15th Annual GCC Translational Pain Research Conference

March 3-4, 2026

BioScience Research Collaborative
6500 Main St.
Houston, Texas

Follow us on X:

@GCC_Research

For today's conference use hashtag:

#GCCTPR26

Follow us on LinkedIn:

Gulf Coast Consortia - Research

Gulf Coast Consortia
QUANTITATIVE BIOMEDICAL SCIENCES

The Gulf Coast Consortia (GCC), located in Houston, Texas, is a dynamic, multi-institution collaboration of basic and translational scientists, researchers, clinicians, and students in the quantitative biomedical sciences, who benefit from joint training programs, topic-focused research consortia, shared facilities and equipment, and exchange of scientific knowledge. Working together, GCC member institutions provide a cutting-edge collaborative training environment and research infrastructure beyond the capability of any single institution. GCC research consortia gather interested faculty around research foci within the quantitative biomedical sciences and currently include Translational Pain Research, Aging and Geroscience Research, Antimicrobial Resistance, Cellular and Molecular Biophysics, Innovative Drug Discovery and Development, Immunology, Mental Health Research, Integrative Development, Regeneration and Repair, and Single Cell & Spatial Omics. GCC training programs focus on Antimicrobial Resistance Training, Biomedical Informatics, Cancer Therapeutics, Interdisciplinary Pharmacology Scientists, Molecular Basis of Infectious Diseases, Molecular Biophysics, and Precision Environmental Health Sciences. Current members include Baylor College of Medicine, Rice University, University of Houston, The University of Texas Health Science Center at Houston, The University of Texas Medical Branch at Galveston, The University of Texas MD Anderson Cancer Center, The Institute of Biosciences and Technology of Texas A&M Health Science Center, and Houston Methodist Research Institute.

GCCTPR Executive Steering Committee

Thank you to our Silver Sponsors

10x GENOMICS

Novogene

Advancing Genomics, Improving Life

Thank you to our Bronze Sponsor

SARTORIUS

March 3-4, 2026

Day 1

10:00-10:05	Welcome **Autumn Marsden**, Gulf Coast Consortia **Andrew Shepherd**, MD Anderson Cancer Center
Moderator	**Jun-Ho La**, University of Texas Medical Branch at Galveston
10:05-11:05	Keynote Presentation *Decoding a Molecular QR Code of Pain: From Mechanism to Therapeutic Translation* **Rajesh Khanna**, University of Florida, College of Medicine
11:05-12:10	Local Talks, Session
Moderator:	**Andrew Shepherd**, MD Anderson Cancer Center
11:05-11:20	*Nav1.8 inhibition by Suzetrigine Reduces Spontaneous Activity in Human Sensory Neurons* **Mario Heles**, MD Anderson Cancer Center
11:20-11:35	*Targeting protein-protein interactions (PPI) between Nav1.7 and FGF13 as a first-in-class mechanism for safe and selective analgesia* **Marijn Lijfijt**, Ion TX Inc.
11:35-11:50	*Nociceptor-Restricted Cannabinoid Receptor 1 Induces Antinociception in a Preclinical Model of Persistent Painful Diabetic Peripheral Neuropathy* **Audrey Green**, University of Texas, Dallas
11:50-12:05	*Anatomy, Projectome, and Functional Annotation of Mouse Spinal Cord Neural Diversity* **Helen Poldsam**, University of Texas Southwestern Medical Center
12:05-1:15	Networking lunch
1:15-2:15	Local Talks, Session II
Moderator:	**Peter Grace**, MD Anderson Cancer Center
1:15-1:30	*Sialylated Immunoglobulin G Relieves Established Mechanical Allodynia After Peripheral Nerve Injury* **Nathan Fiore**, MD Anderson Cancer Center
1:30-1:45	*Mast Cell Specific GPCR Regulates UTI Immune Response and Pain* **Dustin Green**, University of Texas Medical Branch at Galveston
1:45-2:00	*Gp120 Derived Extracellular Vesicle (sEV) Induced HIV-1 Associated Pain* **Subo Yuan**, University of Texas Medical Branch at Galveston
2:00-2:15	*Translating Auricular Point Acupressure from Mechanism to Real-World Pain Care* **Jennifer Kawi**, University of Texas Health Science Center, Houston

2:15-2:30 Networking break

Moderator: **Pat Dougherty**, MD Anderson Cancer Center

2:30-3:00 Data Blitz Presentations

DRG Meningeal Tertiary Lymphoid Structures Function as a Pro-Nociceptive Niche through B cell–Dependent Germinal Center Formation and Plasma Cell Differentiation After Peripheral Nerve Injury
Tusar Acharya, MD Anderson Cancer Center, Poster #19

Enhancing Sensory Motor Swallow Brain Networks Through Individualized Neuromodulation – A Feasibility Intervention for Oropharyngeal Cancer Pain Following Treatment
Michael Davis, Baylor College of Medicine, Poster #3

Inflammatory Neuropathy in Mouse Models of Colorectal Cancer: Implications for Chemotherapy-Induced Peripheral Neuropathy
Caitlyn Gaffney, MD Anderson Cancer Center, Poster #6

Life-long Hydroxyurea Treatment Decreases Chronic Pain in Sickle Cell Disease Mice by Limiting TRPA1 Sensitization in Peripheral Nociceptors
Nya Gayluak, UT at Dallas, Poster #8

Influence of Sex and Estrous Stage on Sensory and Affective Responding of Pain
Kaitlyn Little, University of Houston, Poster #10

Sex-Divergent Transcriptomic Regulation of Sensory Neurons in Oral Cancer
Jaclyn Merlo, UT San Antonio, Poster #11

Understanding the mechanism of Painful Radiation Induced Oral Mucositis (RIOM)
Priya Saraswathi Narayanan, UT at San Antonio, Poster #13

3:00-4:00 Poster session I, even # posters-event hall

4:00-5:00 Poster session II, odd # posters-event hall

5:00-6:00 Reception-Pre-function

<u>Day 2</u>

8:00-8:55 Mentoring Breakfast - event hall
Rajesh Khanna, University of Florida, College of Medicine
Angelika Lampert, Uniklinik RWTH
Carmen Dessauer, University of Texas Health Science Center, Houston
Nele Haelterman, Baylor College of Medicine
Andrew Shepherd, MD Anderson Cancer Center

9:00-9:05 Welcome
Andrew Shepherd, MD Anderson Cancer Center

Moderators: **Diana Tavares-Ferreira**, University of Texas, Dallas
Ted Price, University of Texas, Dallas

9:05-10:05 Keynote Presentation
Decoding Neuropathic Pain: Sodium Channels and Sensory Neuron Subtypes
Angelika Lampert, Uniklinik RWTH

10:05-11:05 Local Talks, Session III

Moderator: **Terry Walters**, University of Texas Health Science Center, Houston

10:05-10:20 *Pain-Associated Nociceptor Hyperexcitability in a Mouse Model of Pseudoachondroplasia (mPSACH) Depends Upon Activity of Nav1.8 Channels*
Karen Posey, University of Texas Health Science Center, Houston

10:20-10:35 *Spatial Mapping of Nociceptive Neuronal Subtypes in the Mouse Bladder*
Ridma Sirimanna, University of Texas, Dallas

10:35-10:50 *Frontal Alpha Activity and its Dynamic Modulation During Cognitive Pain Processing in Chronic Pain*
Swathi Suresh, Tarleton State University

10:50-11:05 *Pain and Persistent Opioid Use Signatures After Trauma*
Consuelo Walss-Bass, University of Texas Health Science Center, Houston

11:05-11:25 Networking break

11:25-12:25 Local Talks, Session IV

Moderator: **Nele Haelterman**, Baylor College of Medicine

11:25-11:40 *Therapeutic Targeting of the Spinal Cord Output Cell-Type Map of Pain States*
Vivek Dubey, University of Texas Southwestern Medical Center

11:40-11:55 *Spatial Atlas of the Mouse Trigeminal Ganglion*
Tingting Yang, Baylor College of Medicine

11:55-12:10 *Deciphering the Molecular Mechanisms of Joint Pain in Equine Osteoarthritis via snRNA-seq*
Jun Wang, Baylor College of Medicine

12:10-12:25 *Evolutionary Architecture of Tongue Cell Types Revealed by Cross-Species Single-Cell Transcriptomics: Implications for Oral Sensation and Pain*
Cristal Villalba, University of Texas Health, San Antonio

12:25-12:30 Presentation of awards and closing remarks
Andrew Shepherd, MD Anderson Cancer Center

Rajesh Khanna, PhD
Richard and Thelma O.C. Barney Term Professor
Pharmacology & Therapeutics
University of Florida College of Medicine

Decoding a Molecular QR Code of Pain: From Mechanism to Therapeutic Translation

Dr. Rajesh Khanna is the Richard and Thelma O.C. Barney Term Professor in the Department of Pharmacology & Therapeutics at the University of Florida College of Medicine, where he directs the Center for Advanced Pain Therapeutics and Research (CAPToR). A leading expert in chronic pain neurobiology, his research centers on indirect modulation of voltage-gated ion channels (e.g., CaV2.2 and NaV1.7) through regulatory proteins such as CRMP2 and neuropilin-1, pioneering non-opioid therapeutics with enhanced efficacy and reduced side effects. His translational program integrates molecular biology, electrophysiology in human and animal sensory neurons, behavioral pharmacology, and drug discovery across species, yielding candidates like compound 194, CBD3063 and insights into repurposed agents such as (S)-lacosamide. Previously, he held faculty roles and leadership positions at New York University (Director, NYU Pain Research Center), the University of Arizona, and Indiana University School of Medicine. Dr. Khanna co-founded Regulonix LLC to advance non-opioid pain drugs, holds multiple patents, and has garnered national recognition through editorial roles, board service with the United States Association for the Study of Pain. His lab is supported but DoD and NIH funding including HEAL and RF1 awards.

Keynote presenter

Angelika Lampert, MD
Director of the Institute of Neurophysiology
at RWTH Aachen University, German

Decoding Neuropathic Pain: Sodium Channels and Sensory Neuron Subtypes

Angelika Lampert, MD, is the Director of the Institute of Neurophysiology at RWTH Aachen University, Germany. She serves as the Speaker of the Scientific Center for Neuropathic Pain Aachen (SCNAACHEN). Her research focuses on inherited neuropathic pain syndromes linked to sodium channel mutations and other etiologies.
The Lampert lab investigates the biophysics and structure–function relationships of voltage-gated sodium channels and their disease-associated mutations, as well as patient-derived induced pluripotent stem cells (iPSCs) differentiated into peripheral sensory neurons as models of human neuropathies. Using techniques such as Patch-Seq, her team identifies sensory neuron subclasses that contribute to the generation of neuropathic pain and explores the specific roles of sodium channel subtypes in these processes.

Dr. Lampert studied medicine in Jena, Germany, and Strasbourg, France, and completed her MD thesis at the Max Planck Group for Molecular and Cellular Biophysics in Jena in 2003. Following postdoctoral training at Yale University in New Haven, CT, she established her research group in Erlangen, Germany, before moving to Aachen in 2013, where she was appointed full professor.

Keynote presenter

Additional presenters, in alphabetical order

Vivek Dubey, PhD

Postdoctoral Fellow, University of Texas Southwestern Medical Center

Therapeutic Targeting of the Spinal Cord Output Cell-Type Map of Pain States

Dr. Vivek Dubey is a Postdoctoral Fellow in Dr. Allan-Hermann Pool's lab at the University of Texas Southwestern Medical Center. His research focuses on dissecting the neural mechanisms underlying chronic pain and developing new, non-addictive therapeutic strategies for its management. Dr. Dubey earned his PhD in Neuroscience from All India Institute of Medical Sciences (AIIMS), New Delhi, where he investigated the molecular and network mechanisms underlying temporal lobe epilepsy (TLE). His doctoral research integrated neuroanatomical, electrophysiological, and molecular approaches to advance understanding of the pathophysiology of epilepsy.

Nathan Fiore, PhD
Research Scitentist
MD Anderson Cancer Center
Sialylated Immunoglobulin G Relieves Established Mechanical Allodynia After Peripheral Nerve Injury

Dr. Fiore is a research scientist in the Grace Laboratory at University of Texas MD Anderson Cancer Center with a background in neuroscience and neuroimmunology. Completed a PhD at the University of Sydney studying neuroimmunology and neuropathic pain, using rodent models to identify neuroimmune mechanisms underlying individual differences in pain and immune dysfunction in chronic pain patients. He completed postdoctoral training at the University of New South Wales (UNSW), focusing on sex-specific microglial signatures and immunomodulatory treatments in mouse models of peripheral nerve injury and chemotherapy-induced peripheral neuropathy. Currently he is investigating the role of B cells and pro-nociceptive IgG in neuropathic pain following nerve injury.

Audrey Green, BS
PhD Student
University of Texas, Dallas

Nociceptor-Restricted Cannabinoid Receptor 1 Induces Antinociception in a Preclinical Model of Persistent Painful Diabetic Peripheral Neuropathy

Audrey Green obtained her Bachelor of Science in biology from the University of Oklahoma in 2022 and Master of Science in Applied Cognition and Neuroscience from the University of Texas at Dallas in 2024. Audrey is currently a 3rd year PhD student in the Systems and Cellular Neuroscience program at the University of Texas at Dallas. She has worked in Dr. Michael Burton's Neuroimmunology and Behavior lab since 2022 where she studies the antinociceptive properties of peripherally-restricted cannabinoid receptor 1 during neuropathic pain.

Dustin Green, PhD
Assistant Professor
Neurobiology
University of Texas Medical Branch, Galveston
Mast Cell Specific GPCR Regulates UTI Immune Response and Pain

Dr. Green's background is in neuroscience, with a focus on studying pain physiology and immunology. He completed my doctoral training in the lab of Ken Hargreaves at UTHSCSA. He then took a Postdoctoral position in the lab of Xinzhong Dong, at Johns Hopkins. While in the Dong lab he studied a novel family of G protein-coupled receptors in mice called Mrgprs that are involved in contributing to pain and itch. He is currently studying the neuroimmune interactions between immune cells and peripheral afferents, focusing on how Mrgprs contribute to inflammatory pain and itch.

