5th Annual GCC Mental Health Conference

Sept. 19, 2023

BIOSCIENCE RESEARCH COLLABORATIVE
6500 Main St.
Houston, Texas

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 Antimicrobial Resistance, Cellular and Molecular Biophysics, Innovative Drug Discovery and Development, Immunology, Mental Health Research, Integrative Development, Regeneration and Repair, Single Cell Omics, and Translational Pain Research. GCC training programs currently focus on Biomedical Informatics, Computational Cancer Biology, Molecular Biophysics, Pharmacological Sciences, Precision Environmental Health Sciences and Antimicrobial Resistance. 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 M. D. Anderson Cancer Center, The Institute of Biosciences and Technology of Texas A&M Health Science Center and Houston Methodist Research Institute.

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Gulf Coast Consortia - Research

September 19th, 2023

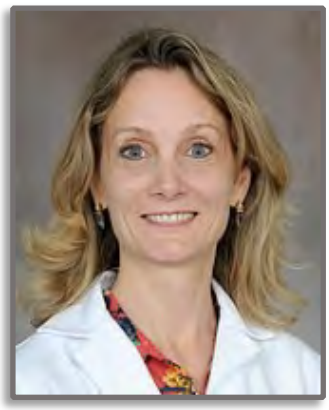
8:50	Welcome **Suzanne Tomlinson**, Gulf Coast Consortia **Fernanda Laezza**, Univ. of Texas Medical Branch
9:00	Keynote presentation *Breaking the Cycle: Exploring the Interplay Between Stress and Drug Use* **John Mantsch**, Medical College of Wisconsin
Session I Moderator	**Therapeutic Strategies for Psychiatric Disorders: Drugs and Devices** Christopher Verrico, Baylor College of Medicine
9:45	*Inventing an Intuitive Software System for Clinical Use of Wearables Data in Inpatient Psychiatry* **Michelle Patriquin**, Baylor College of Medicine/Menninger
10:00	*Toward Treating Mental Health Disorders with Minimally Invasive Bioelectronics* **Jacob Robinson**, Motif and Rice University
10:15	*Emerging Technologies in Healthcare - Digital Therapeutics* **Cameron Badger**, Big Health
10:30	*Digital Behavioral Healthcare Models* **Lacey Tezino**, Passport Journeys
10:45	Panel discussion
11:05	Coffee Break
Session II Moderator	**Stem Cell Research in Psychiatry** Consuelo Walss-Bass, Univ. of Texas Health Science Center Houston
11:20	*Opioid Exposure Affects Neural Stem Cell and Brain Development* **Ping Wu**, Univ. of Texas Medical Branch at Galveston
11:35	*Using Induced Pluripotent Stem Cells to Study Aging in Psychiatric Disorders* **Gabriel Fries**, Univ. of Texas Health Science Center Houston
11:50	*The Use of iPSC Models in Neuropsychiatric Research* **Xiaolong Jiang**, Baylor College of Medicine
12:05	Panel discussion
12:25	Lunch Break & networking, Poster viewing 12:25 Lunch 1:00 Poster session

1:45	Keynote presentation *Exploiting Individual Differences in cue-motivated Behavior to Identify the Neural Processes that Underlie Psychiatric Symptomatology* **Shelly Flagel**, Univ. of Michigan
Session III Moderators	**Therapeutic Strategies for Substance Use Disorder** Francesco Versace, MD Anderson Cancer Center Thomas Green, Univ. of Texas Medical Branch Galveston
2:30	*A Boost for Treatment? Developing an Anti-Fentanyl Vaccine* **Terri Kosten**, Univ. of Houston
2:45	*Contingency Management: Why it Pays to Quit* **Joy Schmitz**, Univ. of Texas Health Science Center Houston
3:00	*Clinical Phenotyping and Subtypes in Substance Use Disorders* **David Houghton**, Univ. of Texas Medical Branch Galveston
3:15	Panel discussion
3:35	Coffee Break
Session IV Moderator	**Gene X Environment Interaction in Psychiatry** Sonia Villapol, Houston Methodist Research Institute
3:50	*Neuromodulation of Bioengineered Organoids with Reactive Human Astrocytes* **Robert Krencik**, Houston Methodist Research Institute
4:05	*Biological and Environmental Risk Factors that Contribute to Persistent Mental Health Issues After Stroke* **Mario Dulay**, Houston Methodist Research Institute
4:20	*Exploring the Neurochemical Impact: How Peripheral Inflammation/Infection Influences the Brain* **Tatiana Barichello**, Univ. of Texas Health Science Center Houston
4:35	Panel discussion
4:55	Closing Remarks **Elizabeth Zuniga-Sanchez**, Baylor College of Medicine **Christopher P. Fagundes**, Rice Univ.
5:00	Reception

Presenters
In alphabetical order

Cameron Badger, EMBA
Director of Business Development
Big Health
Emerging Technologies in Healthcare - Digital Therapeutics

Cam Badger is a commercial leader that has extensive experience in the pharmaceutical, and digital therapeutics industry. Over the past 10 years Cam has engaged healthcare providers, health plans, and employers on integrating new therapeutics into their workflow to benefit their patients, caregivers, and employees.



Tatiana Barichello, PhD
Assistant Professor
Psychiatry and Behavioral Sciences
Univ. of Texas Health Science Center
Houston

Exploring the Neurochemical Impact: How Peripheral Inflammation/Infection Influences the Brain

Tatiana Barichello obtained her Pharmacy degree from the Federal University of Santa Catarina in Brazil. She earned a Master's and a Doctorate in Biochemistry from the Federal University of Rio Grande do Sul, Brazil. She was a Professor of Microbiology/Immunology and Dean at the School of Pharmacy at Unesc, Brazil. Since 2014, Dr. Barichello has been a faculty at the Department of Psychiatry and Behavior Science at UTHrealth, Houston. Dr. Barichello's research focuses on the relationship between inflammation, infection, and cognitive impairment. https://pubmed.ncbi.nlm.nih.gov/?term=barichello+T&sort=date&size=200

Mario Farin Dulay, Jr., PhD
Director, Neuropsychology Service, Neurosurgery, Houston Methodist Hospital
Director, The Houston Institute for Neuropsychological Knowledge (THINK) Lab
Assistant Professor, Neuropsychology in Neurological Surgery, Weill Cornell Medicine
Adjunct Assistant Professor, Psychology, Univ. of Houston
Adjunct Assistant Professor, Medicine, Texas A&M Univ. School of Medicine
Biological and Environmental Risk Factors that Contribute to Persistent Mental Health Issues After Stroke

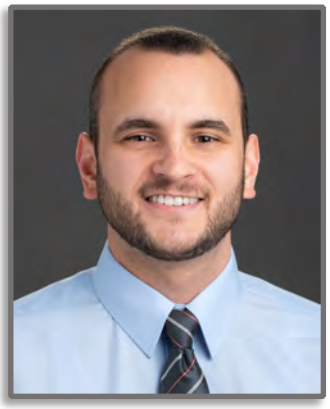
Dr. Dulay, Director of the Neuropsychology Service in Neurosurgery at Houston Methodist Neurological Institute (and Director of The Houston Institute for Neuropsychological Knowledge (Dulay THINK lab) uses neuropsychological, neurophysiological, psychophysical and neuroimaging methods to understand brain disease, and improve neurorehabilitation and neuropsychotherapy methods to bolster cognitive and emotional outcome after an acquired brain injury. Main research agendas nowadays involve understanding the human brain pre-to-post resective surgeries (standard resection, tailored ablative surgery) and neuromodulation surgeries (VNS, DBS, RNS) for intractable epilepsy, as well as evaluation and treatment of poststroke cognitive difficulties, poststroke depression, poststroke anxiety disorders, pseudobulbar affect, and post stroke apathy. Conducts pre- and post-brain-surgery neurocognitive evaluations, helps to diagnose dementia, provides cognitive rehabilitation treatments, and determines disability levels and long-term prognoses in his clinical duties. Undergraduate degree at San Diego State University, PhD in clinical neuropsychology at University of Cincinnati, internship at Baylor College of Medicine (BCM) Department of Psychiatry & Behavioral Sciences, postdoc at Texas Institute for Rehabilitation and Research (TIRR) at Memorial Hermann hospital and postdoc at BCM Department of Neurosurgery. Assistant Professor of Neuropsychology in Neurological Surgery at Weill Cornell Medicine, Adjunct Assistant Professor in the Dept. of Psychology at the University of Houston, Adjunct Assistant Professor in the Dept. of Medicine at Texas A&M University School of Medicine.

