlsx	Venue	2. Coffee breaks		X		
160	GLG 2022 exit survey2.xlsx	Venue	2. Coffee breaks		X		
161	Copy of GLG 2022 exit survey_1021221104a.x	Venue	2. Coffee breaks		x		
162	GLG 2022 exit survey.xlsx	Venue	2. Coffee breaks		x		
163	GLG 2022 exit survey_ZML.xlsx	Venue	2. Coffee breaks		X		
164	GLG 2022 exit survey_102122845a.xlsx	Venue	2. Hotel rooms				N.A.
165	GLG 2022 exit survey2.xlsx	Venue	2. Hotel rooms		X		Temperature setting was tricky.
166	AIB GLG 2022 exit survey.xlsx	Venue	2. Hotel rooms	X			
167	Gow GLG 2022 exit survey.xlsx	Venue	2. Hotel rooms	X			
168	Copy of GLG 2022 exit survey_1021221104a.x	Venue	2. Hotel rooms	x			
169	GLG 2022 exit surve3y.xlsx	Venue	2. Hotel rooms	x			
170	GLG 2022 exit survey_ZML.xlsx	Venue	2. Hotel rooms	X			
171	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	2. Hotel rooms	x			
172	Gulbarsen GLG 2022 exit survey.xlsx	Venue	2. Hotel rooms		X		
173	GLG 2022 exit survey.xlsx	Venue	2. Hotel rooms		x		
174	Pablo GLG 2022 exit survey1.xlsx	Venue	2. Hotel rooms		XXX		
175	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	3. Hotel price / value	x			Better than big city meetings!
176	GLG 2022 exit survey_102122845a.xlsx	Venue	3. Hotel price / value				N.A.
177	AIB GLG 2022 exit survey.xlsx	Venue	3. Hotel price / value	X			
178	Gow GLG 2022 exit survey.xlsx	Venue	3. Hotel price / value	X			
179	GLG 2022 exit surve3y.xlsx	Venue	3. Hotel price / value	x			
180	GLG 2022 exit survey_ZML.xlsx	Venue	3. Hotel price / value	X			
181	Gulbarsen GLG 2022 exit survey.xlsx	Venue	3. Hotel price / value		X		
182	GLG 2022 exit survey2.xlsx	Venue	3. Hotel price / value		X		
183	Copy of GLG 2022 exit survey_1021221104a.x	Venue	3. Hotel price / value		x		
184	GLG 2022 exit survey.xlsx	Venue	3. Hotel price / value		x		
185	Pablo GLG 2022 exit survey1.xlsx	Venue	3. Hotel price / value		XXX		
186	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	3. Other	x			Opening dinner/reception was lovely
187	GLG 2022 exit survey.xlsx	Venue	3. Other			x	To few options for vegeterians (breakfast and dinner)
188	Gulbarsen GLG 2022 exit survey.xlsx	Venue	3. Other		X		
189	Gulbarsen GLG 2022 exit survey.xlsx	Venue	3. Other		X		
190	GLG 2022 exit survey 103122 ll15A.xlsx	Venue	4. Auditorium	x			A little more seating to help people spread out perhaps
191	AIB GLG 2022 exit survey.xlsx	Venue	4. Auditorium	X			
192	Gulbarsen GLG 2022 exit survey.xlsx	Venue	4. Auditorium	X			
193	Gow GLG 2022 exit survey.xlsx	Venue	4. Auditorium	X			
194	GLG 2022 exit survey_102122845a.xlsx	Venue	4. Auditorium	X			
195	Copy of GLG 2022 exit survey_1021221104a.x	Venue	4. Auditorium	x			
196	GLG 2022 exit surve3y.xlsx	Venue	4. Auditorium	x			
197	GLG 2022 exit survey.xlsx	Venue	4. Auditorium	x			
198	GLG 2022 exit survey_ZML.xlsx	Venue	4. Auditorium	X			
199	Pablo GLG 2022 exit survey1.xlsx	Venue	4. Auditorium	XXX			
200	GLG 2022 exit survey2.xlsx	Venue	4. Auditorium		X		