Mario Heles, [PhD
Postdoctoral Fellow
MD Anderson Cancer Center
Nav1.8 Inhibition by Suzetrigine Reduces Spontaneous Activity in Human Sensory Neurons

Dr. Heles is currently a postdoctoral fellow in Dr. Dougherty's laboratory at MD Anderson Cancer Center, where his research focuses on cancer pain and chemotherapy-induced peripheral neuropathy (CIPN). He received his PhD from Charles University in Prague, where he investigated mechanisms of neuropathic pain, with a particular emphasis on understanding why opioids lose efficacy in neuropathic conditions. During his doctoral training, he combined behavioral, molecular, and electrophysiological approaches to dissect nociceptor sensitization and peripheral mechanisms of pain modulation. His current work centers on translational pain research, integrating human tissue studies, electrophysiology, and mechanistic approaches to better understand CIPN and cancer-related pain. Dr. Heles works closely with cancer-neuroscience researchers, particularly in the area of head and neck cancer pain, aiming to bridge preclinical models with human-relevant systems and improve therapeutic strategies for neuropathic and cancer-associated pain.

Jennifer Kawi, PhD, MSN, FNP-BC, CNE, FAAN
Lee and Joseph Jamail Distinguished Professor
Department of Research
University of Texas Health Science Center, Houston
Translating Auricular Point Acupressure from Mechanism to Real-World Pain Care

Dr. Jennifer Kawi's clinical expertise in pain management, having practiced as a board-certified family nurse practitioner, formed the foundation for her scientific inquiry. Her program of research is focused on chronic pain and various biobehavioral socioeconomic factors influencing pain including self-management, pain care disparities, opioid use, and pain interventions involving digital health care, and integrative medicine, specifically auricular point acupressure. Dr. Kawi has received funding from NIH and other external funding sources as a primary investigator. Her most recent one includes a 6.79M UGrant series on "Personalized Auricular Point Acupressure for Chronic Pain Self-Management in Rural Populations."

Marijn Lijffijt, PhD, MBA
Chief Executive Officer
IonTX Inc.,
Targeting Protein-Protein Interactions (PPI) Between Nav1.7 and FGF13 as a First-In-Class Mechanism for Safe and Selective Analgesia

Dr. Marijn Lijffijt is the Chief Executive Officer of IonTX Inc., a preclinical-stage biotech company developing precision neurotherapeutics for neuropsychiatric and neurological disorders through new ways of modulating voltage-gated ion channels. With nearly two decades of expertise in neuropsychopharmacology and translational medicine, Dr. Lijffijt bridges the gap between academic research and commercial drug development.

Dr. Lijffijt earned his Ph.D. in Neuropsychopharmacology from Utrecht University and completed his MBA at the University of North Alabama. Prior to joining IonTX, Dr. Lijffijt pursued an academic career within Psychiatry and Behavioral Sciences at the University of Texas (UT) Medical Branch, UT Health at Houston, Baylor College of Medicine, and the VA Hospital at Houston. His research career spans multiple domains, including serving as investigator on phase I and phase II clinical trials in mental health, serving as investigator on studies to assess brain mechanisms related to Gulf War Illness, misophonia, bipolar disorder, major depressive disorder, post-traumatic stress disorder, antisocial personality disorder, attention deficit hyperactivity disorder, and addictions, developing EEG biomarkers for treatment-resistant depression, and examining NMDA receptor mechanisms in psychiatric disorders. His work has been published in leading journals including Neuropsychopharmacology.

At IonTX, Dr. Lijffijt has established operational infrastructure, driven fundraising efforts, and secured competitive NIH grants to advance the company's pipeline of novel voltage-gated ion channel modulators that will change dysregulated cell firing in people living with cocaine use disorder or burn-associated chronic pain and its. He has also written grants to forward IonTX’s assets for treat early Alzheimer’s disease, chronic pain, or anhedonia. His unique combination of scientific depth, translational medicine expertise, and executive leadership positions IonTX at the forefront of precision medicine innovation in neuropsychiatry.

Helen Poldsam, PhD
Computational Biologist
University of Texas Southwestern
PhD Candidate
Tallinn University of Technology
Anatomy, Projectome and Functional Annotation of Mouse Spinal Cord Neural Diversity

Helen Poldsam is a computational biologist at the UT Southwestern Medical Center, and a PhD candidate in Biomedicine at Tallinn University of Technology. Her research focuses on single-cell and spatial transcriptomic approaches to map cell type diversity in the spinal cord, with applications to understanding pain processing circuits performed as part of her PhD work in the Pool Lab at UTSW. Helen holds a Bachelor's and Master's degree from Harvard University.

Karen LaShea "Shea" Robinson Posey, PhD
Associate Professor of Pediatrics
University of Texas Health Science Center Houston
Pain-Associated Nociceptor Hyperexcitability in a Mouse Model of Pseudoachondroplasia (mPSACH) Depends Upon Activity of Nav1.8 Channels

Dr. Karen LaShea "Shea" Robinson Posey is an Associate Professor of Pediatrics at McGovern Medical School. She earned her PhD in Biochemistry from the University of Houston, and her post-doctoral work at TAMU Center of Biotechnology focused on understanding the molecular interactions between DNA and homing endonucleases, making functional predictions based on crystallography. Dr. Posey's career in pseudoachondroplasia/dwarfism and cartilage biology was shaped by her mentorship with Dr. Jacqueline T. Hecht. Her research aims to define mechanisms contributing to cartilage-related conditions, including pseudoachondroplasia and osteoarthritis, with the goal of developing mechanism-driven therapeutic approaches to improve cartilage and joint health. This work seeks to enhance the quality of life for individuals with dwarfing conditions and osteoarthritis. Dr. Posey studies pseudoachondroplasia (PSACH), a severe dwarfing condition associated with early-onset osteoarthritis and lifelong joint pain, often reported as soon as a child can speak. Using an in-house developed PSACH mouse model, her team has identified mechanisms underlying chondrocyte dysfunction and loss, revealing a self-perpetuating pathological loop involving ER stress, inflammation, and oxidative stress, which impedes autophagy clearance of the ER. Understanding this pathological loop is particularly exciting, as it may be involved in other ER stress-related conditions, such as neurodegenerative disorders, diabetes, and osteoarthritis. This research has laid the groundwork for potential therapies for pseudoachondroplasia. Notably, the PSACH mouse model shows early signs of pain that worsen with age and joint degeneration. Dr. Posey's recent collaboration with Dr. Terry Walters, an experienced pain researcher at McGovern Medical School UT Health, offers opportunities to explore the lifelong pain mechanisms in PSACH, paving the way for non-surgical therapeutics for PSACH joint pain.

Ridma Sirimanna

PhD Student
Molecular and Cell Biology
University of Texas, Dallas

Spatial Mapping of Nociceptive Neuronal Subtypes in the Mouse Bladder

Ridma Sirimanna is a PhD student in Molecular and Cell Biology at the University of Texas at Dallas. Studying under her PI, Dr. Xintong Dong, her work focuses on profiling nociceptive neuronal subtypes in the mouse bladder and their role in neuroimmune interactions. Ridma is passionate about translational neurobiology and aims to develop targeted therapeutic strategies for visceral pain management.

Swathi Suresh, PhD
Postdoctoral Research Associate
Neuroscience
Tarleton State University
Frontal Alpha Activity and Its Dynamic Modulation During Cognitive Pain Processing in Chronic Pain

Dr. Swathi Suresh is a Postdoctoral Research Associate in the Neuroscience Department at Tarleton State University. She earned an MS in Cancer Immunology from the University of Nottingham and subsequently completed an MS and PhD in Neuroscience at Central Michigan University, where her work focused on chronic neuroinflammation, glymphatic dysfunction, and cellular senescence in transgenic mouse models. She then pursued a postdoctoral fellowship at the Icahn School of Medicine at Mount Sinai, studying Alzheimer's disease and neurodegenerative mechanisms, followed by clinical research experience at Columbia University involving cognitive assessments and patient-centered study design. At Tarleton, she contributes to an NIH-funded project examining the multidimensional experience of pain through high-density EEG, overseeing all aspects of the study, and research workflow development. Her broader scientific interests bridge molecular and systems neuroscience, with a particular focus on how inflammation, aging, and neurodegenerative processes shape cognition, pain perception, and brain–body interaction.

Cristal Villalba, PhD
Postdoc
University of Texas Health Science Center, San Antonio
Evolutionary Architecture of Tongue Cell Types Revealed by Cross-Species Single-Cell Transcriptomics: Implications for Oral Sensation and Pain

Dr. Cristal Villalba is a computational biologist specializing in single-cell and spatial transcriptomics of sensory systems and pain. She develops robust, shareable analytics pipelines and partners with clinicians and engineers to translate discovery into impact, while leading practical training in reproducible data science.

Consuelo (Chelo) Walss-Bass, PhD

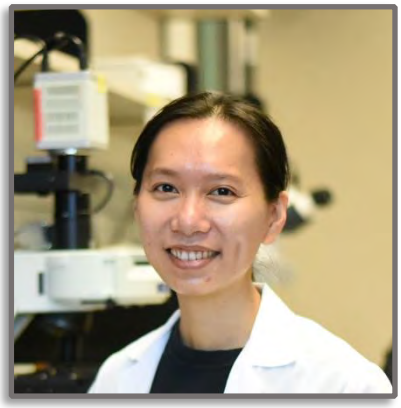
Professor and John S. Dunn Foundation Distinguished Chair,
Louis A. Faillace, MD, Department of Psychiatry and Behavioral Sciences University of Texas Health Science Center at Houston

Pain and Persistent Opioid use Signatures After Trauma

Dr. Walss-Bass received a Bachelor of Science degree in chemical engineering from the Instituto Tecnologico, Mexico, a Master's of Science degree in chemistry from the University of Texas at San Antonio and a Doctor of Philosophy degree in biochemistry from the University of Texas Health Science Center at San Antonio. She trained in psychiatric genetics as a postdoctoral fellow at the University of Texas Health Science Center at San Antonio. She is currently Professor with tenure and the Director of the Psychiatric Genetics Program and the UTHealth Houston Brain Collection in the Department of Psychiatry and Behavioral Sciences at the University of Texas Health Science Center in Houston.

The central focus of the Walss-Bass laboratory is to identify the biological causes and risk factors that lead to severe mental health disorders such as schizophrenia, bipolar disorder, and substance use disorders. Despite tremendous advances in genetics, molecular/cellular biology, and psychopharmacology, the fundamental biological nature of psychiatric disorders remains largely elusive. To address this problem, her laboratory utilizes a model of collaboration and dialogue between investigators working in the laboratory and investigators working directly with patients, to correlate behavioral and physiological outcomes with genetic underpinnings and biological mechanisms.

The Walss-Bass' talk will focus on a longitudinal study of patients who were discharged with an opioid prescription after a traumatic injury, and the identification of biomarkers of risk for chronic pain and persistent opioid use.



Jun Wang, PhD
Instructor
Baylor College of Medicine
Deciphering the Molecular Mechanisms of Joint Pain in Equine Osteoarthritis via snRNA-seq

Dr. Wang is currently an Instructor in Dr. Brendan Lee's laboratory at Baylor College of Medicine. She earned her bachelor's degree in Biotechnology, with a double major in Computer Science, from Zhejiang University in China. She completed her Ph.D. in Molecular Evolution at the University of Chicago, followed by postdoctoral training at Wayne State University and Baylor College of Medicine. Dr. Wang has published 37 peer-reviewed papers, including 16 as first author, in the fields of genomics, genetics, and bioinformatics. Her research in the Lee laboratory focuses on elucidating the molecular mechanisms underlying osteoarthritis.

Tingting Yang, PhD
Bioinformatics Programmer
Molecular & Human Genetics
Baylor College of Medicine
Spatial Atlas of the Mouse Trigeminal Ganglion

Tingting Yang is a bioinformatics programmer in the Department of Molecular & Human Genetics in Baylor College of Medicine. She received her bachelor's degree in biotechnology and Ph.D. in biophysics from Fudan University. She also earned her master's degree in Statistics from Rice University. She is working on the data analysis of single cell genomics and spatial genomics.

Subo Yuan, MD, PhD
Assistant Professor
Neurobiology
University of Texas Medical Branch, Gavleston
Spatial Atlas of the Mouse Trigeminal Ganglion

Dr. Yuan's lab is a pain research lab with ongoing research in the following areas:

Our lab investigates the pathogenesis of neuropathy caused by diseases and neuronal injury. The mechanism of neuropathy intermingled with nociceptors, immunocytes and keratinocyte cells and the interaction outcomes is the focus of our neuropathy work. My current R01 grant focuses on the pathogenesis of HIV-1 gp120, an envelope viral protein and anti-retroviral therapy induced neuropathy and chronic pain. In addition, we also work on diabetic associated neuropathy.

Another ongoing project is to investigate the pathogenesis of small extracellular vesicles (sEV) in HIV associated chronic pain. sEV are tiny lipid bound sacs released by various type cells containing biological active cargos that act for intercellular communication by remotely or locally releasing cargo to recipient cells, influencing the recipient cells physiological and pathological processes. Our goal is to elucidate the nociceptive role of sEV in inducing and maintaining chronification of different types of pain.

We welcome the next generation of scientists to join us and work together to investigate the mechanisms of chronic pain.

Lightning poster talk presenters
in order of presentation

DRG Meningeal Tertiary Lymphoid Structures Function as a Pro-Nociceptive Niche through B cell–Dependent Germinal Center Formation and Plasma Cell Differentiation After Peripheral Nerve Injury
Tusar Acharya, MD Anderson Cancer Center
Poster #19

Enhancing Sensory Motor Swallow Brain Networks Through Individualized Neuromodulation – A Feasibility Intervention for Oropharyngeal Cancer Pain Following Treatment
Michael Davis, Baylor College of Medicine
Poster #3

Inflammatory Neuropathy in Mouse Models of Colorectal Cancer: Implications for Chemotherapy-Induced Peripheral Neuropathy
Caitlyn Gaffney, MD Anderson Cancer Center
Poster #6

Life-long Hydroxyurea Treatment Decreases Chronic Pain in Sickle Cell Disease Mice by Limiting TRPA1 Sensitization in Peripheral Nociceptors
Nya Gayluak, UT at Dallas
Poster #8

Influence of Sex and Estrous Stage on Sensory and Affective Responding of Pain
Kaitlyn Little, University of Houston
Poster #10

Sex-Divergent Transcriptomic Regulation of Sensory Neurons in Oral Cancer
Jaclyn Merlo, UT San Antonio
Poster #11

Understanding the mechanism of Painful Radiation Induced Oral Mucositis (RIOM)
Priya Saraswathi Narayanan, UT at San Antonio
Poster #13

Poster presenters
in alphabetical order

Data blitz presenters
are in gray.