Shelly Flagel, PhD
Professor
Psychiatry
Univ. of Michigan

Exploiting Individual Differences in Cue-Motivated Behavior to Identify the Neural Processes that Underlie Psychiatric Symptomatology

Dr. Flagel earned her undergraduate degree (B.S.) in Biopsychology from the University of Michigan and then her Ph.D. in Neuroscience from the University of Michigan. She remained at the University of Michigan for her postdoctoral work with Dr. Huda Akil and ultimately became a faculty member there in the Department of Psychiatry. She has climbed through the ranks, most recently serving as the Interim Co-Director of the Michigan Neuroscience Institute and is now a Professor within the Institute and in the Department of Psychiatry. She is also an Associate Director of their Neuroscience Graduate Program. Her research focuses on the neural circuitry underlying individual differences in vulnerability to psychiatric disorders, with a focus on addiction and impulse control disorders. While her laboratory centers around a rodent model, she has recently started using a cross-species approach, that includes non-human primates and human children. Her work has been well-funded by NIDA for the past decade and more recently by NINDS the Pritzker Neuropsychiatric Research Consortium.

Keynote Presenter



Gabriel R. Fries, PhD
Assistant Professor
Translational Psychiatry Program
Univ. of Texas Health Science Center Houston

Using Induced Pluripotent Stem Cells to Study Aging in Psychiatric Disorders

Gabriel R. Fries, PhD, is an Assistant Professor in the Faillace Department of Psychiatry and Behavioral Sciences and a translational researcher in the field of biological psychiatry. His research focuses on the epigenetic basis of mood disorders, with a particular interest in bipolar disorder, suicide, and molecular mechanisms of stress. Fries' studies use basic science and investigation of postmortem tissues, cells, (epi)genomes, and clinical datasets to better understand disease mechanisms and inheritance, with the ultimate goal of designing novel medications and improving the lives of patients.

Fries received his Master's degree and his PhD in Biochemistry from the Federal University of Rio Grande do Sul, Brazil. He also completed a research fellowship at the Max Planck Institute of Psychiatry in Germany, before joining the UTHealth Department of Psychiatry and Behavioral Sciences as a postdoctoral research fellow (2015-2018) and later as an Instructor (2018-2019). As an Assistant Professor, he currently collaborates with basic and clinical investigators on the search for the genetic and epigenetic underpinnings of severe mental illnesses. He has published over 130 peer-reviewed articles (h-index = 37) and received awards for his research work from multiple scientific societies, including the American College of Neuropsychopharmacology (ACNP), the International Society for Bipolar Disorders (ISBD), the Society of Biological Psychiatry (SOBP), and the International College of Neuropsychopharmacology (CINP). Dr. Fries has received several grants as a Principal Investigator and is currently funded by the National Institute of Mental Health (NIMH), the Milken Institute, the American Foundation for Suicide Prevention (AFSP), the McGovern Medical School, and the UTHealth Department of Psychiatry and Behavioral Sciences.

David C. Houghton, PhD
Assistant Professor
Psychiatry and Behavioral Sciences
John Sealy School of Medicine
Univ. of Texas Medical Branch Galveston

Developing Personalized Treatment Approaches for Opioid Misuse in Pain Patients

Dr. Houghton joined the psychiatry faculty at UTMB in 2019, and he holds a joint appointment with the UTMB Center for Addiction Sciences and Therapeutics. He received his doctoral degree from Texas A&M University and completed his pre-doctoral clinical internship and post-doctoral fellowship at the Medical University of South Carolina.

Dr. Houghton conducts research on persons of all ages with psychiatric conditions characterized by compulsive and/or addictive behavior. Specifically, he is interested in developing precision medicine approaches in which treatments are tailored to individuals. He hopes that his findings will eventually lead to improved psychotherapeutic and pharmacological treatments for compulsive/addictive disorders.

Xiaolong Jiang, PhD
Assistant Professor
Neuroscience & Ophthalmology
Baylor College of Medicine
The Use of iPSC Models in Neuropsychiatric Research

Xiaolong Jiang received MD degree in Zhejiang University China and his PhD in Neuroscience at Uniformed Service University of the Health Science. He then completed his Postdoc training at the University of Virginia before joining Baylor College of Medicine. He is now Assistant Professor at Department of Neuroscience, Baylor College of Medicine.

Therese A. Kosten, PhD
Professor
Director of Developmental, Cognitive, & Behavioral Neuroscience
Director of Michael C. Gibson Addiction Research Program
Principal Investigator
Developmental Cognitive Neuroscience
Univ. of Houston
A Boost for Treatment? Developing an Anti-Fentanyl Vaccine

Therese A. Kosten, Ph.D. is the Moores Professor of Psychology and Head of the Developmental, Cognitive, and Behavioral Neuroscience Graduate program at the University of Houston where she is also the Scientific Director of the UH Animal Behavior Core. Dr. Kosten is the Director of the Michael C. Gibson Addiction Research Laboratory at UH. Research in the Gibson Addiction Laboratory investigates precipitants and consequences of Alcohol and Substance Use Disorders by employing sophisticated behavioral models in rodents with the aim of developing more effective treatment strategies and elucidating underlying mechanisms. Much of our research uses standard pharmacological manipulations but we also explore new avenues such as developing and testing anti-drug vaccines as well as examining alterations in the gut microbiota due to drug exposure. Some research is aimed at assessing epigenetic effects in response to developmental manipulations. Other research investigates the role of stress and explores potential sex differences in responses to psychoactive drugs and addresses related topics including emotion and learning that are relevant to affective and post-traumatic stress disorders.

Robert Krencik, PhD
Assistant Professor of Neurosurgery, Academic Institute
Assistant Member, Research Institute
Houston Methodist
Weill Cornell Medical College
Neuromodulation of Bioengineered Organoids with Reactive Human Astrocytes

Dr. Krencik received his PhD in Neuroscience at the University of Wisconsin-Madison and conducted a postdoctoral fellowship at the University of California, San Francisco. He is now a Principal Investigator in the Center for Neuroregeneration and Department of Neurosurgery at Houston Methodist Research Institute, and is also affiliated with Weill Cornell Medicine, Rice University, and Texas A&M University. His specific research goals are to uncover the mechanisms by which human astrocytes contribute to neuronal activity during healthy and diseased states. The experimental approach utilized in his lab consists of modulating functionally mature neural networks in the form of bioengineered organoid cultures that are generated from human pluripotent stem cells. Altogether, the purpose of these studies is to subsequently translate research findings into novel therapeutic strategies.

John Mantsch, PhD
Florence Williams Professor and Chair of Pharmacology & Toxicology at the Medical College of Wisconsin

Breaking the Cycle: Exploring the Interplay Between Stress and Drug Use

Dr. John Mantsch is the Florence Williams Professor and Chair of Pharmacology & Toxicology at the Medical College of Wisconsin. He received his B.S. in Psychology from Allegheny College and his Ph.D. in Pharmacology from Louisiana State University Medical Center in Shreveport. He completed his postdoctoral training under the mentorship of Dr. Mary Jeanne Kreek in the Laboratory of the Biology of Addictive Diseases at the Rockefeller University.

Dr. Mantsch joined the Department of Biomedical Sciences at Marquette University in 2001 and served as department chair from 2009-2020. In 2021 he joined the Medical College of Wisconsin as Professor and Chair of Pharmacology & Toxicology. In addition to his NIH-supported basic research program focused on the neurobiology of stress and addiction, Dr. Mantsch is involved in medication development and community-partnered research. He is a co-founder of Promentis Pharmaceuticals, Inc., a company that is developing therapies for neuropsychiatric disorders, and he leads a multidisciplinary research team that is working in partnership with community organizations and health agencies to understand neighborhood-level factors that influence mental health and substance use disorder outcomes and overdose risks.

Dr. Mantsch is the Chair of the Board of Scientific Counselors for the National Institute on Drug Abuse and a current member of the Pathophysiology of Mental Disorders and Addiction study section. He is also an editorial board member of the journal, Neuropsychopharmacology and a Fellow in the Milwaukee Social Development Commission Institute on Poverty and Systemic Racism. Dr. Mantsch has authored more than 70 scientific articles and book chapters and has taught and directed courses in pharmacology for undergraduate, graduate, health professional, and medical students.

Keynote Presenter

Michelle A. Patriquin, PhD
Director of Research
Senior Psychologist, The Menninger Clinic
Inventing an Intuitive Software System for Clinical Use of Wearables Data in Inpatient Psychiatry

Dr. Michelle Patriquin is the Director of Research and a Senior Psychologist at The Menninger Clinic and Associate Professor in the Department of Psychiatry and Behavioral Sciences at Baylor College of Medicine in Houston, Texas. She is board certified by the American Board of Professional Psychology (ABPP) and is a licensed psychologist. The Menninger Clinic is an inpatient and outpatient psychiatric hospital that has been a nationally ranked in the top 10 by US News for over 30 years. She has authored over 185 publications and presentations on her research, which has examined the subjective and objective precursors of mental illness including sleep problems, anxiety, depression, and suicide. Her work is funded by federal and foundation grants. She has been honored with multiple awards for her research and mentorship, including the New Investigator Award by the American Society of Clinical Psychopharmacology and the Rising Star Award by the Association for Psychological Science. She is has served as a standing reviewer for over 4 years on a NIH SBIR/STTR panel reviewing grants at the intersection of novel technology (apps, AI, sensors) and psychiatry/biobehavioral areas. At The Menninger Clinic, she leads a large, diverse team of faculty and staff to better understand mental health diagnoses and outcomes for intensive psychiatric treatment settings. Across her clinical work and research, she is committed to increasing evidence-based practice – particularly by designing and leveraging new technology – within intensive mental health treatment settings.

Jacob T. Robinson, PhD
Associate Professor, Rice Univ.
Co-Founder and CEO, Motif Neurotech

Toward Treating Mental Health Disorders with Minimally Invasive Bioelectronics

Jacob Robinson is an Associate Professor in Electrical & Computer Engineering and Bioengineering at Rice University, and an Adjunct Associate Professor in Neuroscience at Baylor College of Medicine. His research group uses nanofabrication technology to create miniature devices to manipulate and monitor neural circuit activity. He received a B.S. in Physics from UCLA in 2003 and a Ph.D. in Applied Physics from Cornell University in 2008. He then began a postdoctoral research position in the Department of Chemistry and Chemical Biology at Harvard University, where he created silicon nanowire devices to probe the electrical and chemical activity of living cells. In 2012, he joined the ECE and BioE departments at Rice. Dr. Robinson is a performer on several DARPA neurotech and bioelectronics programs and currently leads one of the N3 teams creating non-surgical neural interfaces. Dr. Robinson is the recipient of the DARPA Young Faculty Award, the Materials Today Rising Star Award, and is a Senior Member of IEEE. He previously served as the co-chair of the IEEE Brain Initiative and a core member of the IEEE Brain Neuroethics working group. He is the co-founder and CEO of Motif Neurotech, a neurotechnology company founded in 2022 out of his work on bioelectronics for wireless management of depression, started at Rice University with collaborators at Baylor College of Medicine and the University of Texas Health Science Center in Houston, TX.

http://www.robinsonlab.com
https://motifneuro.tech/

Joy Schmitz, PhD
Professor, Faillace Department of Psychiatry and Behavioral Sciences, McGovern Medical School, Uni. of Texas Health Science Center Houston

Contingency Management: Why it Pays to Quit

Dr. Schmitz is the Louis A. Faillace Professor of Psychiatry and Director of the Center for Neurobehavioral Research on Addiction (CNRA) at UTHealth. As Director, she oversees all administrative responsibilities and provides leadership in research and training activities. Research at the CNRA has been continuously funded by federal grants (NIDA, SAMHSA), private foundations, and the pharmaceutical industry. The current research portfolio at CNRA is translational, ranging from studies of genetics and cellular processes of addiction, to human laboratory-based studies of stress and alcohol use, to randomized clinical trials and treatment service delivery programs.

Dr. Schmitz has a long-standing research program focusing on the development and evaluation of behavioral and pharmacological treatments for substance use disorders. Her work seeks to improve patient outcomes by identifying moderators and mediators of treatment effects. She collaborates with leaders in neuroscience and genetics to develop targeted treatments aimed at biological factors underlying drug addiction. Dr. Schmitz has a long track record of continuous NIH funding and has completed studies with the NIDA Clinical Trials Network. She is author on more than 180 peer-reviewed publications and numerous book chapters.

Dr. Schmitz serves the scientific community in various capacities. Locally, she is a standing member of the UTHealth Internal Review Board (IRB), Six-Year Tenure Review Committee, the Neuroscience Research Center, and the department Executive Committee. Nationally, she serves as a standing member of the NIH's Interventions to Prevent and Treat Addictions (IPTA) Study Section. She is a founding member and Fellow of the Society for Research on Nicotine and Tobacco (SRNT) and an Associate Editor for Nicotine & Tobacco Research. She is an active member of the College of Problems on Drug Dependence (CPDD) and Division 12 of the American Psychological Association (APA).

Throughout her career she has maintained strong involvement in the career development of new investigators, serving as primary mentor of numerous graduate students, postdoctoral fellows and junior faculty, including NIH career-development (K) awardees. She has been recognized with mentoring awards, including the UTHealth President's Award for Mentoring Women, the Women Faculty Forum Clinical Excellence Award, and invited mentor for the NIDA Diversity Scholars Network meeting. She was recipient of the UTHealth President's Scholar Award for Excellence

in Research in 2020. She received the 2023 Marian W. Fischman Lectureship Award from the College on Problems of Drug Dependence (CPDD) in recognition of her contributions as an outstanding woman scientist in drug abuse research.

Lacey Tezino
Founder and CEO
Passport Journeys
Digital Behavioral Healthcare Models

Lacey Tezino is the Founder & CEO of an exciting new startup, Passport Journeys, the world's first teletherapy app focused on mother-daughter relationships. The app works by assigning each mother-daughter pair to a licensed clinician to facilitate healing, bonding and growth. The monthly subscription includes bi-weekly online therapy, a prescribed bonding activity, a thoughtful journal prompt and an assigned worksheet to help with communication. The app was recently approved and published on both IOS and Google app stores. Lacey plans to bring the app across the 50 states and then to targeted international markets. Before jumping into entrepreneurship, Lacey created a successful career as a healthcare IT leader with Cerner/Oracle. She worked in Doha, Qatar for three years to digitize clinical documentation for the entire nation by transforming 8 hospitals and 23 clinics from paper to electronic health records. Lacey most recently served as the Director of IT for the Menninger Clinic, one of the top 10 best psychiatric hospitals in the U.S.

Ping Wu, PhD
John S. Dunn Distinguished Chair in Neurological Recovery
Professor and Vice Chair for Research, Department of Neuroscience, Cell Biology & Anatomy
Univ. of Texas Medical Branch Galveston
Opioid Exposure Affects Neural Stem Cell and Brain Development

Dr. Wu received a medical degree from the Beijing Medical University (Peking University Health Science Center) in 1984, and Ph.D. at the University of Texas Medical Branch (UTMB) at Galveston in 1991. She is currently a Full Professor and Vice Chair for Research in the Department of Neurobiology and has been holding the John S. Dunn Distinguished Chair in Neurological Recovery since 2005.

Dr. Wu is an expert in stem cell research. Her innovative techniques and findings have earned both national and international recognition. The studies in Dr. Wu's team include 1) transplantation of primed neural stem cells to treat spinal cord injury, traumatic brain injury and ALS in rodent animal models, 2) application of human and rodent neural stem cell-derived neuron/astrocyte coculture to study the pathological mechanisms of neurotrauma, neuroinfection and substance abuse, and 3) usage of human neurons/astrocytes for drug screening. Dr. Wu's current studies, funded by two NIH multiple PI R01s in 2022, focus on the role of neural stem cells and microglia in brain development following insults such as viral infection and opioid exposure.