Presenter First Name	Presenter Last Name	Presenter Institution	Title of Submission	Poster number
Tusar	Acharya	MD Anderson Cancer Center	DRG Meningeal Tertiary Lymphoid Structures Function as a Pro-Nociceptive Niche through B cell–Dependent Germinal Center Formation and Plasma Cell Differentiation After Peripheral Nerve Injury	19
Isabella	Bourtin	Rice University	Insomnia and Pain Following Traumatic Injury: Associations with Resting State EEG	1
Michael	Davis	Baylor College of Medicine	Enhancing Sensory Motor Swallow Brain Networks Through Individualized Neuromodulation – A Feasibility Intervention for Oropharyngeal Cancer Pain Following Treatment	3
Sandeep	Dembla	MD Anderson Cancer Center	Role of Angiotensin II in Driving Myeloid Dysfunction and Neuropathy in Type 2 Diabetes	4
Vivek	Dubey	UT Southwestern Medical Center, Dallas	Therapeutic Targeting of the Spinal Cord Output Cell-Type Map of Pain States	31
Hajira	Elahi	MD Anderson Cancer Center	Human iPSC-Derived Sensory Neurons Reveal Pain Mechanisms in Fibrodysplasia Ossificans Progressiva	5
Nathan	Fiore	MD Anderson Cancer Center	Sialylated Immunoglobulin G Relieves Established Mechanical Allodynia After Peripheral Nerve Injury	20
Caitlyn	Gaffney	MD Anderson Cancer Center	Inflammatory Neuropathy in Mouse Models of Colorectal Cancer: Implications for Chemotherapy-Induced Peripheral Neuropathy	6
Chrystine	Gallegos	UT Health Houston	Satellite Glial Cell Activation by TGFβ Increases Excitability of Isolated Rat and Human Dorsal Root Ganglion Neurons	7
Nya	Gayluak	UT at Dallas	Life-long Hydroxyurea Treatment Decreases Chronic Pain in Sickle Cell Disease Mice by Limiting TRPA1 Sensitization in Peripheral Nociceptors	8
Audrey	Green	UT Dallas	Nociceptor-Restricted Cannabinoid Receptor 1 Induces Antinociception in a Preclinical Model of Persistent Painful Diabetic Peripheral Neuropathy	21
Dustin	Green	UTMB	Mast cell specific GPCR regulates UTI immune response and pain	22
Kathy	Guerra	UT Health Houston	A Promising Target for Chronic Pain After Spinal Cord Injury: Investigations of Macrophage Migration Inhibitory Factor (MIF)	9

Poster presenters
in alphabetical order

Data blitz presenters
are in gray.

Presenter First Name	Presenter Last Name	Presenter Institution	Title of Submission	Poster number
Mario	Heles	MD Anderson Cancer Center	Nav1.8 inhibition by Suzetrigine reduces spontaneous activity in human sensory neurons	23
Khalil	Kasm	MD Anderson Cancer Center	Sensory neuron activity promotes tumor progression in malignant peripheral nerve sheath tumor (MPNST) via TRPV1^{+} nociceptor signaling	32
Marijn	Lijffijt	IonTX Inc.	Targeting protein-protein interactions (PPI) between Nav1.7 and FGF13 as a first-in-class mechanism for safe and selective analgesia	24
Kaitlyn	Little	University of Houston	Influence of Sex and Estrous Stage on Sensory and Affective Responding of Pain	10
Jaclyn	Merlo	UT San Antonio	Sex-Divergent Transcriptomic Regulation of Sensory Neurons in Oral Cancer	11
Sreya	Mitra	University of Houston	Mapping Corneal Nociceptive Innervation	12
Priya Saraswathi	Narayanan	UT at San Antonio	Understanding the mechanism of Painful Radiation Induced Oral Mucositis (RIOM)	13
Helen	Poldsam	UT Southwestern Medical Center	Anatomy, Projectome and Functional Annotation of Mouse Spinal Cord Neural Diversity	25
Abhira	Ravirala	UT Southwestern Medical Center	Elucidating the spinal output circuit mechanisms for cancer pain	14
Anna	Ronck	Tarleton State University	Theta (4–7 Hz) Band EEG Activity Associated with Cognitive Processing of Sensory and Affective Pain Descriptor Words	15
Ridma	Sirimanna	UT Dallas	Spatial Mapping of Nociceptive Neuronal Subtypes in the Mouse Bladder	26
Swathi	Suresh	Tarleton State University	Frontal Alpha Activity And Its Dynamic Modulation During Cognitive Pain Processing In Chronic Pain	27
Samantha	Tilley	Baylor College of Medicine	AAV Viral Tracing of Primary Sensory Neurons Using SELECTIV Mice	16
Janet	Van Cleave	UT Health Houston	Pain is Associated with Decreased Emotional and Physical Function In Head and Neck Cancer Patients Residing in Rural and Urban Settings	17
Gabrielle	Williams	UT Southwestern Medical Center	Investigating the Functional Circuit Assembly of the Anterolateral Pathway	18
Tingting	Yang	Baylor College of Medicine	Spatial Atlas of the Mouse Trigeminal Ganglion	28
Subo	Yuan	UT Medical Branch	An unknown endogenous algogens, gp120 derived extracellular vesicle (sEV) induced HIV-1 associated pain	29

Poster abstracts in alphabetical order

Poster 19

DRG Meningeal Tertiary Lymphoid Structures Function as a Pro-Nociceptive Niche through B cell–Dependent Germinal Center Formation and Plasma Cell Differentiation After Peripheral Nerve Injury

Tusar K. Acharya[1], Vipul Pandey[1], Kendal F. Willcox[1], Gabriel Lucena-Silva[2], Michael Lacagnina[1], Pooja Singhmar[1], Younus A. Zuberi[1], David Ruiz[1], Thiago Cunha[2], Cobi J. Heijnen[3], Peter M. Grace*[1]

1. Laboratories of Neuroimmunology, Department of Symptom Research, The University of Texas MD Anderson Cancer Center, Houston, Texas 77030, USA

2. Center for Research in Inflammatory Diseases (CRID), Ribeirão Preto Medical School, University of Sao Paulo, Ribeirão Preto, Sao Paulo, 14040-900, Brazil.

3. Department of Psychological Sciences, Rice University, Houston, Texas, 77005, USA

*Corresponding author, Email: pgrace@mdanderson.org

Abstract

Neuroimmune interactions at the dorsal root ganglia (DRG) contribute to neuropathic pain following peripheral nerve injury (PNI). Recent work has implicated adaptive humoral immunity among these mechanisms, demonstrating that IgG production by B cells gates hyperactivity of DRG neurons through IgG-Fc gamma receptor signaling. Here, we demonstrate that after PNI, leukocyte clusters are organized in the DRG meninges, enveloped by lymphatic endothelium and apposed to high endothelial venules. These clusters are consistent with tertiary lymphoid structures (TLSs), are dominated by B cells (including proliferating B cells), and contain follicular helper T cells, macrophages and dendritic cells. Single cell RNA sequencing analyses revealed enrichment of germinal center B cells in the dorsal root meninges after PNI, which was further confirmed by detecting the expression of germinal center–associated markers within TLSs. As germinal centers are sites of B cell maturation, we further identified the presence of plasma cells within DRG meningeal TLSs, indicating B cell differentiation inside the TLSs. Germinal center B cells regulate TLSs, as these structures were absent following conditional deletion of the *Ezh2* histone methyltransferase from B cells. Intrathecal administration of a CD20 monoclonal antibody to locally deplete B cells at the time of injury also disrupted TLS organization. Conversely, intrathecal transfer of B cells to muMt mice (B cell-deficient) was sufficient for TLSs to organize after PNI. Allodynia did not develop when TLS organization was experimentally disordered after PNI (*Ezh2* deletion, intrathecal CD20 mAb treatment, or muMt genotype). These data reveal a pivotal role for germinal center B cells in TLS organization, and a pathological link between DRG meningeal TLSs and neuropathic pain.

Poster 1

Insomnia and Pain Following Traumatic Injury: Associations with Resting State EEG

Isabella Bourtin[1], Heather E. Webber[2], Joy M. Schmitz[2], Darrion Mouton[2], Kendall Farrell[2], Bruno Kluwe-Schiavon[2], Consuelo Walss-Bass[2]

1. Rice University
2. Faillace Department of Psychiatry and Behavioral Sciences, University of Texas Health Science Center at Houston

Corresponding author: Consuelo Walss-Bass, Faillace Department of Psychiatry and Behavioral Sciences, University of Texas Health Science Center at Houston, 1941 East Rd, BBSB suite 3110, Houston, TX, E-mail: consuelo.walssbass@uth.tmc.edu

Pain following traumatic injury is a major concern, due to its interference with everyday functioning, such as sleep. Prior work has implicated waking spectral EEG as a biomarker associated with both pain and sleep disturbance. Here we assessed the spectral features of pain and insomnia specifically following traumatic injury hospitalization. Thirty-four participants who were hospitalized for traumatic injury (n=34) completed self-report assessments of pain and insomnia and resting-state EEG at a 7-day follow-up. Alpha (8-13Hz) and theta (4-7Hz) power during 3-minutes of eyes closed were calculated using the Fast-Fourier transformation. Associations between EEG variables and pain and sleep measures, including the ISI (Insomnia Severity Index), PCS (Pain Catastrophizing Scale), PVAQ (Pain Vigilance and Awareness Questionnaire), and BPI (Brief Pain Inventory), were analyzed via bivariate correlations. Elevated alpha at the central electrodes and widespread theta were significantly associated with higher ISI ($r=.37$, $p<.05$; $r=.39$, $p<.05$). Greater alpha was associated with greater pain interference, primarily at central electrodes ($r=.39$, $p<.05$). Theta power was associated with higher PCS scores, strongest at central electrodes ($r=.45$, $p<.05$). No consistent associations were observed between EEG and pain intensity or PVAQ. These findings support the potential of alpha and theta as biomarkers of risk for persistent pain and insomnia following trauma, and that region-specific patterns suggest different neural mechanisms underlie various dimensions of pain-related dysfunction.

Acknowledgements: This research was funded by a Wellcome Leap Grant. We would also like to thank the patients for their willingness to participate in this study.

Poster 3

Enhancing Sensory Motor Swallow Brain Networks Through Individualized Neuromodulation – A Feasibility Intervention for Oropharyngeal Cancer Pain Following Treatment.

Michael Davis[1], Brian Bishara[1], Sandy Reddy[1], Rohan Vemu[1], Cameron Noorkhbash[1], Anthony Allam[1,2], Alastair Thompson[1], Emmanouel Froudarakis[3,5], T. Dorina Papageorgiou[2,3,4,6,7,8]

1. School of Medicine, Baylor College of Medicine
2. Department of Psychiatry, Baylor College of Medicine
3. Department of Neuroscience, Baylor College of Medicine
4. Department of Physical Medicine & Rehabilitation, Baylor College of Medicine
5. Institute of Molecular Biology and Biotechnology, FORTH, Crete, Greece
6. Department of Electrical and Computer Engineering, Rice University
7. Center for Space Medicine, Baylor College of Medicine
8. Department of Neuroengineering, Rice University

Corresponding author: T. Dorina Papageorgiou, Department of Neuroscience, Baylor College of Medicine, 1 Baylor Plz, Houston, Texas, E-mail: t.dorina.papageorgiou@bcm.edu

About 70 percent of head and neck cancer cases, the sixth most common type of cancer, involve oropharyngeal cancer (OPC). Surgery or radiation therapy, the primary treatment for OPC, can cause neuropathy of the glossopharyngeal and hypoglossal nerves, which results to chronic cranial neuropathy. This leads to swallowing difficulties, voice changes, loss of taste, tongue fibrosis, all of which can significantly reduce quality of life and can even be fatal because of aspiration. The Papageorgiou lab has developed an individualized neuromodulation (iNM) intervention that can serve as treatment for chronic cranial neuropathy (U.S. Patent Application No. 16/954,256). This study shows how iNM can strengthen and increase oxygenated hemoglobin (HbO2) in brain motor and sensory networks that regulate swallowing – a physiologic response that is characterized by motor and sensory control. With no current evidence-based standard of care, iNM may offer a valuable and much needed intervention to neurorehabilitate dysphagia in OPC patients. Here, we test the feasibility of iNM in healthy participants. Healthy subjects (n=30) participated in a two-day iNM study. On Day one, we delineated the brain spatial networks and amplitude generated by swallow, interleaved with periods of tongue-rest(baseline) and tongue-movement. Our innovation is based on targeting individualized networks by extracting HbO2 magnitude and spatial extents. Each participant's TMSC cortical selectivity was extracted and targeted for iNM. On Day two, participants underwent iNM and control-No-iNM conditions. Support vector machine (SVM) classified cortical swallowing patterns versus tongue-at-rest generated via iNM and control conditions. We quantified the HbO2 magnitude for each network's areas separately, by computing dynamic causal modeling to elucidate the association between brain states and the physiological responses of TMSC. iNM increased performance compared to the control condition, as evidenced through classification accuracies. iNM strengthened motor, sensory, and attention networks by increasing the magnitude, spatial extent, and decreasing the variance of HbO2 in the regions that regulate swallowing. Spatiotemporal quantification of the HbO2 signal can help standardize iNM protocols with a goal to neurorehabilitate head and neck cancer patients.