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Poster 1

FGF13 Ligands Represent Promising Scaffolds for the Development of New Anti-Pain Medications

Arman P[1], Haghighijoo Z[1], Goode N[1], Baumgartner T[1], Gupta A[2], Cuny G[3], Laezza F[1]

1. Department of Pharmacology & Toxicology, University of Texas Medical Branch, Galveston
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Pain is a multifactorial symptom that is directly and indirectly associated with many acute and chronic clinical manifestations. Pain can be further classified as nociceptive, neuropathic, or more recently nociplastic. Out of the 3, neuropathic pain has been associated with cancerous and non-cancerous etiologies. Despite the wide variety of pathologies, there are only 2 FDA approved drug classes for neuropathic pain: antidepressants and antiepileptics. As the current gold standard of pharmacological care, the antiepileptic drug class is composed of non-selective voltage-gated Ca^{2+} and Na^{+} channel antagonists that modulate neuronal activity and thus pain. From the central to the peripheral nervous system, there are 9 voltage-gated sodium channel (VGSC) isoforms ($Na_v1.1$-1.9) that are heterogeneously expressed throughout the human body. With respect to a unique class of intracellular fibroblast growth factors (iFGFs), advancements in our knowledge have highlighted this group as intrinsic modulators of VGSCs. By interrogating the biologically relevant pairs of iFGFs and VGSC isoforms, the potential to create a new drug class based on protein:protein interactions (PPIs) that specifically target pain relevant pathways could advance non-opiate neuropathic pain management. Regarding relevant pairings, $Na_v1.7$ and FGF13 have already been studied in stimulated dorsal root ganglion (DRG) pain models. For example, heat nociception and histaminergic-induced itch experiments have concluded that FGF13, in conjunction with $Na_v1.7$, work to increase inward I_{Na} resulting in increased neuronal outputs facilitating flick and itch responses, respectively. Given the literature surrounding $Na_v1.7$ and FGF13, *it reasonably stands that disrupting or modifying this specific protein interaction by identifying druggable cavities within FGF13 could provide a therapeutic benefit for pain pathologies*. To answer this question, a high-throughput (50 million small molecules) *in silico* screen was employed to highlight initial compounds of interest. In regards to the outcome of this large screen, 4 potential binding sites were found including an area participating in the $Na_v1.7$:FGF13 interface. Using this interface as an inclusion criteria, the initial 50 million compounds were reduced to 127 small molecules. Synthetic derivation and optimization potential were further used to narrow down the 127 compounds to a list of 20 compounds. In a single and dose-response format, these top 20 hits were then biophysically screened in a split-luciferase assay in order to validate our *in silico* conclusions. *As potential modulators of the $Na_v1.7$:FGF13 complex assembly, 5 compounds showed statistically significant differences compared to controls*.

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Poster 2

Perceived Discrimination and School Engagement among Urban Youth: The Role of Self-efficacy

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School engagement is an integrated, multidimensional construct consisting of attitude, investment, and commitment that students make toward school. Engagement in school is particularly relevant during adolescence, as previous findings suggest that students become more disengaged from school as they progress from elementary to high school. This is particularly relevant for minoritized youth. Active engagement in school enhances motivational factors (e.g., value, interest, competence, and mastery goals) and classroom contextual factors (e.g., peer support, teacher-student relationships, and autonomy support) that are critical to adolescents' academic success and transition into adulthood. Research suggests that 73% of minority urban youth experienced some form of discrimination and 42% of those experiences were "somewhat-" or "very disturbing". Perceived discrimination, specifically at school, has been linked with poorer academic performance, including lower grade point averages and student engagement. In contrast to discrimination experiences, self-efficacy has shown to be a key construct to promoting students' engagement and learning in the classroom. Accordingly, the present study aims to examine the extent to which self-efficacy moderates the association between perceived discrimination and school engagement.

Data were collected from 455 high school students (M_{age}= 16.54). Students were recruited across three high schools and three community organizations in the Chicagoland area. Each participant completed the *Adolescent Discrimination Distress Index,* the *Authoritative School Climate Survey, School Engagement subscale*, the *Brief Generalized Self-Efficacy Scale,* and the *School-Based Student Risk Scale*.

Correlational analyses demonstrated statistically significant, moderate to large correlations between constructs of interest in the expected directions. There was a statistically significant moderator effect of self-efficacy on the relationship between perceived discrimination and school engagement, as evidenced by the addition of the interaction term explaining an additional 20% of the total variance, $p < .0000$. The conditional effects of perceived discrimination at all values of self-efficacy were significant, however the relationship was the strongest for those that reported high levels of self-efficacy. That is, youth who reported less discrimination endorsed more school engagement at all levels of self-efficacy, with those who reported higher self-efficacy experiencing the strongest effect (b = -.29).

Findings from the present study suggest that self-efficacy plays an important role in the relationship between perceived discrimination and student engagement, even after controlling for student risk. Self-efficacy may be a salient focus of school-based interventions, especially when targeting minoritized populations. Future research should work to identify external factors contributing to the relationship between perceived discrimination and school engagement, in an effort to maximize academic engagement and success for adolescents.

Poster 2

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Poster 3

Deep Brain Stimulation of the Medial Forebrain Bundle: Effect on Suicidality Measures in Treatment-Resistant Depression

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Background

While promising in open-label trial, randomized-controlled trials of deep brain stimulation (DBS) for treatment-resistant depression (TRD) yielded inconclusive results. Interestingly, DBS seemed to be associated with incremental and sustained efficacy in the long term. Recently, the superolateral branch of the medial forebrain bundle (slMFB), a white matter tract part of the reward network, has gained interest as a potential target for DBS. The goal of this study was to characterize the trajectories of suicidality measures in a cohort of patients with TRD treated with slMFB DBS, and to compare these trajectories with those of other depressive dimension, such as anhedonia and behavioral activation.

Methods

Patients with severe TRD were included, and slMFB locations were mapped using diffusion sequences. A 4-week single-blind sham stimulation phase was followed by active stimulation. Later in the study course, the sham-stimulation duration was increased to three months. Clinical assessments were performed at baseline and weekly for the first year, then every 12 weeks until two years of follow-up, then every 6 months until five years of follow-up. Clinical assessments included the Montgomery–Åsberg Depression Rating Scale (MADRS), the Snaith-Hamilton Pleasure Scale (SHAPS), Behavioral Activation System (BAS) and the Columbia-Suicide Severity Rating Scale (C-SSRS).
To evaluate trends in the clinical measures of interest, we used generalized additive mixed models (GAMM). To test for any difference in the effect of the two stimulations conditions (sham- vs active stimulation), we used an interrupted time series (ITS) approach.

Results

No significant trends at the 1-year follow-up were found for any of the suicidality measures considered. Potential explanation for this result may be the low power of the analysis (due to the limited sample size), and the absence of suicidality symptoms for a significant number of participants at enrollment or during follow-up. Also, it might be that the maximal effect of DBS on TRD patient is not reached at one year.

Trends for depressive symptoms intensity (MADRS total scores) and intensity of anhedonia (SHAPS total scores) were significantly negative at both the 1 year and > 2 years follow-up, indicating reduction in depressive symptoms and anhedonia severity. Behavioral activation, as

measured by total BAS scores, showed a significant positive trend only at the > 2 years follow-ups.

The ITS modelling results showed no significant immediate or sustained effect of device activation for any of the measures considered. A major limitation of the ITS modelling was the imbalance in the number of time points for the two conditions (active- vs sham-stimulation).

Conclusions

In our cohort of TRD patients treated with slMFB DBS, different depression dimensions showed distinct trajectories during follow-up. Depression overall severity, anhedonia and behavioral activation improve at short- and long-term follow-ups (only long-term follow-up for behavioral activation), while no significant trends were evident for suicidality measures. We failed to detect a significant immediate or sustained effect of device activation.