Acknowledgements: This study is supported by grants to T.D. Papageorgiou from the McNair Medical Institute, the Robert and Janice McNair Foundation, TIRR Mission Connect, Baylor College of Medicine Junior Faculty Award, BCM - NRI and Houston Methodist Collaborative grant, and NIH – T32

Poster 4

Role of Angiotensin II in Driving Myeloid Dysfunction and Neuropathy in Type 2 Diabetes

Sandeep Dembla[1], Andrew J Shepherd[1#]

1. Department of Symptom Research, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA

[#] Corresponding author: Andrew J Shepherd, Department of Symptom Research, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
E-mail: AJShepherd@mdanderson.org

Abstract

Diabetic peripheral neuropathy (DPN) is a major complication of type 2 diabetes (T2D) and is closely linked to chronic inflammation and dysregulated myeloid cell activity. Elevated angiotensin II (Ang II), a pro-inflammatory renin - angiotensin peptide, is increasingly implicated in pathological myelopoiesis, whereas its counter-regulatory peptide Ang-(1–7) is anti-inflammatory. We hypothesize that T2D increases Ang II levels, disrupting myeloid development and contributing to neuropathy.

Using high-fat diet (HFD) and $Lepr^{db/db}$ mouse models, we examined Ang II driven myeloid abnormalities using flow cytometry, colony-forming assays, and peptide quantification. ELISA revealed significantly elevated Ang II in diabetic mice. Flow cytometry of bone marrow, spleen, and blood showed increased polymorphonuclear and monocytic immature myeloid cells, along with expansion of the c-$Kit^{+}Lin^{-}Sca\text{-}1^{+}$ (KLS) hematopoietic stem/progenitor population, indicating broad disruption of early myeloid development. CFU assays demonstrated enhanced output of myeloid progenitors, particularly CFU-GM colonies, consistent with amplified and dysregulated myelopoiesis in T2D.

Calcium imaging in dorsal root ganglion (DRG) neurons from diabetic mice revealed selective alterations in TRPA1 activity, while TRPV1 and TRPM3 responses remained largely unchanged. This selective TRPA1 sensitization aligns with reports showing that oxidative stress and inflammatory mediators preferentially potentiate TRPA1 signaling, suggesting a mechanistic link between myeloid dysfunction, ROS, and nociceptive hypersensitivity.

Together, these findings indicate that elevated Ang II in T2D drives dysfunctional myelopoiesis and may promote neuropathic pain through altered immune–neuronal signaling. Ongoing work aims to define hormonally mediated shifts in myeloid cell function and the lasting epigenetic changes that may underlie chronic pain in T2D.

Funding grant: R01DK132019

Poster 31

Therapeutic Targeting of the Spinal Cord Output Cell-Type Map of Pain States

Vivek Dubey[1], Satoshi Ishishita[1], Allan-Hermann Pool[1, 2]

1. Department of Neuroscience, O'Donnell Brain Institute, University of Texas Southwestern Medical Center
2. Department of Anesthesia and Pain Management, University of Texas Southwestern Medical Center

Chronic pain including lower back, musculoskeletal, and cancer associated pain often requires lifelong treatment with abuse-prone analgesics, leaving approximately 8.5% of U.S. adults with persistent high-impact pain. There is an urgent need for durable, non-addictive therapeutic strategies. Spinal projection neurons (SPNs), which transmit nociceptive signals from the spinal cord to the brain, represent a promising but underexplored target for precision pain management. However, the specific SPN subclasses that mediate distinct pain modalities, and their therapeutic targeting remain poorly defined. In preliminary work, we have identified 12 transcriptionally distinct SPN subclasses, each defined by unique cell-surface receptors that are selectively enriched in specific SPN populations and absent or under-represented in the >100 other spinal cord neuron types. Here, we tested the feasibility of SPN level pain interventions by targeted lesioning of five of the 12 spinal projection neurons with Tacr3 targeting ligand-drug conjugate Neurokinin-B-Saporin using intraspinal injections. Targeted lesioning of Tacr3+ neurons at the L3–L4 level demonstrated profound attenuation of heat and mechanical evoked pain behaviors as well as eliminating mechanical hypersensitivity in a chronic neuropathic pain state while leaving locomotion intact. Furthermore, we characterize new targets for targeted lesioning of pain mediating SPN classes for a combinatorial and once-and-done strategy for chronic pain management. Together, these findings demonstrate the feasibility of SPN subclass-specific immunotoxin strategies for precise and durable pain relief and advance non-addictive precision therapeutics for chronic pain.

Poster 5

Human iPSC-Derived Sensory Neurons Reveal Pain Mechanisms in *Fibrodysplasia Ossificans Progressiva*

Hajira Elahi[1], Asta Arendt-Tranholm[2], Theodore J. Price[2], Patrick M. Dougherty[1]

1. University of Texas MD Anderson Cancer Center, Department of Pain Medicine-Research
2. University of Texas at Dallas, School of Behavioral and Brain Sciences

Corresponding author: Patrick M. Dougherty, University of Texas MD Anderson, 1515 Holcombe Blvd, Houston, TX, Email: pdougherty@mdanderson.org

Fibrodysplasia Ossificans Progressiva (FOP) is a rare and devastating genetic disorder characterized by progressive heterotopic ossification caused by a gain-of-function mutation in the *ACVR1* gene. Alongside musculoskeletal anomalies, individuals with FOP often endure another persistent and debilitating challenge: excruciating pain. The rarity of FOP, coupled with substantial risks associated with invasive procedures to obtain patient specimens, has limited the ability to comprehensively study the mechanisms underlying pain in this disorder. To address this gap, we generated induced pluripotent stem cells (iPSCs) from four unique FOP patients and differentiated them into sensory neurons (iSNs) with functional characteristics closely resembling those of human dorsal root ganglion neurons. These iSNs serve as a valuable and robust model for investigating the effects of *ACVR1* mutations on sensory neuron function. Using whole-cell patch clamp recordings, we examined the physiological consequences of the *ACVR1* mutation on baseline excitability and stimulus-evoked responses in patient-derived iSNs and compared these to wild type iSNs. This approach not only addresses a critical limitation in FOP research by providing non-invasive, scalable model systems to study FOP-pain, but also establishes a foundation for developing targeted therapies for pain management. These findings significantly enhance our understanding of the interplay between genetic mutations and sensory neuron dysfunction, underscoring the transformative potential of iPSC-based models in advancing research on rare genetic disorders.

Poster 20

Sialylated Immunoglobulin G Relieves Established Mechanical Allodynia After Peripheral Nerve Injury

Nathan T. Fiore, Kendal F. Willcox, Jonathan T. Lu, Cobi J. Heijnen., Peter M. Grace; University of Texas MD Anderson Cancer Center.

Abstract: B cells contribute to neuropathic pain following chronic constriction injury (CCI) of the sciatic nerve by secreting immunoglobulin G (IgG), which engages Fc gamma receptors (FcγRs) to enhance dorsal root ganglia (DRG) neuron excitability. Emerging studies show that IgG–FcγR interactions depend on sialic acid in the IgG Fc region. Specifically, terminal sialylation imparts anti-inflammatory properties to IgG, such as stimulating macrophage subsets to produce IL-10. In this study, we used *in vitro* glycoengineering and passive transfer of sialylated IgG to determine whether sialylated IgG could promote pain resolution following peripheral nerve injury in mice. IgG was purified from the serum of CCI mice using Protein A (MabSelect SuRe 70) resin-based columns and then sialylated by incubation with β-galactoside α-2,6-sialyltransferase 1 in the presence of CMP-sialic acid overnight. Sialylated IgG (n=12) or control IgG (n=12) was passively transferred to recipient CCI mice via intrathecal injections (40μg daily for three consecutive days), starting 14 days post-CCI. Mechanical allodynia was assessed regularly using von Frey filaments. Passive transfer of sialylated IgG relieved established mechanical allodynia for at least 1 week after injections ($F_{1,22}$=123.6 $p<0.001$), compared to mice who received IgG control. Moreover, mice receiving sialylated IgG exhibited a higher ratio of anti-inflammatory macrophages (CD206:F4/80, $t=3.303$ $p<0.05$) and increased *Il10* gene expression ($t=2.402$ $p<0.05$) in the DRG. These data suggest that sialylated IgG engages DRG macrophages to secrete IL-10 and thereby promote pain resolution following nerve injury. Therefore, enhancing IgG sialylation may offer a novel approach to restrain inflammation and treat neuropathic pain.

Poster 6

Inflammatory Neuropathy in Mouse Models of Colorectal Cancer: Implications for Chemotherapy-Induced Peripheral Neuropathy

Caitlyn M. Gaffney[1], Angela M. Casaril[1], Iqbal Mahmud[2], Bo Wei[2], Karen M. Valadez[1], Elizabeth A. Kolb[1], Fisher R. Cherry[1], Theresa A. Guise[3], Philip L. Lorenzi[2], Lei Shi[3], Carolyn L. Hodo[4], Andrew J. Shepherd[1*]

1. Laboratories of Neuroimmunology, Department of Symptom Research, Division of Internal Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
2. Department of Bioinformatics and Computational Biology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
3. Department of Endocrine Neoplasia and Hormonal Disorders, Division of Internal Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA
4. Michale E. Keeling Center for Comparative Medicine and Research, The University of Texas MD Anderson Cancer Center, Bastrop, TX 78602, USA.

Corresponding author: Andrew Shepherd, Laboratories of Neuroimmunology, Department of Symptom Research, Division of Internal Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA., E-mail: ajshepherd@mdanderson.org

Introduction

Colorectal cancer (CRC) is the third most common cancer in the United States; each year about 150,000 individuals are diagnosed. Advances in screening and treatment regimens, including oxaliplatin-based regimens, such as 5-fluorouracil, leucovorin & oxaliplatin ('FOLFOX') have led to an increase of long-term CRC survivorship. However, FOLFOX and similar regimens cause prolonged neurological symptoms in up to 80% of patients. Current studies of chemotherapy-induced peripheral neuropathy (CIPN) are done in young-adult, cancer-free, male rodents that are naïve to surgery. However, this does not model adjuvant chemotherapy seen clinically. Here, we developed a combined model of orthotopic colorectal cancer and adjuvant oxaliplatin, to more accurately replicate clinical CIPN.

Methods

Cecal injections using the MC38 colon adenocarcinoma cell line were induced in 8-week and 52-week-old C57BL/6 mice. Three weeks following injection, mice underwent cecum removal surgery (cecectomy). The mice were then allowed to recover for 10 days before FOLFOX treatment began. Tumor growth was monitored for 8 weeks by bioluminescent imaging before, during, and after cecectomy and FOLFOX treatment. Behavioral tests were conducted throughout this process to characterize stimulus-evoked pain sensitivity (von Frey and coldgreaves), ongoing/spontaneous pain sensitivity (CPP and burrowing), motor coordination (beam walk), and gait (Catwalk).

Results

We have previously shown neuronal dysfunction directly induced by tumor growth alone. Mice with CRC develop peripheral neuropathy associated with subtle locomotor deficits, without overt hypersensitivity. Our findings suggest CRC can be causally linked to a subacute form of chronic inflammatory demyelinating polyneuropathy across species, which may represent an under-reported, yet important risk factor for neurological dysfunction in CRC survivors. Indeed, matched

dosing of oxaliplatin or a FOLFOX-like regimen caused more persistent hypersensitivity in CRC mice.

Conclusions

Collectively our data suggest that subclinical neuropathy secondary to CRC may represent an under-reported, yet important risk factor for persistent neurological dysfunction in CRC survivors. Further studies are needed to elucidate the mechanisms involved in CIPN induced by FOLFOX treatment in aged mice.

Poster 7

Satellite Glial Cell Activation by TGFβ Increases Excitability of Isolated Rat and Human Dorsal Root Ganglion Neurons

Chrystine Gallegos[1], Alexis Bavencoffe[1], Kayla Johnson[1], Yong Li[1], Jazmin Grant[1], Sarah Ravdel[1], Patrick M. Dougherty[2], Oshri Avraham[3], Edgar T. Walters[1], Anibal Garza-Carbajal[1], and Carmen Dessauer[1#]

1. Department of Integrative Biology and Pharmacology, McGovern Medical School at the University of Texas Health Science Center, Houston, TX
2. Department of Pain Medicine Research, The University of Texas MD Anderson Cancer Center, Houston, TX
3. Department of Cellular Biology, University of Georgia, Athens, GA

[#]Corresponding author: Carmen W. Dessauer, Department of Integrative Biology and Pharmacology, McGovern Medical School at UTHealth Houston, Email: carmen.w.dessauer@uth.tmc.edu

Satellite glial cells (SGCs), which tightly enwrap all types of dorsal root ganglion (DRG) neurons, are uniquely positioned to modulate sensory neuron activity and are increasingly recognized as key contributors to chronic pain. In neuropathic pain states, SGCs exhibit altered transcriptional profiles and increased expression of glial fibrillary acidic protein (GFAP), a hallmark of SGC activation. However, the potential circulating factors that drive SGC activation and their regulatory influence on DRG neuron excitability are largely unknown. To address these gaps, we employed a pure culture of isolated SGCs that mirrors the signaling pathways and transcriptional landscape of SGCs within DRGs. Using this model, we screened circulating factors known to be elevated in rodent pain models and in patient chronic pain conditions for their stimulation of multiple signaling pathways in SGCs. One of these factors, transforming growth factor beta (TGFβ), induces GFAP expression in pure SGCs and significantly alters SGC gene expression. Furthermore, TGFβ-induced differentially expressed genes overlap with those revealed by single-cell RNA sequencing of SGCs in rodent models of neuropathic pain. Moreover, TGFβ activation of pure SGCs, but not TGFβ or SGCs alone, promotes DRG neuron hyperexcitability and depolarizing spontaneous fluctuations—probable drivers of chronic pain. Importantly, TGFβ treatment of *human* DRG cultures containing numerous SGCs also promotes neuronal hyperexcitability. Thus, TGFβ can drive SGC activation, which in turn increases DRG neuronal excitability, indicating the therapeutic potential of targeting SGCs for treatment of chronic pain.

Acknowledgements: This research was funded by National Institute of Neurological Diseases and Stroke, RO1 NS091759, National Institute of General Medicine T32 GM139801, Marilyn and Frederick R. Lummis, Jr., M.D. Fellowship in Biomedical Sciences, Dr. John J. Kopchick Fellowship, and the John P. and Kathrine G. McGovern Foundation.