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Poster 4

Pharmacologically Targeting the Nav1.6:GSK3β Complex to Mitigate Hyperexcitability in Neuropsychiatric Disorders

Authors

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Abstract

Voltage-gated Na+ channels (Nav channels) are transmembrane proteins with critical regulatory roles in synaptic function and neuronal firing. Nav1.6 is the most densely expressed Nav channel isoform in the adult human brain. Importantly, Nav1.6 plays a critical role in action potential initiation due to its localization at the axon initial segment, and therefore serves as the primary target for modulation of neuronal excitability. The Nav1.6 channel is regulated through its interactions with various auxiliary proteins and signaling molecules. Recent studies from our laboratory have revealed that glycogen synthase kinase 3β (GSK3β) binds the Nav1.6 C-terminal tail domain (CTD) and phosphorylates the T1938 residue of its CTD, indicating that GSK3β regulates the Nav1.6 channel via a dual-function mechanism including phosphorylation and complex formation. Functionally, genetic silencing of GSK3β suppresses Nav1.6-encoded currents, while increased phosphorylation of T1938 via GSK3β stimulates Nav1.6 activity and promotes maladaptive firing of neurons under vulnerable conditions. This evidence suggests that dysregulated GSK3β-mediated modulation of Nav1.6 facilitates neuropathological hyperexcitability. Using the split-luciferase complementation assay, we have identified a small molecule ligand that significantly inhibits Nav1.6/GSK3β complex assembly compared to vehicle control and binds appreciably to both GSK3β and Nav1.6 using surface plasmon resonance. Furthermore, 1063 reduces Nav1.6-mediated sodium currents in heterologous cells in a manner reminiscent of GSK3β knockdown. Using *ex vivo* patch clamp electrophysiology in 2-4 month old 3x-Tg-AD mice, we show that hyperexcitability in the CA1 region of the hippocampus is effectively mitigated following shRNA-mediated knockdown of GSK3β, and that the effects of 1063 in this region are dependent on the presence of GSK3β. To better understand the functional interface between Nav1.6 and GSK3β, mutagenesis screening was performed; indicating that this interaction is mediated by residues of the GSK3β axin-binding domain. Functional studies revealed that expression of the GSK3β axin-binding domain is sufficient to inhibit GSK3β-mediated upregulation of Nav1.6-mediated currents in heterologous cells, suggesting that residues within this region are required for GSK3β's functional effects on the channel. Cooperatively, these results illustrate a novel, druggable interface between Nav1.6 and GSKβ that holds potential as a therapeutic target for various hyperexcitability-related disorders.

Poster 5

Delivery of Telomerase Reverse Transcriptase mRNA as a Therapeutic Approach for Treating Traumatic Brain Injury

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Traumatic brain injury (TBI) is a major cause of death and chronic disabilities in the United States. Moderate to severe TBI can cause significant impairments in mental and motor functions or death. Currently, there is a lack of therapeutic countermeasures to address acute and chronic consequences. Within this context, gene therapy can provide an unmet solution in protecting against several neurodegenerative disorders, including TBI. Efficient mRNA therapies require a Lipid Nanoparticles (LNP) carrier to protect mRNA from degradation in the body fluids. Telomeres are repetitive non-coding DNA sequences have a pivotal role in tissue repair and aging. Telomere shortening in the brain results from blood flow impairment and cell-death related inflammation and affects tissue regeneration ability. A catalytic subunit of telomerase, an enzyme responsible for maintaining telomere length (TL) during cell division, is *telomerase reverse transcriptase (TERT*). TL dysfunction has been implicated in neuroinflammatory and neurodegenerative processes and proposed as a marker for TBI outcomes, while TERT was shown to be important in neuronal survival and cognition, protecting against oxidative stress and blocking neuronal apoptosis. While TERT is a potential target in neurological disorders, no studies evaluating RNA therapy to address TL in TBI were reported yet. Our findings demonstrate that brain cells after TBI suffer from telomere shortening compared to uninjured brains. Therefore, we have developed a LNP system encapsulating TERT to reverse the shortening of telomeres as well as neuroinflammation resulting from TBI. We performed a moderately intense controlled cortical impact injury (a mouse model of TBI) in 6-month-old adult male and female C57BL/6J mice and delivered TERT mRNA-LNPs or luciferase (Luc) to the brain via retro-orbital injection immediately following TBI. We also found that TERT mRNA-LNPs administration can selectively target the injured brain using in vivo imaging system (IVIS). We also demonstrate using immunofluorescence techniques that treatment with TERT-LNP reduces cell death (caspase-3+ and TUNEL+ cells) and microglia activation (Iba-1+) in male mice. TERT mRNA-LNPs showed significant suppression of inflammatory cytokines, tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) measured by Fluorescent in situ hybridization (FISH). In summary, these findings strongly suggest that delivering TERT mRNA-LNPs to the brain has the potential to effectively address acute TBI.

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Poster 6

Epigenetic GrimAge Acceleration and History of Suicide Attempt in Bipolar Disorder

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Background: Bipolar disorder (BD) is associated with reduced life expectancy and excess mortality. Suicide is a major cause of mortality in BD, and a previous history of suicide attempt has been associated with decreased lifespan. Although the risk of suicide in BD is high, excess mortality is mainly explained by somatic comorbidities, with recent studies suggesting that premature cellular senescence contributes to the shortened life expectancy seen in psychiatric disorders.

Hypothesis/Goals: History of suicide attempt in individuals with BD is associated with an acceleration of GrimAge, an epigenetic clock trained on time-to-death data and associated with mortality and lifespan.

Methods: Study participants from a discovery cohort (Houston) included individuals with BD with no history of suicide attempt (BD/non-SA, n = 66), individuals with BD with a lifetime history of suicide attempt (BD/SA, n = 77), and healthy controls (HC, n = 51) matched for age, sex, and race. An index of GrimAge acceleration (AgeAccelGrim) was computed based on peripheral blood genome-wide DNA methylation (DNAm) levels measured by the Infinium MethylationEPIC Beadchip (Illumina) and chronological age. ANCOVA models were used to compare groups for AgeAccelGrim as well as DNAm-based smoking pack-years and seven age-related plasma proteins. Results from the patient-specific comparisons were independently validated in a replication cohort (Iowa) including BD/non-SA (n = 47) and BD/SA (n = 47).

Results: In the discovery cohort, HC, BD/non-SA, and BD/SA significantly differed for AgeAccelGrim after controlling for age, sex, genetic ancestry, years of education, body mass index, smoking status, and blood cell counts ($F(2,175) = 7.864$, $p < 0.001$), with the highest AgeAccelGrim found in BD/SA, with an excess of mortality of 3 years compared to HC ($p = 0.001$). BD/SA also showed a significantly higher AgeAccelGrim than BD/non-SA in the unadjusted model ($p = 0.002$) and after adjusting for covariates in the discovery cohort ($p = 0.027$). Furthermore, this between-group difference was also replicated in an independent cohort in both unadjusted ($p = 0.02$) and adjusted models ($p < 0.001$).

Conclusions: Epigenetic GrimAge acceleration may contribute to premature morbidity and mortality in BD patients with a lifetime history of suicide attempt. These findings pair with existing evidence that not only BD, but also suicide attempt, may be associated with an acceleration of biological aging, and provide putative biological mechanisms for premature mortality in these conditions.

Poster 6

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Poster 7

Prelimbic Cortex Neurons Signal Risk-taking vs. Risk-avoiding Responses During an Approach-avoidance Conflict Test in Rats

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Neurons in the prelimbic (PL) subregion of the medial prefrontal cortex change their firing rates in response to fear- and reward-associated cues. PL activity is essential for the retrieval of both fear- and reward-related memories. However, it remains unknown how PL neurons respond during situations of conflict when reward cues and fear cues occur together in a fear-inducing context. To explore this question, male adult Long-Evans rats previously trained to press a lever for sucrose during the presentation of audiovisual cues were implanted with single-unit recording electrodes in the PL. Rats were then exposed to an approach-food vs. avoid-predator odor conflict model comprised of three phases: (i) reward phase, only food cues presented; (ii) cat odor phase, only a fear-inducing cat odor presented; and (iii) conflict phase, food cues concomitantly presented with cat odor. The next day, rats were returned to the same chamber and exposed to the food cues in the absence of the predator odor (fear-inducing context). During the approach-food vs. avoid-predator odor conflict test, rats displayed increased defensive behaviors and reduced food-seeking responses during conflict phase compared to reward phase. During the contextual fear test, clustering analysis of food-seeking responses revealed two distinct behavioral phenotypes. Risk-takers (RT) exhibited increased lever-press responses, whereas risk-avoiders (RA) exhibited reduced lever presses and increased avoidance behavior. Using single-unit electrophysiological recordings, we observed differences in PL food-cue responses between RT and RA during the fear-inducing context session. In RT, the magnitude of excitatory food-cue responses was maintained in the same cells from early to late phases. In contrast, excitatory food-cue responses in RA were attenuated from early to late phases. Both RT and RA showed the same number of excitatory and inhibitory food-cue responses during the early and late phases of the fear-inducing context. However, in RT, a larger fraction of the cells responded to food cues during both early and late phases (~19%) when compared to RA (~7%), suggesting that persistent food-cue responses in RT may help to maintain lever presses constant across the session. Tracking the activity of PL neurons trial-by-trial revealed an attenuation in the magnitude of the food-cue responses when rats did not press the lever (*i.e.*, risk-avoiding trials) compared to when rats pressed it (*i.e.*, risk-taking trials), which is consistent with our RT vs. RA food-cue comparison above. Together, these results demonstrate that modifications in food cue responses in PL neurons are associated with changes in behavioral choice during situations of conflict, suggesting a role for PL neurons in risky-decision making.

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Poster 8

TNFα and IL-18 Plasma Levels are Increased in Children and Adolescents with Familial Risk for Bipolar Disorder

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Bipolar Disorder (BD) is a chronic and severe psychiatric illness, affecting adolescents and young adults. It has a 60–80% heritability rate, and offspring of adults with bipolar disorder have a higher risk of bipolar spectrum disorders and psychiatric disorders. People with BD have altered both central and peripheral immune proteins, suggesting that inflammation plays a role in BD pathogenesis. Children who have a parent with BD, especially those who develop mood disorders, exhibit abnormal neuroimmune responses. Early intervention can improve disease progression and outcomes, while delays in treatment is associated with poorer outcomes. There are currently no molecular markers for intervention in high-risk individuals, or early detection and specific treatment targets for early-onset patients. This led us to investigate the presence of inflammation in plasma from children and adolescents with BD or a family history of the disease. Plasma samples were collected from 100 children and adolescents aged 7 through 17, at UTHealth's Center of Excellence in Mood Disorders. Upon clinical interview, patients were divided into three groups: 29 individuals with early-onset BD (BD Youth); 25 individuals with familial risk for BD (BD Offspring), all of whom had at least one parent with BD but did not have affective or non-affective diagnoses at the time of enrollment; and 46 non-psychiatric controls (Control). Tumor Necrosis Factor alpha (TNFα), Interferon (IFN), Interleukin (IL)-1b, IL-2, IL-6, IL-10, and IL-18 plasma levels were measured by commercial kits. Statistical analyses were performed to determine if each cytokine levels were different among groups. Outliers were removed and age, sex, and ethnicity were used as biological variables. Significance levels were determined at a value of 0.05. Data analyses were carried out using IBM SPSS Statistics 28 and GraphPad Prism 9. One-Way ANOVA followed by Tukey's multiple comparisons test revealed that TNFα and IL-18 plasma levels was increased in BD Offspring when compared to BD Youth and Control ($F(2, 87) = 4.617$, $p = 0.0124$, and $F(2, 89) = 7.069$, $p = 0.0014$, respectively). Other cytokines were below detection levels. Our results support the findings of the literature that individuals with familial risk possess an aberrant immune state, which further confirms plays a significant role in BD's physiopathology. While early-onset BD patients are expected to have similar proinflammatory profiles, many of them are prescribed medications that can influence cytokines levels, a confounder that was not investigated in this study. Future perspectives of this study include further investigation of inflammation and its role in BD risk, as well as its impact on depression and manic episodes, and the functioning and cognitive scores of patients. This can help identify warning signs earlier and improve intervention efforts.

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Poster 9

Early Antidepressant Use is Associated with Rapid Cycling Bipolar Disorder

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Rapid cycling bipolar disorder (RCBD) is associated with a more severe disease course. Identification of risk factors is crucial for prevention and appropriate treatment. We aimed to determine if the use of antidepressants as the first psychiatric medication ever prescribed is associated with RCBD in a sample of individuals diagnosed with BD. Individuals diagnosed with BD were enrolled from three clinical sites. All sociodemographic and clinical data were collected cross-sectionally. We built a Bayesian generalized linear multilevel model with RCBD as the dependent variable with a Bernoulli distribution. The site of enrollment was included as a random effect. We used the R package “brms,” which provides an interface to the Stan programming language. The sample size was 114 (86 with BD type I, 28 with BD type II). 32/114 (28%) were diagnosed with RCBD. Antidepressants were prescribed as the first psychiatry medication in 74/114 (65%) of BD patients. In a model including the first psychiatric medication ever prescribed, age of BD diagnosis, alcohol use disorder (AUD), substance use disorders (SUD), and gender as fixed effects and site of enrollment as random effect, only site of enrollment (coefficient estimate: 1.25, CI 95% 0.24-3.41), antidepressant as first medication prescribed (-1.30, -2.51 - 0.19) and presence of AUD (1.21, 0.21-2.27) were independently associated with the RCBD phenotype. After controlling for clinical variables known to be associated with RCBD, we found antidepressant use as the first psychiatric medication and AUD to be independently associated with RCBD.

Poster 10

Co-Occurring OCD and Autism: Clinician Perspectives

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Obsessive Compulsive Disorder (OCD) and Autism Spectrum Disorder (ASD) present with similar behaviors and symptoms, complicating diagnosis and treatment. Research has found that up to 37% of autistic individuals display OCD behaviors and symptoms. However, when examining clinicians, research found low confidence rates in treating co-occurring psychiatric conditions in autistic clients. The present study aims to (1) better understand the relationship between OCD and autism in realms such as treatment barriers, symptoms presentation, and treatment modifications and (2) determine the training needs for clinicians when working with autistic individuals exhibiting OCD behaviors. Eleven community clinicians were interviewed using a semi-structured, open-ended interview style. Questions related to OCD and autism discussed exposure therapy training, experience treating autistic youth with OCD symptoms, and challenges related to this treatment. Interview transcripts were analyzed using thematic analysis. Clinicians discussed topics that fell under three major concepts: treatment barriers, strategies for symptom differentiation, and treatment modifications. Results highlight varied clinician understanding of current research findings related to OCD-autism co-occurrence and many clinicians voiced their desire for more training. Findings indicate training should include information on identifying and treating OCD in autistic clients.

Poster 11

The Relationship Between Alcohol Use and Sexual Risk Behavior in Young Adults Experiencing Homelessness in Harris County

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Alcohol use is a growing public health concern due to its relationship to the development of health concerns and its link to sexual risk behaviors (i.e., condomless sex, sex with multiple partners). Young adults between the ages of 18-30 are in a developmental period known as “emerging adulthood” which is characterized by peak levels of substance use and associated problems. Minimal research has been conducted with the population of young adults experiencing homelessness who, in addition to experiencing a period of rapid developmental change, are also faced with unique challenges related to insecure housing. The purpose of this study is to examine how alcohol use contributes to sexual risk behaviors in young adults experiencing homelessness in Harris County. Participants enrolled in the HEARTS (HIV Education Awareness and Referral for Substance Use Treatment) program (N = 102) at UTHealth Houston were screened and assessed during intake using several assessments including the Alcohol Use Disorders Identification Test (AUDIT) and the sexual risk sub scores in the Risk Assessment Battery (RAB). Participants enrolled were between the ages of 18-30 and primarily consisted of heterosexual, non-Hispanic, Black males. (51.49% heterosexual, 84.31% non-Hispanic, 63.73% Black, 53.92% male). A linear regression analysis using STATA showed that more severe alcohol use significantly predicts a higher score on the RAB. $R^2 = 0.09$, $F(1, 93) = 9.45$, $p < 0.05$. This finding supports our hypothesis that higher rates of alcohol consumption are likely to lead to riskier sexual behaviors in young adults experiencing homelessness. Our results provide important insights into alcohol use and high-risk sexual behavior focusing on unhoused young adults in Harris County and have implications for developing psychoeducational and psychotherapeutic strategies addressing both domains. Further research and interventions focused on identifying alcohol misuse and high-risk sexual behaviors are needed to better assist unhoused young adults and guiding them to improve mental, physical and sexual wellness. Limitations of results include small sample size and the self-report nature of the surveys.