Poster 8

Life-long Hydroxyurea Treatment Decreases Chronic Pain in Sickle Cell Disease Mice by Limiting TRPA1 Sensitization in Peripheral Nociceptors

Nyabouk J Gayluak[1], Sourav S Roy[1], Ashley N Plumb[1], Hemanth Mydugolam[1], Zulmary Manjarres[1], Lindsey E Ramos Freitas[1], Amanda M Brandow[2], Michael D Burton[1], Katelyn E Sadler[1]

1. Department of Neuroscience, School of Behavioral and Brain Sciences, and Center for Advanced Pain Studies, The University of Texas at Dallas, Richardson, Texas
2. Department of Pediatrics, Division of Hematology/Oncology/Bone Marrow Transplantation, Medical College of Wisconsin; Milwaukee, WI, USA

Corresponding author: Katelyn Sadler, Department of Neuroscience, Center for Advanced Pain Studies, University of Texas at Dallas, 800 W Campbell Rd, Richardson, TX 75080, Email: katelyn.sadler@utdallas.edu

Pain is the primary reason individuals with sickle cell disease (SCD) seek medical care. Although more than 50% of individuals with SCD develop chronic pain as they age, it is unclear if and how standard-of-care SCD therapies influence pain trajectories. Hydroxyurea (HU) is a prescribed disease-modifying therapy that increases fetal hemoglobin and reduces red blood cell sickling. Although HU is administered as early as nine months of age, its impact on the development of lifelong chronic SCD pain has not been systematically evaluated. In this study, we used the Townes SCD mouse model to assess the effects of lifelong HU treatment on chronic SCD-associated pain and associated mechanisms of resulting phenotypes. We found that lifelong HU treatment reduces chronic SCD mechanical hypersensitivity. This decrease in mechanical allodynia was mirrored by a decreased abundance of circulating monocytes. In support of the idea that vehicle-treated SCD mice exist in a pro-inflammatory state, a higher proportion of sensory neurons isolated from vehicle-treated SCD mice responded to lipopolysaccharide (LPS), a component of Gram-negative bacterial cell membranes, as compared to neurons isolated from HU-treated SCD mice. We determined that this LPS sensitization depends on neuronal TRPA1 expression as inhibition of TRPA1 reduced both LPS-induced calcium flux in neurons and chronic mechanical hypersensitivity in SCD mice. These findings provide additional support for HU use in individuals with SCD and show a potential mechanistic link between innate immune signaling and chronic pain. Our results contribute to ongoing efforts to identify preventative strategies for chronic pain in SCD.

Acknowledgements: This study was supported by the NIH R00HL155791 to KES and the Rita Allen Foundation Award to KES.

Poster 21

Nociceptor-Restricted Cannabinoid Receptor 1 Induces Antinociception in a Preclinical Model of Persistent Painful Diabetic Peripheral Neuropathy

Audrey Green[1] and Michael Burton[1]

1. Department of Neuroscience, University of Texas at Dallas

Corresponding author: Michael Burton, Department of Neuroscience, University of Texas at Dallas, 800 W. Campbell Road, Richardson, TX, Email: Michael.burton@utdallas.edu

Current treatments of painful diabetic peripheral neuropathy (PDPN) are ineffective for long-term use. The cannabinoid receptor 1 (CB1R) has been studied as an alternative target for pain relief; however, due to debates about the analgesic site of action of CB1R (central or peripheral), psychoactive central effects, and use of incongruent preclinical pain models, preclinical findings fail to translate clinically. We hypothesize strictly engaging CB1R in the peripheral nervous system during neuropathic pain would circumvent unwanted side-effects allowing for long-term use. Furthermore, we posit CB1R agonism produces antinociception during neuropathic pain due to changes in expression of CB1R and molecules within the endocannabinoid system (ECS). Preclinical studies, including our preliminary data, of peripherally-restricted CB1R agonism demonstrate alleviation of neuropathic but not acute pain, highlighting a timing-dependent effect caused by molecular changes within the ECS, which is clinically beneficial as opioids effectively manage acute pain. Altogether, there remains a gap in understanding the cellular circuitry and/or molecular changes within the ECS underlying its antinociceptive properties. To determine the role of peripheral CB1R in alleviating PDPN and identify responsible mechanisms, we used a novel genetic mouse model where CB1R expression is restored to $Na_v1.8^+$ nociceptors ($Na_v1.8^{+/-}$:$CB1R^{TB/TB}$) on a CB1R-null background ($CB1R^{TB/TB}$). Utilizing these animals, we examined the ability of peripheral CB1R to produce antinociception during PDPN. To explore the mechanism behind the timing-dependent effect of CB1R agonism, we assessed for changes in CB1R expression at a persistent point of PDPN. Three genotypes of mice (Wild-Type, $CB1R^{TB/TB}$(whole-body null), and $Na_v1.8^{+/-}$:$CB1R^{TB/TB}$) received 50 mg/kg streptozotocin (STZ) or vehicle intravenously daily across 5 days to induce PDPN. Spontaneous pain, via facial grimacing, mechanical hypersensitivity, via von Frey, and thermal hypersensitivity, via hot and cold plate, were assessed 10, 13, 16, 19, 22, and 25 days following initial STZ injection. On day 25, mice were intraperitoneally administered 0.1 mg/kg CP-55,940, a CB1R agonist, or vehicle and pain-like behaviors were evaluated 20- and 60-minutes post injection. Immediately following day 25 behavior, changes in CB1R gene expression, via qPCR, induced by PDPN was quantified in crucial nervous system areas of the ECS: the lumbar dorsal root ganglia (L4-L6), brain stem, and lumbar spinal cord. We observed the $Na_v1.8^{+/-}$:$CB1R^{TB/TB}$ mice recapitulated the WT behavioral phenotype of antinociception following CB1R agonism, suggesting nociceptor CB1R is sufficient to alleviate PDPN. However, we did not observe PDPN-induced changes in CB1R gene expression in any genotype group across tissues. This suggests changes CB1R expression is not responsible for the observed behavioral effects, and other mechanisms, such as changes in expression of other molecules within the ECS, are responsible for nociceptor CB1R-induced antinociception. Altogether, this data begins to verify mechanisms of nociceptor CB1R antinociception during PDPN, ultimately bridging the gap between preclinical and clinical studies.

Acknowledgements: This work is supported by R21DK130015, R35GM147094, Rita Foundation Award in Pain, and the University of Texas Rising STARS program research support grant.

Poster 22

Mast Cell Specific GPCR Regulates UTI Immune Response and Pain

Waris Muhammad Khuwaja[1], Colin Guth[2], Nicole De Nisco[1], Linghui Nie[3], Sudeshna Rakshit[3], Priyanka Pundir[2], Xintong Dong[1], Dustin P. Green[3],

1. Department of Biological Sciences, University of Texas at Dallas, Richardson, TX 75205
2. Department of Molecular and Cellular Biology, College of Biological Science, University of Guelph, Guelph, ON N1G 2W1, Canada
3. Department of Neurobiology, University of Texas Medical Branch, 301 University Blvd, Galveston, TX, USA

Urinary tract infections (UTIs) are among the most common bacterial infections worldwide and disproportionately affect the elderly. Furthermore, these infections can return and recurrent urinary tract infections (rUTI) represent a significant clinical burden, affecting nearly 25% of women within six months of an initial acute episode. While the underlying cause, invasion by uropathogenic *Escherichia coli* (UPEC), is understood, rUTIs remain poorly controlled, undermanaged, and gaps remain about our understanding of neuroimmune components that contribute to this debilitating recurrent disease. We discovered a mast cell-specific GPCR, Mrgprb2, that suppresses bladder immunity against UPEC. RNA-seq data reveals that in the absence of Mrgprb2, genes associated with inflammatory response are downregulated. Flow cytometry data shows that Mrgprb2 KO infected bladder has less immune cell and neutrophil infiltration. Our preliminary data also shows that Mrgprb2 is expressed on bladder mast cells localized near nerve fibers and these Mrgprb2+ mast cells are significantly increased in the bladder after UPEC infection. Importantly, our preliminary data demonstrated that knocking out Mrgprb2 increased bacterial clearance and reduced mechanical and ongoing pain weeks past initial infection. Additionally, Mrgprb2 knock out mice had significantly reduced bladder inflammation. Our goal is to reveal the functions of Mrgprb2 and its human homolog MRGPRX2 in bladder immunity and pain.

Poster 9

A Promising Target for Chronic Pain After Spinal Cord Injury: Investigations of Macrophage Migration Inhibitory Factor (MIF)

Kathy M. Guerra[1,2], Lisa R. Abe[1,2], Juan J. Herrera[1], Kimaya Savargaonkar[1], Carmen W. Dessauer[1], Edgar T. Walters[1], Alexis Bavencoffe[1]

1. Department of Integrative Biology and Pharmacology, McGovern Medical School at the University of Texas Health Science Center, Houston, TX, United States.
2. MD Anderson Cancer Center UTHealth Graduate School of Biomedical Sciences, Houston, TX, United States.

Corresponding author: Alexis Bavencoffe, Department of Integrative Biology and Pharmacology, UTHealth McGovern Medical School, 6431 Fannin St., 77030, Houston, TX. E-mail: alexis.bavencoffe@uth.tmc.edu.

Chronic pain afflicts more than half of people living with spinal cord injury (SCI). Despite extensive research, the mechanisms driving chronic pain after SCI remain poorly understood. Current treatments are inadequate, reinforcing the need to identify effective therapeutic targets. Our project focuses on a pleiotropic contributor, the pro-inflammatory cytokine macrophage migration inhibitory factor (MIF), for which we have recently demonstrated complex contributions to pain-related nociceptor hyperactivity after SCI. This study aims to define the necessity of MIF for chronic pain after SCI by using a mouse model of global deletion of MIF ($MIF^{-/-}$). Age- and sex-matched wild-type (WT) and $MIF^{-/-}$ mice underwent baseline assessment of mechanical sensitivity using the mechanical conflict (MC) assay. Mice then received a thoracic T9 spinal contusion, followed by Basso Motor Scale (BMS) scoring weekly for a month and post-injury behavioral testing. Lumbar dorsal root ganglia were harvested, and whole-cell patch-clamp (current clamp) recordings were performed on dissociated small-to-medium diameter sensory neurons, putative nociceptors. As previously reported, compared to nociceptors isolated from uninjured (naïve) WT mice, neurons from injured animals exhibit a pain-related nociceptor hyperactive state, showing ongoing firing when recorded either at rest or when artificially depolarized to -45 mV. This hyperactivity is supported by alterations in neuronal excitability, including depolarized resting membrane potential, hyperpolarized firing threshold, reduced rheobase, elevated input resistance, and enhanced depolarizing spontaneous fluctuations of membrane potential. These injury-evoked electrophysiological changes were significantly attenuated in $MIF^{-/-}$ neurons, and no sex differences were observed. Finally, we did not identify distinctions in the measures of basal excitability of neurons from naïve WT versus $MIF^{-/-}$ mice. Overall, this project reinforces the importance of MIF in driving nociceptor hyperactivity after SCI and suggests its relevance as a contributor to chronic pain mechanisms following spinal cord injury.

Acknowledgments: Gulf Coast Consortia NIH TIPS T32 GM139801 (Kathy M. Guerra), Mission Connect, a program of TIRR Foundation, 023-103 (Alexis Bavencoffe), NIH/National Institute of Neurological Disorders and Stroke R01 NS140665 (Alexis Bavencoffe). We are also grateful to Dr. Kyle Poulsen (Michigan State University) for providing the $MIF^{-/-}$ mouse line.

Poster 23

$Na_v1.8$ Inhibition by Suzetrigine Reduces Spontaneous Activity in Human Sensory Neurons

Mario Heles[1], Megan L. Uhelski[1], Yan Li[1], Nicolas Cortes-Mejia[1], Claudio Esteves Tatsui[2], Laurence D. Rhines[2], Robert Y. North[2], Patrick M. Dougherty[1]

1. Department of Anesthesia and Pain Medicine, University of Texas MD Anderson Cancer Center, Houston, TX, USA
   Neurosurgery, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Corresponding author: Patrick M. Dougherty, University of Texas MD Anderson Cancer Center, 1515 Holcombe Blvd., Houston, TX, USA

Chronic pain represents a major burden for many cancer patients and translation of chronic neuropathic pain from animal models to clinical conditions remains the most challenging part of a new drug discovery. While many targets and interventions identified in rodents work well in preclinical research, only a few succeed all the way from basic research to the market. Here, we describe a mechanism of action of the newly approved $Na_v1.8$ inhibitor Suzetrigine. We tested Suzetrigine on human dorsal root ganglia (DRG) neurons obtained from chronic neuropathic pain patients and healthy donor DRG neurons primed *in vitro* by pro-nociceptive chemokines.

Methods: We recovered human DRGs from patients undergoing thoracic vertebrectomy necessitating DRG removal for dorsal root compression relief and organ donors without a history of pain or nerve compression. DRGs were acutely dissociated and cultured for at least 12 hours before the experiment. We used the whole-cell patch-clamp recording technique to assess the spontaneous activity and changes in resting membrane potential, rheobase, and action potential characteristics.

Results: We developed an *in vitro*, human-based model of sensory neuron hypersensitivity in human DRGs. Cytokines of IL-6 family induced spontaneous activity, decreased rheobase, and depolarized membrane potential. Specifically, leukemia inhibitory factor (LIF) and oncostatin M (OSM) decreased rheobase, depolarized resting membrane potential, and increased the frequency of evoked action-potentials. Suzetrigine suppressed the spontaneous activity in both patient DRGs and sensitized donor DRGs. Suzetrigine also increased rheobase and hyperpolarized the resting membrane potential in spontaneously active cells, but had no significant effects on quiescent cells, supporting the activity dependent nature of Suzetrigine.

Conclusion: Ectopic discharge generation in DRGs is a hallmark of neuropathic pain development. We were the first to capture spontaneous activity generated by *in vitro* incubation of human DRGs in OSM, LIF, and IL-6, demonstrating a successful model of human DRG sensitization in a dish. Given the limited availability of human DRGs from patients with chronic pain, this represents a crucial step in translational pain research. Blocking $Na_v1.8$ by Suzetrigine successfully blocked spontaneous activity in both patient and sensitized donor DRGs. Dissecting the complexity of human nociceptor sensitization brings us closer to build better chronic pain models, that are crucial for the development of new, non-opioid analgesics.