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Poster 12

Metabolic and Inflammatory Changes as Biological Mechanisms Underlying the Accelerated Pace of Aging in Bipolar Disorder

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Bipolar Disorder (BD) has been associated with accelerated epigenetic aging and metabolic dysregulation, although the potential link between these findings is unknown. We hypothesized that accelerated aging in BD is associated with a worse metabolic and inflammatory profile. To investigate this hypothesis, we employed DunedinPACE, an epigenetic age estimator, alongside DNA methylation-based surrogates of metabolic, anthropometric, and inflammatory markers. These evaluations encompassed both controls (n=39, 71.2% female) and individuals with BD (n=86, 71.4% female), matched for age, sex, and race/ethnicity. We also computed a 'metabolic index' and an 'inflammatory index' consisting of 14- and 3- markers, respectively indicative of metabolic dysfunction and inflammation. Specifically, varimax rotation was applied after computing Kaiser-Meyer-Olkin and Bartlett's test of sphericity. Finally, the patient group was subdivided (median split) into those with high (n=43) and low DunedinPACE (n=43) for downstream comparisons. Our results showed that individuals with BD had significantly higher DunedinPACE compared to controls when controlling for age, sex, genetic ancestry, and smoking ($p=0.046$). Mediation analyses showed significant indirect effects for the composite index of metabolic dysfunction ($ab=0.22$, BC 95% CI=0.1077 to 0.3616) and inflammation ($ab=0.06$, 95% CI=0.035 to 0.1013) on the relationship between the diagnosis and DunedinPACE. High DunedinPACE was associated with a significantly higher metabolic dysfunction index in both groups ($p<0.001$). Specifically in patients, those with high DunedinPACE had significantly higher levels of c-peptide, insulin-like growth factor-binding protein-4, hepatocyte growth factor, adrenomedullin, TIMP metallopeptidase inhibitor 1, plasminogen activator inhibitor-1, growth/differentiation factor (GDF)-15, GDF-8, cystatin C, beta-2-microglobulin, waist-to-hip ratio, body mass index, body fat, interleukin-6, transforming growth factor-α, and C-reactive protein, as well as lower levels of insulin receptor, leptin, HDL cholesterol, and GHR compared to those with low DunedinPACE ($q<0.001$ for all). We conclude that worsening metabolic parameters are associated with accelerated pace of aging in BD, suggesting them as important targets for prevention of aging acceleration and its consequences in patients.

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Poster 13

Impaired Hippocampal GABA Signaling and Social Behavior Caused by Neurodevelopmental Exposure to Deltamethrin are Rescued by Activation of Parvalbumin Neurons

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Pyrethroid pesticides have become one of the most commonly used classes of pesticide in recent years due to their favorable safety profile in adults. However, recent concerns have been raised regarding their ubiquitous use and associations between increased exposure with the occurrence of neurodevelopmental disorders, including attention deficit hyperactivity disorder (ADHD), and autism spectrum disorder. For example, children with detectable urinary metabolites of pyrethroids are twice as likely to be diagnosed with ADHD. Likewise, animal research indicates that exposure to deltamethrin (DM), a type of pyrethroid pesticide, alters neurogenesis in the hippocampus, locomotor activity, impulsivity, attention, and memory, all of which have been associated with neurodevelopmental disorders in humans. However, the impacts of early-life DM exposure during gestation and lactation on synaptic function in the hippocampus and associated behaviors in developmentally exposed pups have not been well-characterized.

Using a developmental exposure model, in which pregnant dams were exposed to 3 mg/kg/72 hours, equivalent to 1mg/kg/day, DM or vehicle through pregnancy and lactation, we measured synaptic function in the hippocampus beginning between PND 45-60. We found that long-term potentiation showed a significant decrease in male DM-exposed animals, a deficit which is rescued by the application of 0.5 μm clonazepam, indicating a dysfunction in GABA signaling in the hippocampus in DM-treated males. To further delineate disruptions in hippocampal GABA signaling caused by developmental exposure to DM, we targeted parvalbumin (PV) neurons in the hippocampus using a combination of PV-Cre transgenic mice and chemogenetic viral vector injection with AAV-hSyn-DIO-hM3D(Gq)-mCherry. Doing so allowed for targeted, increased firing of PV neurons in the hippocampus of control and DM-exposed animals.

Locomotor activity, novel object recognition, sociability and social novelty were measured in control and DM-exposed males injected with saline or a chemogenetic ligand (0.01 mg/kg JHU37160). No difference was seen in saline injected control and DM-injected animals in locomotor activity, novel object recognition, or sociability. DM exposure during development resulted impaired social novelty in 3-chamber social testing, an effect which was rescued by hippocampal PV neuron activation in DM animals. Likewise, activation of hippocampal PV neurons in controls impaired social novelty in control animals. Additional hippocampal recordings showed that PV neuron activation rescued long-term potentiation in DM animals but caused disruptions in control animals. Taken together, these results may indicate a critical role of hippocampal PV neurons in social behaviors and that the neurotoxic effects of DM exposure during development may be a contributing factor to the occurrence of neurodevelopmental disorders.

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Poster 14

TNFR1 Signaling Converging on FGF14 Controls Neuronal Hyperactivity and Sickness Behavior in Experimental Cerebral Malaria

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Abstract

Excess tumor necrosis factor (TNF) is implicated in cerebral malaria and other neuroinflammatory syndromes. Exploring signaling mechanisms that underly TNF-induced effects on neuronal excitability, we found that the complex assembly of fibroblast growth factor 14 (FGF14) and the voltage-gated Na^+ (Na_v) channel 1.6 ($Na_v1.6$) is increased upon tumor necrosis factor receptor 1 (TNFR1) stimulation via Janus Kinase 2 (JAK2). On account of the dependency of hyperinflammatory experimental cerebral malaria (eCM) on TNF, we performed patch-clamp studies in slices from eCM mice and showed that *Plasmodium chabaudi* infection augments $Na_v1.6$ channel conductance of CA1 pyramidal neurons through the TNFR1-JAK2-FGF14-$Na_v1.6$ signaling network, which leads to hyperexcitability. Hyperexcitability of CA1 pyramidal neurons caused by infection was mitigated via an anti-TNF antibody and genetic silencing of FGF14 in CA1. Furthermore, knockdown of FGF14 in CA1 reduced sickness behavior caused by infection. Thus, FGF14 may represent a therapeutic target for preventing TNF-mediated neuroinflammation.

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Poster 15

Depleting the Gut Microbiota Leads to a Reduction in brain Lesions and the Neuroinflammatory Response Following Brain Injury in Mice.

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Traumatic brain injury (TBI) is a common cause of disability and mortality, especially in young adults and the geriatric population. Following a TBI, the rapid neuroinflammatory response driven by microglia/macrophage cells contributes to subsequent brain damage and neurodegeneration. The gut microbiome plays a crucial role as a modulator of the immune system, influencing microglia activation after a TBI. In this study, we aim to understand the role of the microbiota in the activation of microglia following TBI. We performed a double TBI using moderate-to-severe controlled cortical impact (CCI) injury in 8-10 weeks-old male mice. The first CCI injury was done on day one, and microbiota depletion on days 35, 36, and 37 post-CCI. Bacterial depletion was achieved using a non-absorbable antibiotic (ABX) cocktail (ampicillin [1 mg/mL], gentamicin [1 mg/mL], and vancomycin [0.5 mg/mL]). From the collected fecal samples, a reduction of 77% in the concentration of bacterial DNA was observed in the group treated with ABX compared to the group with the standard microbiome. The second CCI injury was performed on day 39, mice were sacrificed on day 43, and brains were collected for immunohistochemical analysis. We performed histological analysis using cresyl-violet staining to assess lesion volume measurements and immunohistochemical techniques using Iba-1, F4/80, and CD68 antibodies to identify microglia/macrophage cells. Our results showed that the group of animals with microbiota depletion had a 25% decrease in lesion volume among the animals subjected to microbiota depletion, along with diminished microglial reactivity five days after the second CCI injury, as opposed to the group not subjected to ABX treatment. These findings indicate that depleting the microbiota after CCI can be advantageous in the event of a second TBI, as it lessens the injury and acute neuroinflammatory response. ABX treatment reduces microglia activation and lesion volume after TBI in mice. In summary, we found that microbiome depletion in injured mice has a beneficial effect. Therefore, administering ABX in clinical settings after TBI could benefit brain recovery.