Acknowledgements: This work was supported by NIH grants NS111929, NS130608, NS132483, NS102432 and NS104769.

Poster 32

Sensory Neuron Activity Promotes Tumor Progression in Malignant Peripheral Nerve Sheath Tumor (MPNST) via TRPV1⁺ Nociceptor Signaling

Khalil Ali Ahmad Kasm[1], Kechen Ban[1], Madeline Nicole Ansley[1], Anand Singh[1], Khushboo Irshad[1], Sara Maupin-Garcia[1], Cheng-En Shen[1], Seynabou Diagne[1], Maria Monserrath Olivera Acevedo[1], Tahara T Trimble[1], Momo K Harris[1], Aishani S Gargapati[1], Sami Mohd Rasheed[1], Kendal Willcox[1], Rajasekaran Mahalingam[1], Peter M Grace[1], Angela Hirbe[2], Andrew J Shepherd[1], and Yuan Pan[1,3]

1. Department of Symptom Research, the University of Texas MD Anderson Cancer Center, Houston TX
2. Division of Oncology, Department of Internal Medicine, Washington University School of Medicine, St. Louis, MO.
3. Department of Neuro-Oncology, the University of Texas MD Anderson Cancer Center, Houston TX

Abstract:

Purpose: Neural infiltration is a hallmark of malignancy and correlates with poor clinical outcomes across multiple cancers, including MPNST. While neuronal activity is increasingly recognized as a driver of cancer progression and pain, the specific sensory neuron subtypes and mechanisms that promote MPNST growth remain unclear. Using MPNST as a model of a highly innervated and painful cancer, we investigated whether TRPV1⁺ nociceptor activity directly contributes to tumor progression and cancer-associated pain.

Methods: Mouse MPNST cells were engrafted into the sciatic nerve to generate *in vivo* tumor models, followed by behavioral assays for pain and longitudinal tumor growth measurements. Aβ, Aδ, and C fibers were selectively stimulated to causally assess sensory neuron subtype-dependent effects on tumor progression. The TRPV1 antagonist AMG9810 was used in vitro and in vivo to evaluate the role of TRPV1⁺ nociceptors. Primary dorsal root ganglion (DRG) sensory neurons were cultured for multielectrode array recordings, calcium imaging, and co-culture with MPNST cells.

Results: MPNST-bearing mice developed mechanical and thermal hypersensitivity and exhibited ongoing pain. MPNST-conditioned media increased sensory neuron activity, intracellular calcium levels, and ATF3 expression in DRG neurons. Selective stimulation of C fibers, but not Aβ or Aδ fibers, enhanced tumor growth, an effect blocked by TRPV1 inhibition. Conditioned media from activated TRPV1⁺ DRG neurons increased MPNST cell growth *in vitro*, suggesting a role of neuronal activity-dependent paracrine factors in driving tumor cell growth.

Conclusions: MPNST is a painful and aggressive cancer originating in the peripheral nerves. Our findings reveal a feedforward loop that MPNST cells increase TRPV1⁺ nociceptor activity, which further promotes tumor progression. Targeting nociceptors may offer a dual therapeutic benefit by alleviating cancer pain and limiting MPNST growth.

Poster 24

Targeting Protein-Protein Interactions (PPI) Between Nav1.7 and FGF13 as a First-In-Class Mechanism for Safe and Selective Analgesia

Marijn Lijffijt[1], Zahra Haghighijoo[2], Aditya K. Singh[2], Jully Singh[2], Nana Goode[2], Ajay Pal[2], Hamid Teimouri[2], Samantha Davis[2], Amit Gupta[3], Gregory D. Cuny[4], Subo Yuan[5] and Fernanda Laezza[1,2]

1. IonTX Inc
2. Department of Pharmacology & Toxicology, University of Texas Medical Branch
3. TMC/ACT Program
4. Department of Pharmacological and Pharmaceutical Sciences, University of Houston College of Pharmacy Houston
5. Department of Neurobiology, University of Texas Medical Branch

Corresponding author: Marijn Lijffijt, IonTX Inc, 402 Charleston Street, Friendswood, TX, E-mail: marijn.lijffijt@proton.me

Protein–protein interactions (PPIs) are critical for cellular regulation and cell firing, and their disruption contributes to neurological disorders, making them promising therapeutic targets. Drugging PPIs remains challenging due to poorly defined pockets and difficulty replicating native interfaces. We investigated the intracellular interaction between fibroblast growth factor 13 (FGF13) and the C-terminal domain of Nav1.7, as a target for neuropathic pain previously addressed only with peptidomimetics. Leveraging a druggable pocket centered on FGF13 residue R117, we conducted molecular dynamics simulations and virtually screened 15 million compounds from the ZINC library, identifying eight candidates that were further validated using an in-cell split-luciferase complementation assay. Docking analyses confirmed key hydrogen bonds with R117. Two nanomolar-active compounds modulated Nav1.7 currents in patch-clamp assays, producing hyperpolarizing shifts in activation and inactivation, with activity dependent on FGF13. In a neuropathic pain model, IonTX Inc demonstrated analgesic effects in mechanical hypersensitivity testing. Ongoing studies are assessing pharmacokinetics, selectivity, blood–brain barrier permeability, and comprehensive toxicology profiles. These results reinforce the feasibility of targeting PPIs with small molecules and position the FGF13–Nav1.7 interface as a promising site for precision neuropharmacology.

Poster 10

Influence of Sex and Estrous Stage on Sensory and Affective Responding of Pain

Kaitlyn M. Little[1], Carlos A. Lopez Arteaga[1], Brooke E. Franklin[1], Meraa Husainat[1], Colin N. Haile[1], Therese A. Kosten[1]

1. Department of Psychology & TIMES, University of Houston

Corresponding author: Kaitlyn M. Little, Department of Psychology & TIMES, University of Houston, 4849 Martin Luther King Blvd, Houston, TX, kmlittle2@uh.edu

Fentanyl (FEN) is a highly potent synthetic opioid acting through mu opioid receptors and remains a major contributor to rising opioid use disorder (OUD) rates and opioid-related deaths. Sex differences in responses to psychoactive drugs, including opioids, are well documented, and circulating hormones such as estradiol influence addiction vulnerability in females. Preclinical studies show that pain elicits affective responses measurable through ultrasonic vocalizations (USVs), which are altered by opioids. However, few studies examine how opioids impact these affective responses or whether sex or hormonal state modulates them. We examined whether sex, estrous stage, or FEN differentially alter affective and sensory responses to a pain stimulus, as well as neural activation in pain-associated regions. Sensory regions included the periaqueductal gray (PAG), and affective regions included the amygdala. Adult male and female Sprague-Dawley rats were tested for: (1) locomotor activity and affective responses in an open field; (2) sensory and affective responses during FEN analgesia; and (3) neural activation to a pain stimulus through c-Fos expression. Preliminary findings indicate sex- and estrous-related differences in affective and sensory responses to FEN. In the open field, males emitted more USVs at 0.1 mg/kg FEN compared to 0.05 mg/kg and saline ($p < 0.01$), whereas females emitted more USVs at 0.05 mg/kg and minimal USVs at 0.1 mg/kg relative to males ($p < 0.05$). Females also showed greater locomotor activation at 0.05 mg/kg ($p < 0.001$). Tail flick latencies were lower in females at 0.05 mg/kg FEN, corresponding to non-proestrus phases ($p < 0.05$). FEN reduced USVs during tail flick tests in males ($p < 0.05$), with no effect in females. Analyses of c-Fos are ongoing. These outcomes clarify how sex and estrous stage shape behavioral responses to FEN and highlight key sex differences relevant to FEN-induced OUD and analgesia.

Acknowledgements: Office of the Assistant Secretary of Defense for Health Affairs through the Alcohol and Substance Abuse Research Program under Award No. W81XWH-18-2-0044 (CNH) and Michael C. Gibson Addiction Research Program (TAK)

Poster 11

Sex-Divergent Transcriptomic Regulation of Sensory Neurons in Oral Cancer

Jaclyn Merlo[1,2,3], Cristal Villalba [1,2], Tarek Ibrahim[1,2], Ping Wu[2], Li-Ju Wang [4,5], Fang-Mei Chang[1,2], Josue Murillo[1,2,6], Sergey Shein[3], Korri Weldon[4,7], Alexei Tumanov[3], Zhao Lai[4,7], Yidong Chen[4,5] and Shivani Ruparel[1,2]

1. Center of Pain Therapeutics and Addiction Research, School of Dentistry, UT San Antonio, Texas, USA
2. Department of Endodontics, School of Dentistry, UT San Antonio, Texas, USA
3. Department of Microbiology, Immunology and Molecular Genetics, UT San Antonio, Texas, USA
4. Greehey Children's Cancer Institute, UT San Antonio, Texas, USA
5. Department of Population Health Sciences, UT San Antonio, Texas, USA
6. Department of Pharmacology and Physiology, UT San Antonio, Texas, USA
7. Department of Molecular Medicine, UT San Antonio, Texas, USA

Corresponding Author: Jaclyn Nicole Merlo, Center of Pain Therapeutics and Addiction Research, UT San Antonio, 7703 Floyd Curl San Antonio, TX, E-mail: merloj@livemail.uthscsa.edu

Sex-dependent mechanisms of orofacial pain remain poorly defined, particularly in oral cancer where peripheral–neuronal interactions likely shape divergent pain phenotypes. To address this gap, we integrated transcriptomic profiling of tongue-innervating sensory neurons with single-cell analysis of the tumor-bearing tongue microenvironment in male and female mice. Bulk RNA sequencing of FACS-purified, retrogradely labeled neurons revealed several sex-specific features. Female trigeminal ganglia yielded a greater number of labeled neurons, and naïve female tongue-innervating neurons uniquely expressed immune-associated markers such as Csf1r and C1qa, which were absent in males and validated by immunohistochemistry. Functionally, immune cues such as CSF1 selectively sensitized TRPV1 responses in female neurons. Tumor growth triggered distinct transcriptional programs in each sex, with minimal overlap in differentially expressed genes: males showed enrichment of transcriptional regulators, whereas females exhibited increased expression of enzymes, cytokines, and chemokines. To identify potential peripheral drivers of these neuronal differences, we performed single-cell RNA sequencing of tumor-bearing tongue tissue. Cell type annotation identified major epithelial, stromal, vascular, immune, and neural compartments. Female tumors were enriched in CAFs, neutrophils, macrophages, and T cells, whereas male tumors exhibited heightened immune activation characterized by neutrophils, dendritic cells, macrophages, and cycling epithelial and mural/pericyte populations. Ongoing ligand–receptor analyses integrate these datasets to uncover sex-specific peripheral signals that may modulate sensory neuron function. These findings provide systems-level characterization of sex-dependent regulation of tongue-innervating neurons and the tumor microenvironment in oral cancer, revealing distinct tumor–neuron signaling pathways that may underlie sex differences in cancer-associated pain.

Poster 12

Mapping Corneal Nociceptive Innervation

Sreya Mitra[1], Md. Zeeshan Rasheed[1], Khalilah Oni[1], Kofi Asiedu[1], Tomasz Zal[1] and Anna Matynia[1]

1. College of Optometry, University of Houston

Corresponding author: Anna Matynia, University of Houston College of Optometry, JDA 2134, Houston, Texas Email: amatynia@central.uh.edu

The cornea is the most densely innervated tissue in the body and exquisitely tuned to painful stimuli, being predominantly innervated by unmyelinated C-fiber and thinly myelinated Aδ neurons, including PIEZO2-expressing mechano-nociceptors. Despite the high density, estimates suggest that only 1% of the total number of trigeminal neurons innervate the cornea, making it challenging to identify the predicted 200-250 corneal-projecting trigeminal ganglion neurons (cpTGs). The aim of this study was to identify cpTGs, including the hard-to-label PIEZO2 positive neurons. Corneal intrastromal injections (IST) of FM1-43 (1μg/μl) or AAV6-eGFP ($1.62x10^{13}$ GC/ml) were performed using a 34-gauge needle under anesthesia. Intraperitoneal injections (1.12mg/kg, IP) of FM1-43 were also performed. Corneas and TGs were harvested at 2, 7, 28 days or 2 months post injection. Whole mounts of corneas were immunolabeled for beta-III Tubulin and the viral reporter whereas TG sections were labeled with Hoechst for DNA. Images were captured by using an FV3000 confocal microscope (Olympus) and analyzed using an ImageJ plugin (EyeJ) to quantify corneal nerve fiber density. Both IST and IP injections of FM-143 sparsely labels PIEZO2-positive axons in the cornea whereas IST injection of AAV6-eGFP resulted in dense labelling of corneal axons. IST delivery of FM1-43 labels none to very few cpTGs, whereas IP injection of FM1-43 and IST injection of AAV6-eGFP robustly labels a large population of TG neurons in all ganglia or restricted to the ophthalmic ganglia, respectively. AAV6 transduces both C-fiber and Aδ neurons with comparable efficiency and the density of labeled corneal innervation correlates to the number of labeled cpTGs. The combined use of AAV6 using IST and FM1-43 using IP offers the potential to identify a the rare cpTGs and the hard-to-target population of Piezo2-positive cpTG. These will be powerful tools for advancing our understanding of the neuronal circuits mediating corneal pain perception.

Acknowledgements: We gratefully acknowledge the following support: U01EY034567 to AM and the UHCO core grant NIH P30 EY07551.

Poster 13

Understanding the Mechanism of Painful Radiation Induced Oral Mucositis (RIOM)

Priya Saraswathi Narayanan[1,2], Tarek Ibrahim[1,2], Viktor Drel[3], Shankara N Varadarajan[3], Brij B Singh[3], Shivani Ruparel[1,2]

1. Center for Pain Therapeutics and Addiction Research, School of Dentistry, University of Texas Health San Antonio, USA.
2. Department of Endodontics, School of Dentistry, University of Texas Health San Antonio, USA
3. Department of Periodontics, School of Dentistry, University of Texas Health San Antonio, USA

Radiation-induced oral mucositis (RIOM) is a common complication of head and neck cancer radiotherapy, characterized by mucosal swelling, ulceration, and oral pain. Current treatment options are limited, in part due to an incomplete understanding of RIOM pathophysiology. RIOM development is driven by radiation-induced reactive oxygen species (ROS), epithelial cell death, and subsequent inflammatory responses that lead to immune cell infiltration and ulcer formation. Because the ion channel transient receptor potential melastatin 2 (TRPM2) is strongly activated by ROS, we investigated its role in RIOM and assessed whether TRPM2 inhibition could mitigate disease severity. We evaluated TRPM2 expression in human normal oral keratinocytes (NOK) and mouse tongue tissue using immunohistochemistry. TRPM2 function after radiation was examined through calcium imaging and patch-clamp electrophysiology. An in vivo mouse model of RIOM was used to test whether pharmacological inhibition of TRPM2 affects key disease parameters, including epithelial erosion, ulceration, body-weight loss, and reduced food intake. Immunohistochemistry confirmed TRPM2 expression in both human NOK cells and murine oral keratinocytes. Radiation increased calcium influx in NOK cells, an effect blocked by TRPM2 inhibitors. Hydrogen peroxide–evoked TRPM2 activation was also enhanced in irradiated NOK cells. In vivo, mice developed hallmark features of RIOM, including epithelial breakdown, ulceration, impaired feeding, and weight loss. Treatment with the TRPM2 inhibitor clotrimazole significantly reduced radiation-induced oral mucositis in this model. Collectively, these findings identify TRPM2 as a key contributor to RIOM and suggest that targeting TRPM2 in oral keratinocytes may offer a promising strategy for preventing or reducing RIOM severity.

Poster 25

Anatomy, Projectome and Functional Annotation of Mouse Spinal Cord Neural Diversity

Helen Poldsam[#1,3], Liliana Cano Gomez[#1,3], Satoshi Ishishita[1,3], Ethan Irrobali[1], Helen C. Lai[1,3], Allan-Hermann Pool**[1,2,3]

1. Department of Neuroscience, University of Texas Southwestern Medical Center, Dallas, TX
2. Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX
3. Peter O'Donnell Brain Institute, University of Texas Southwestern Medical Center, Dallas, TX

Corresponding author: Allan-Hermann.Pool@utsouthwestern.edu, 6124 Harry Hines Blvd, University of Texas Southwestern Medical Center, Dallas, TX, 75390

Abstract

Spinal cord gives rise to central somatosensation and orchestrates autonomic and motor control. How its cellular diversity achieves physiological functions across sex and along its longitudinal axis and links spinal cord to higher order brain centers remains poorly understood. Here we used retrograde viral tracing with spatial transcriptomics and multiomic profiling of >750k mouse spinal cord neurons to define its output logic and functional cellular repertoire. We identified 70 spinal projection neuron classes that link the spinal cord to hind-, mid-, and forebrain centers implicated in itch, touch, and pain revealing the output wiring logic of the spinal cord. Furthermore, we characterized >500 anatomically and transcriptomically distinct neuron types that revealed extensive (~50%) rostro-caudal cellular specialization as well as first sexually dimorphic neuron classes in the spinal cord. Finally, we assembled a neuron-class-to-phenotype map of the spinal cord based on genetically defined interventions. As a result, we deliver an anatomically and functionally annotated cellular atlas of the complete adult mouse spinal cord to guide future interrogation of the organ.

Acknowledgements: The project is supported by UTSW Endowed Scholars Program, NIMH R01MH134799 (BICAN, Brain Initiative), McKnight Foundation and Rita Allen Foundation.

Poster 14

Elucidating the Spinal Output Circuit Mechanisms for Cancer Pain

Abhira Ravirala[1], Yoshiyuki Omura[1], Seungwon Choi[1]

1. Department of Psychiatry, University of Texas Southwestern Medical Center

Corresponding author: Seungwon (Sebastian) Choi, Department of Psychiatry, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., NC6.206, Dallas, TX, E-mail: Seungwon.Choi@utsouthwestern.edu

Many cancer patients experience severe chronic pain. Prolonged pain can lead to chronic stress in cancer patients, which in turn promotes tumor development, creating a vicious cycle. Therefore, effective pain management is key to not only improving cancer patients' quality of life but also slowing down cancer progression. Spinal cord projection neurons are attractive therapeutic targets for treating cancer pain because they carry all the pain signals emanating from the tumor microenvironment to the brain. Previously, we identified two genetically defined populations of spinal projection neurons (*Tacr1*$^+$ and *Gpr83*$^+$ subsets) that convey peripheral signals from the spinal cord to the lateral parabrachial nucleus in the brain, underlying affective aspects of touch and pain perception. Our research aims to determine spinal cord circuit mechanisms for cancer pain transmission and to use this knowledge to identify novel therapeutic targets for treating cancer pain. One of the most common sources of cancer-induced pain is bone destruction in metastatic bone cancer. Bone tumors compress and damage nearby peripheral nerves and secrete various cytokines that stimulate nociceptors, causing more severe pain as the tumor grows. Human bone cancer pain can be modeled in mice by injecting cancer cells, such as melanoma and lung cancer cells, directly into the tibia. We successfully established these mouse bone cancer pain models and confirmed that animals with bone cancer exhibit movement-evoked and spontaneous pain, reflected in decreased paw weight-bearing on cancer-injected limbs and increased pupil dilation responses, respectively. Intriguingly, TACR1 and GPR83 are structurally related "druggable" GPCR family members, both coupled to Gq signaling pathways, suggesting they may modulate the activity of Tacr1+ and Gpr83+ spinal projection neurons, respectively. To determine if neuropeptide signaling mediated by TACR1 and GPR83 is necessary for cancer pain transmission in the spinal cord, we have been analyzing spontaneous and movement-evoked bone cancer pain following spinal cord-specific knockout of the receptor genes, using newly generated transgenic mouse lines with conditional alleles for Tacr1 and Gpr83, combined with spinal cord-specific Cre lines (i.e., Lbx1-Cre). To explore the contribution of their ligands to cancer pain transmission, we also generated transgenic mouse lines with conditional alleles for the corresponding ligand genes, Tac1 (for TACR1) and Pcskn1 (for GPR83). We are currently testing whether primary sensory neuron-specific knockout of these genes, using a pan-sensory neuron Cre line (Adv-Cre), can reduce bone cancer-induced pain. Overall, our study will provide a mechanistic framework for understanding how ascending spinal pathways transmit cancer pain signals to the brain as well as new therapeutic substrates for treating cancer pain.

Acknowledgements: This work was supported by the Cancer Prevention and Research Institute of Texas (CPRIT) and the National Institutes of Health (NIH).

Poster 15

Theta (4–7 Hz) Band EEG Activity Associated with Cognitive Processing of Sensory and Affective Pain Descriptor Words

Anna Ronck, Bryten Ballenger, Sarah Love, Katie Malavar, Lauren Rowden, Ava Norris, Madison Wirtz, Swathi Suresh, Anna Park, & Amber Harris Bozer

1. Department of Neuroscience, Tarleton State University

Corresponding author: Anna Ronck, Department of Neuroscience, Tarleton State University, 1333 W Washington St., Stephenville, TX, Email: anna.ronck@outlook.com

Pain is multidimensional and has sensory-discriminative, affective-motivational, and cognitive-evaluative dimensions. While neural mechanisms that support the processing of pain have been extensively investigated, the EEG cortical activity that is linked to cognitive processing of pain, specifically within the sensory and affective dimensions, is under-explored. Thalamic dysrhythmia, a sustained theta-band thalamic activity is linked to cortical dysfunction which could potentially disrupt the cognitive processing of pain across the sensory and affective domains. The aim of this study was to assess theta (4-7 Hz) EEG activity originating from the C3 and C4 (somatosensory cortices), the F7 (thalamus), and F8 (anterior cingulate and insula) electrodes during the delivery of sensory and affective pain descriptor words and acute pain induced by a cold pressor. EEG data were collected (Magstim EGI; 256 electrodes) from each participant (N=20; 11 females) during four time bins: resting state EEG, during presentation of sensory and affective pain descriptor words, cold pressor pain with resting state EEG, and cold pressor pain with sensory and affective words. Pain descriptor words were taken from the validated Short Form McGill Pain Questionnaire and included sensory and affective words. One-way repeated measures ANOVAs were computed for theta EEG activity for each electrode of interest (C3, C4, F7, F8), and each time bin with sensory and affective treated as separate conditions, resulting in six distinct bins: (1) baseline resting state, (2) sensory pain descriptor words, (3) affective pain descriptor words, (4) cold pressor, (5) cold pressor with sensory words, and (6) cold pressor with affective words. Within-subjects tests revealed a main effect of time for theta activity for all four electrodes (C3, C4, F7, F8) (all $p < .001$). Bonferroni comparisons revealed that cold pressor pain alone did not elicit a significant increase in activity in any electrode ($p > .05$). In contrast, there was a significant increase in theta while processing sensory and affective pain descriptor words during the presentation of affective words with cold pressor pain at all electrodes, and during the presentation of sensory pain descriptors at C4 and F8 during cold pressor pain ($p < .05$). Affective words elicited higher theta activity than sensory words, with greater averages across all electrodes, including C3 and C4, typically associated with sensory processing. These findings expand the knowledge of theta band EEG activity in the processing of sensory and affective pain and can contribute to creating an in-depth understanding of the EEG cortical activity that underlies pain.

Acknowledgements: Research reported in this poster was supported by the National Institute of Neurological Disorders and Stroke of the National Institutes of Health under award number R15NS137286. This content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Poster 26

Spatial Mapping of Nociceptive Neuronal Subtypes in the Mouse Bladder

Ridma Sirimanna[1], Kathleen Halliburton[1], Xintong Dong[1]

1. Department of Biological Sciences, The University of Texas at Dallas, Richardson, TX, USA

Corresponding author: Xintong Dong, Department of Biological Sciences, The University of Texas at Dallas, Richardson, TX, USA, E-mail: Xintong.Dong@UTDallas.edu

The bladder is innervated by sensory neurons that contribute to normal bladder functions such as voiding and pain during cystitis or urinary tract infections. Recent advances in single-cell RNA sequencing have enabled a detailed classification of sensory neurons in dorsal root ganglia (DRG) into distinct neuronal subtypes, leading to the development of genetic tools to specifically label and study them in vivo. While most of this work focused on skin innervation, the specific subtypes of sensory neurons in the bladder have not been carefully characterized, and their anatomical distributions are unclear. To address this gap, we performed a comprehensive neuronal profiling of the bladder using Cre-dependent tdTomato reporter mouse lines to map the spatial distribution of specific nociceptive neuronal subtypes. Phosphoinositide interacting regulator of TRP (Pirt)-Cre; TdTomato mice that drive tdTomato expression in all sensory neurons served as a reference, revealing widespread innervation across the bladder. We found that Mas-related G protein-coupled receptor Mrgprd, and calcitonin gene-related peptide (CGRP) expressing nociceptor populations were present prominently in the urothelium layer of the bladder. In contrast, sodium channel Nav1.8 positive nociceptors were expressed in both epithelial and muscle layers of the bladder, and itch specific Mrgpra3 nociceptors were not observed in the bladder. These findings support our hypothesis that these subpopulations of sensory neurons may play distinct roles based on their localization, such as detecting chemical stimuli in the urine or regulating bladder contraction. This spatial map establishes groundwork for future studies investigating how distinct neuronal populations contribute to bladder function and to the pathogenesis of urological disorders.

Acknowledgments: This work is supported by NIH R01DK140039.

Poster 27

Frontal Alpha Activity And Its Dynamic Modulation During Cognitive Pain Processing In Chronic Pain

Swathi Suresh[1], Sarah Love[1], Anna Ronck[1], Bryten Ballenger[1], Katie Malavar[1], Lauren Rowden[1], Ava Norris[1], Madison Wirtz[1], Anna Park[2], Amber Harris Bozer[1]

1. Department of Neuroscience, Tarleton State University, Texas A&M System, Stephenville, Texas
2. Department of Psychology and Counseling, University of Central Arkansas, Conway, Arkansas

Corresponding author: Amber Harris Bozer, Department of Neuroscience, Tarleton State University, Texas A&M System, 1333 W Washington St., Stephenville, TX, E-mail: Bozer@tarleton.edu

Pain is a multidimensional experience comprising sensory–discriminative and affective–motivational dimensions that are supported by distinct yet interacting neural systems. Neural oscillations in the alpha frequency band are dynamic and context dependent, and are purported to reflect thalamocortical inhibitory gating mechanisms that regulate access to sensory information and higher-order processing. In chronic pain, resting-state alpha activity is frequently altered, suggesting changes in inhibitory control and attentional engagement. While alpha oscillations have been widely implicated in pain processing, their role in differentiating sensory versus affective pain dimensions during cognitive evaluation remains incompletely understood.

The present study examined frontal alpha modulation during cognitive processing of sensory–discriminative and affective–motivational pain dimensions, with nociceptive input manipulated independently. Twenty healthy control participants and eight individuals with knee osteoarthritis completed EEG recordings (256-channel; Magstim) across six experimental conditions: resting state, sensory pain descriptor words, affective pain descriptor words, an acute cold pressor task, cold pressor paired with sensory words, and cold pressor paired with affective words. Pain descriptor words were drawn from validated subscales of the McGill Pain Questionnaire. Alpha power (8–13 Hz) was extracted from frontal electrodes F7 and F8, selected for their relevance to thalamocortical and insular–cingulate systems involved in pain processing.

Mixed-model repeated-measures analyses revealed no significant main effect of group and no Group × Condition interaction, indicating similar alpha modulation patterns across experimental conditions in osteoarthritis and control participants. In contrast, a robust within-subjects effect was observed at both frontal sites ($p < .001$). Alpha power during the cold pressor task alone did not significantly differ from resting state, whereas conditions involving cognitive processing of pain descriptor words produced significant alpha modulation. Notably, alpha power increased during word-processing conditions even in the absence of nociceptive stimulation.

These findings demonstrate that frontal alpha activity is strongly modulated during the cognitive evaluation of pain dimensions, with similar modulation patterns observed regardless of diagnostic status or concurrent nociceptive input. The results suggest that alpha oscillations are particularly sensitive to dimension-specific cognitive processing of pain, rather than to nociceptive stimulation alone, in this experimental context. Ongoing work will expand sample size and incorporate broader cortical analyses to further delineate alpha dynamics across pain dimensions.

Poster 27

Acknowledgements: This work was supported by the National Institutes of Health (NIH) under an R15 (AREA) award and by institutional research support from Tarleton State University. The authors thank the undergraduate researchers of the Behavioral Neuroscience and Psychophysiology Laboratory, and the study participants for their contributions.

Poster 16

AAV Viral Tracing of Primary Sensory Neurons Using SELECTIV Mice

Samantha Tilley[1,2], Shae Stafford-Trujillo[1,2], Jiansen Yan[1], Nele Haelterman[1], Ben Arenkiel[1,2]

1. Department of Molecular and Human Genetics, Baylor College of Medicine
2. Jan and Duncan Neurological Research Institute, Texas Children's Hospital

Corresponding Author: Samantha Tilley, Department of Molecular and Human Genetics, Baylor College of Medicine, 1 Baylor Plaza, Houston, Texas, E-mail: Samantha.tilley@bcm.edu

Chronic musculoskeletal pain results in significant morbidity, especially given increased life expectancy. In a recent study, 70% of patients suffering from chronic musculoskeletal pain relied on pharmacological pain management. Effective pain management requires identification of peripheral mechanisms underlying joint pain to reduce reliance on therapies with high dependence risks, such as opioids. Towards this, identifying the sensory circuit pathways that transduce join paint is vital to developing a targeted peripheral therapeutic. However, current viral tracing efforts to reliably trace and identify the sensory neuron subtypes that innervate joints have remained inconsistent and difficult to reproduce. Viral transduction of sensory neurons has potential to carry a therapeutic payload which would offer new potential alternatives for pain management therapies. To overcome the challenges of virally tracing knee-innervating sensory neurons, we explored whether the newly developed SELECTIV (SELectiv Expression and Controlled Transduction In Vivo) mouse line, which allows Cre-dependent overexpression of AAV receptors, enhances viral transduction in targeted joint-associated tissues to permit reliable viral tracing from the knee joint. To test if SELECTIV mice can robustly label and identify the sensory neurons that innervate the mouse knee joint, we generated SELECTIV-Vglut2-Cre and SELECTIV-Advillin-Cre mice, followed by targeted knee injections of GFP-expressing AAV vectors. 2 weeks following unilateral intra-articular knee injections of an AAV-EGFP, we isolated lumbar dorsal root ganglia (DRG; L3–L5) to assess retrograde DRG AAV-EGFP expression compared to contralateral and uninjected controls. Preliminary data show EGFP expression in lumbar DRG neurons that innervate the knee. Compared to non-SELECTIV experiments, EGFP expression was more appreciable in DRGs obtained from SELECTIV models. Furthermore, IHC staining for GFP improved sensitivity for detecting knee-innervating sensory neurons revealing the neural tracts. These findings suggest that the SELECTIV system enhances transduction efficacy of peripheral sensory neurons, though additional validation and optimization are required. Together, this work provides a path toward more reliable approaches for labeling joint-innervating neuronal populations. Combined with other profiling methods, this strategy may help better identify candidate molecular markers of joint pain responses, informing future strategies towards more effective and/or targeted pain management.

Acknowledgements: This work was supported by the National Institutes of Health through the Helping to End Addiction Long-term Initiative and the National Institute of Arthritis and Musculoskeletal and Skin diseases (UCAR082200).

Poster 17

Pain is Associated with Decreased Emotional and Physical Function in Head and Neck Cancer Patients Residing in Rural and Urban Settings

Janet H. Van Cleave, PhD[1], Jennifer A. Rodriguez, BS[1], Carolina Gutiérrez, MD[2]

1. Department of Nursing Research, UTHealth Houston Cizik School of Nursing
2. Department of Physical Medicine and Rehabilitation Department, UTHealth Houston McGovern Medical School

Corresponding Author: Janet H. Van Cleave, PhD, Department of Nursing Research, UTHealth Houston Cizik School of Nursing, 6901 Bertner Ave, Houston TX, Email: janet.h.vancleave@uth.tmc.edu

Research in the general cancer population indicates that the prevalence of pain is greater in patients residing in rural locations than in urban locations. However, little is known about pain prevalence and characteristics among patients with head and neck cancer (HNC) residing in rural and urban locations in Southeast Texas. This descriptive study presents pain prevalence and characteristics reported by 33 patients receiving care in an academic surgical oncology clinic in Southeast Texas with a 13 county (7 urban, 6 rural) catchment area. The investigator team approached patients during a surgical oncology visit to determine interest in participating in the study. After obtaining informed consent, the team emailed patients a secure link to patient-reported outcome measures (PROs) built in REDCap (Research Electronic Data Capture) 1 week before their next HNC surgical oncology visit to enable real-time interventions during the visit. The digital PROs consisted of a valid patient-reported measure for HNC, the NYU Electronic Patient Visit Assessment (ePVA), a patient-reported measure composed of 21 symptom categories common to HNC, and the European Organization for Research and Treatment of Cancer (EORTC) QLQ-C30, a valid health-related quality of life (HRQoL) measure. Rural-Urban Commuting Area codes were used to classify patient addresses as urban versus rural. The participants' mean age was 63, and were primarily male (73%), White (82%), non-Hispanic (55%), and living in urban locations (57%). A majority of patients (57%, 19 of 33) reported experiencing pain, primarily at the throat (47%, 9 of 19), described as tenderness (58%, 11 of 19) and soreness (47%, 9 of 19). The presence and interference of pain in daily lives were significantly and negatively associated with decreased emotional and physical function (emotional: $p<.0001$; physical: $p<.01$). There was no association between rural or urban residence and pain prevalence ($p=.638$). The study findings suggest that pain interfering with patients' function is prevalent in HNC populations across both rural and urban populations in Southeast Texas. Further research is needed to determine the psychosocial and biobehavioral mechanisms involved in the association of pain and functional impairment. Study limitations include a single-site design and a small sample size, which limit the generalizability of the study's findings.

Acknowledgements: This research is funded in part by the National Institutes of Health (NIH), National Cancer Institute: *Implementing the NYU Electronic Patient Visit Assessment (ePVA) for Head and Neck Cancer In Rural and Urban Populations (R01CA282149)*

Poster 18

Investigating the Functional Circuit Assembly of the Anterolateral Pathway

Gabrielle Williams[1], Isabel Taggart[1], Gina Lee[1], Seungwon Choi[1]

1. Department of Psychiatry, University of Texas Southwestern Medical Center

Corresponding author: Seungwon (Sebastian) Choi, Department of Psychiatry, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., NC6.206, Dallas, TX, E-mail: Seungwon.Choi@utsouthwestern.edu

Each day, we experience myriad somatosensory stimuli: hugs from loved ones, warm showers, a mosquito bite, and sore muscles after a workout. These tactile, thermal, itch, and nociceptive signals are detected by peripheral sensory neurons that innervate the skin, propagated to the spinal cord, and transmitted to the brain via ascending spinal pathways. Therefore, ascending spinal circuits are key loci for early sensory integration and computation, which play critical roles in somatosensory perception and behavioral responses. While much is known about how we sense the external world in adults, very little is known about when and how we begin sensing and responding to diverse somatosensory stimuli during development. Our research seeks to address this fundamental question in developmental neuroscience by investigating the developmental logic behind the functional assembly of ascending spinal circuits. We first determined the developmental timing of anatomical and synaptic maturation in ascending spinal circuits, along with the emergence of behavioral responses to mechanical and thermal stimuli, in developing mouse embryos and neonates, using a unique set of mouse genetic tools combined with advanced anatomical and behavioral approaches. We are currently testing the hypothesis that early tactile and thermal experiences shape the functional assembly of ascending spinal circuits by analyzing sensory mutants that lack normal tactile and thermal sensation. Sensory abnormalities are prominent features of neurodevelopmental disorders such as autism spectrum disorders (ASDs). To investigate the potential role of impaired ascending spinal circuit assembly in the development of ASD-related sensory deficits, we began analyzing the functional assembly of these circuits in mouse models of ASD. Our study will provide insights into the mechanisms underlying somatosensory circuit development and identify potential therapeutic targets for treating neurodevelopmental disorders.

Acknowledgements: This work was supported by the Cancer Prevention and Research Institute of Texas (CPRIT) and the Whitehall Foundation.

Poster 28

Spatial Atlas of the Mouse Trigeminal Ganglion

Tingting Yang[1], Jin Li[3], Ye Zheng[3], Rose Ismail Elsaeidi[2], Yumei Li[3], Nele A. Haelterman[1], Stephen C. Pflugfelder[2], Joshua J. Emrick[4], Brendan Lee[1], Rui Chen[1,3]

1. Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, TX
2. Department of Ophthalmology, Baylor College of Medicine, Houston, TX
3. Gavin Herbert Eye Institute - Center for Translational Vision Research, Department of Ophthalmology, University of California Irvine School of Medicine, Irvine, CA
4. Department of Biologic and Materials Sciences & Prosthodontics, School of Dentistry, University of Michigan, Ann Arbor, MI

Corresponding author: Rui Chen, Gavin Herbert Eye Institute - Center for Translational Vision Research, Department of Ophthalmology, University of California Irvine School of Medicine, 850 Health Sciences Road, Irvine, CA, E-mail: ruic20@hs.uci.edu; Brendan Lee, Department of Molecular and Human Genetics, Baylor College of Medicine, 1 Baylor Plaza, Houston, TX, E-mail: blee@bcm.edu

The trigeminal ganglion (TG) is the primary neural hub for facial sensation and pain, containing three branches, ophthalmic (V1), maxillary (V2), and mandibular (V3) that innervate distinct anatomical areas to relay touch, temperature, and nociceptive signals. While recent studies have revealed the cellular diversity of the TG, little is known about how different cell types interact with each other or about how these interactions change in the context of aging or disease to modify pain sensation. To elucidate the extent of cellular and anatomical heterogeneity across and within the TG's branches, we generated a whole-organ, spatially resolved mouse TG atlas, revealing this organ's cellular organization at single cell resolution. We designed a Xenium gene panel by augmenting the mouse brain panel (247 genes) with 97 TG-specific gene markers and three reporters (eGFP, tdTomato, mCherry). We profiled the left and right TGs of a wildtype mouse, generating 87 consecutive 12-μm sections along the dorsoventral axis. After image-based segmentation, major cell classes were annotated by marker genes; neuronal subtypes were determined through integration with a single-cell RNA-seq reference. From a total of 3,340,355 cells (205,248 neurons; 3,135,107 non-neuronal), we identified all 8 major cell classes, encompassing 11 non-neuronal subtypes, and 15 neuronal subtypes.

The left and right TG showed closely matched proportions at both the major-class and neuronal-subtype levels, indicating left–right symmetry. Along the dorsoventral axis, both TGs exhibited increasing proportions of Mrgprd and Th neurons and decreasing proportions of Ntrk3+Ntrk2 and Trpm8 neurons, revealing an intra-ganglionic neural gradient. Neighborhood analysis further showed shifts in co-localizations of neuron types along the dorsoventral axis. In addition, we identified domain differences among the 3 anatomical branches. Beyond cell types, 11 local functional niches were identified with branch-dependent distributions. Finally, we reconstructed this data into a three-dimensional volume, providing the first mouse TG reference volume supporting true 3D analysis and registration of new 2D sections for apples-to-apples comparisons across specimens and conditions.

Together, this atlas provides a framework to link TG microanatomy to branch-specific sensory and pain functions and a baseline for interpreting disease, injury, and transgenic perturbations in a spatial context.

Acknowledgements: This study is supported by the National Institutes of Health through the National Eye Institute (EY034692) and the Helping to End Addiction Long-term Initiative and the National Institute of Arthritis and Musculoskeletal and Skin diseases (UC2AR082200).

Thank you to our Silver Sponsors

Novogene

Advancing Genomics, Improving Life

10x GENOMICS

Thank you to our Bronze Sponsor

SARTORIUS

Novogene

From core technologies to comprehensive solutions in DNA, RNA, amino acids, and proteins.

Genomics Sequencing

- Whole Genome Sequencing
- Exome Sequencing
- Long-read Sequencing
- *De novo* Sequencing
- 16S/18S/ITS Amplicon Sequencing
- Full-length Amplicon Sequencing
- Shotgun Metagenomic Sequencing

Transcriptomics

- Transcriptomics Sequencing
- Single Cell RNA-Seq
- Spatial Transcriptome Sequencing
- Non-coding RNA-Seq
- Whole Transcriptome Sequencing
- Meta-transcriptome Sequencing
- Exosome Long Non-coding/Small RNA-Seq
- Ribosome Profiling Sequencing
- Prokaryotic RNA-Seq
- Full-length Transcript Sequencing

Epigenomics

- Whole Genome Bisulfite Sequencing
- Enzymatic Methylation Sequencing
- Reduced Representation Bisulfite Sequencing
- ChIP-seq
- RIP-seq
- ATAC-seq
- Hi-C

Metabolomics and Proteomics

- Targeted Metabolomics (Human, Animal, and Plant)
- Untargeted Metabolomics (Human and Animal)
- Quantitative Lipidomics (Human and Animal)
- Widely-Targeted Metabolomics (Plant)
- Olink Reveal

Novogene - Your Trusted Genomics Partner

Novogene is a pioneer in applying advanced molecular biology technology and high-performance computing to research in the fields of life science and human health. Our vision is to be the global leader in providing genomic services and solutions. Owning the world's largest sequencing capacity, we utilize our deep scientific knowledge, first-class customer service, and unsurpassed data quality to help clients achieve their research goals in the rapidly evolving world of genomics. Novogene is committed to becoming your trusted genomics partner.

- Trusted International Enterprise
- Extensive Experience
- Professional Expertise and Technology
- Standardized Processes and Services
- High Quality and Efficient Delivery

Certified Service Provider

10x GENOMICS Certified Service Provider

illumina CSPro CERTIFIED SERVICE PROVIDER

ULTIMA GENOMICS CERTIFIED SERVICE PROVIDER

PacBio PACBIO CERTIFIED SERVICE PROVIDER

☎ 916-252-0068 ✉ inquiry@novogeneusa.com e www.novogene.com