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Poster 16

Unraveling Synaptic Specificity in Nervous System Development: Role of Non-Clustered Protocadherins

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Cellular and synaptic specificity is an essential mechanism of the nervous system in development used to address neuronal diversity. Precise cellular expression profiles of molecular players, such as cell adhesion molecules, are essential to determine cellular and synaptic specificity within the brain. Disruption of cell adhesion molecule expression is associated with different mental health conditions during development and neurodegeneration. The non-clustered protocadherins present a specific expression profile throughout the brain and have been associated with mental diseases such as intellectual disability (ID), epilepsy, Fragile-X syndrome (FXS), and autism spectrum disorders (ASDs). However, the molecular mechanism played for the non-clustered Pcdhs remains unclear. In this study, we use the mouse retina as a model to unveil the non-clustered protocadherin function and its potential implications for cellular and synaptic specificity. We postulate first that Pcdh9 is indispensable as a cell adhesion molecule in developing the cellular specificity in neurons, and disruption could lead to the underdevelopment of neurons. We found distinct cell adhesion molecules belonging to the non-clustered Protocadherin family to be expressed in cone bipolar but not rod bipolar cells. Protocadherin 9 (Pcdh9) is highly expressed in the developing synaptic layer and restricted to cone bipolar cells. To assess the function of Pcdh9 in synaptic connectivity, we crossed a floxed allele of Pcdh9 with a Chx10cre transgenic mouse line to conditionally remove Pcdh9 throughout the developing retina. We refer to this cross as Pcdh9 CKO. Using available antibodies, Retinas from Pcdh9 CKO and controls were fixed and analyzed for synaptic defects at various developmental ages. Disruption of Pcdh9 results in cone bipolar cells type 2 failing to extend their dendrites to the synaptic layer or OPL. We also observe that cone terminals have an abnormal morphology due to loss of Pcdh9. In addition to the morphological defects, we also observe defects in synaptic connectivity where there is a significant decrease in protein expression of the pre-synaptic marker Bassoon in Pcdh9 CKO compared to controls. These data suggest that Pcdh9 may be essential in mediating connectivity between cone photoreceptors and their cone bipolar targets. Our findings highlight a new role for Pcdh9 in the synaptic connectivity of the cone pathway. Future work will focus on disseminating the cellular and molecular mechanisms of Pcdh9 in the synaptic specificity of the developing retina. These insights contribute to our understanding of neural specificity and provide avenues for addressing neurodevelopmental disorders.

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Poster 17

The Gut-Brain Connection: Exploring Gut Dysbiosis and Depressive Symptoms Following COVID-19 - A Cross-Sectional Study

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Despite initial respiratory symptoms, neuropsychiatric manifestations have been reported in many COVID-19 patients. Gut dyshomeostasis and immune dysfunction after SARS-CoV-2 infection leads to long COVID mental health disturbance in COVID-19 survivors. We aimed to investigate the psychological symptoms, gut microbiome status, and C-reactive protein levels (CRP) in post-COVID individuals. This cross-sectional study included age and sex-matched individuals with health controls (n = 236) and post-COVID (n = 114). The evaluation of existing MDD, anxiety disorder, and the potential for suicide risk was conducted using the MINI International Neuropsychiatric Interview (MINI-Plus). This structured clinical interview relies on DSM-IV criteria for its assessment. The depressive and anxiety symptoms were assessed by the Hamilton Rating Scale and the stress level by an inventory of stress symptoms and circulating CRP levels measured. In a small cohort (post-COVID = 18 and controls = 46), the gut microbiome was evaluated by 16S rRNA sequencing. Post-COVID individuals exhibited greater severity of depressive symptoms ($p = 0.034$), higher levels of stress ($p = 0.020$), and CRP ($p = 0.014$) as compared to controls. There was no difference in α-diversity (which measures species richness and relative abundance of each species) but β-diversity (referred to as true diversity or between-group diversity) (Figure 1; $p = 0.001$) was significantly different between control and post-COVID groups. Interestingly, post-COVID individuals with depression had greater CRP levels than those with COVID-19 without current MDD ($p = 0.023$). Individuals who have experienced the aftermath of COVID-19 have exhibited pronounced psychological symptoms and changes in gut microbiome levels. While it is essential to conduct longitudinal research to fully comprehend the mental health trajectory of COVID-19-affected individuals, the concentration of CRP holds the potential as a valuable biomarker for identifying post-COVID depression at an early stage.

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Figure 1: **Altered gut microbiome in post-COVID-19 patients**: Feces from COVID-19 (n = 18) and control (n = 46) individuals subjected to 16S rRNA sequencing, (A) β-

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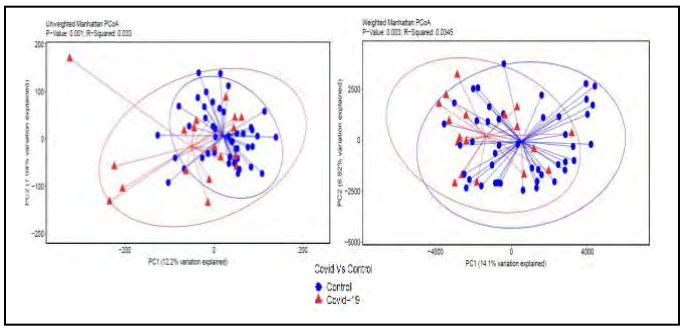

diversity results revealed a significant difference between COVID-19 and control subjects, unweighted, p=0.001; weighted, p=0.003.

Poster 18

Proteomic-based Interactome of Nav1.6 Reveals Sex-Specific Biosignatures of Resilience and Vulnerability.

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Background: Resilience and vulnerability to neuropsychiatric disorders are linked to molecular changes underlying excitability that are still poorly understood. In previous studies we identified the voltage-gated Na^+ channel Nav1.6 as a mediator of neuroplasticity induced by environmentally enriched (EC) or isolated (IC) conditions which are used as models for resilience and vulnerability. Protein-protein interactions play a key role in regulating Nav1.6 channel function and whether the relative composition of the Nav1.6 interactome in EC/IC models is different is not known. This study aimed at characterizing the interactome of Nav1.6 in EC/IC models to search for proteomic-based biosignatures of maladaptive plasticity underlying resilience and vulnerability to neuropsychiatric disorders.

Hypothesis/Goals: The hypothesis tested in this study was that the Nav1.6 protein-protein interaction network is differentially regulated by EC/IC in the striatum and hippocampal regions.

Methods: Immunoprecipitation, Western blotting, LC/MS/MS.

Results: To determine the impact of the EC/IC behavioral paradigm on the composition of the Nav1.6 channel macromolecular complex, we housed rats in environmentally enriched (EC) and isolated conditions (IC) for 30 days. The Nav1.6 immunoprecipitated fractions of EC and IC rats revealed 165 and 63 protein interactors of Nav1.6 differentially expressed in the striatum and hippocampus respectively. PANTHER protein class analysis revealed that most of the differentially expressed proteins in the striatum and hippocampus are involved with RNA metabolism (19%) and translation (25%) respectively. Classification by biological process revealed 31% of proteins in the striatum and 34% of proteins in the hippocampus are involved in cellular processes. Also, 32% and 31% of proteins in the striatum and hippocampus respectively are involved in binding while and 23%(striatum) and 25%(hippocampus) have catalytic activity.

Conclusion: The results of this study offer valuable information for identifying new molecular targets suitable for the development of novel neurotherapeutics.

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Poster 19

Characterization of Human Synaptic $GABA_A$ Receptors Function in Alcohol Use Disorder Post-Mortem Brains

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Previous research highlights epigenetic changes made to $GABA_A$ Receptors ($GABA_ARs$) following chronic alcohol use which may be related to synaptic changes of GABAergic signaling in individuals with Alcohol Use Disorder (AUD). Because $GABA_ARs$ play an integral role in alcohol pharmacodynamics and GABAergic signaling, AUD may lead to changes in their sensitivity to the endogenous GABA agonist. However, $GABA_ARs$ has not been electrophysiologically characterized in native human tissue of individuals with AUD. In this study, we used two electrode voltage clamp (TEVC) electrophysiology and microtransplantation synaptic membranes (MSM) methods to determine potential changes of $GABA_ARs$ affinity to GABA in AUD. We used postmortem dorsolateral prefrontal cortex (DLPFC) samples from 6 control subjects and 6 subjects diagnosed with AUD (mean age of 53 ± 7 years; post-mortem interval 27 ± 9 hours) that were collected by the University of Texas Health Science Center at Houston Brain Collection. Ion current responses values were integrated with label-free proteomics datasets from the same brain region. We built concentration-response curves for GABA to determine EC_{50} and pEC_{50} values for each control and AUD sample. Additionally, we compared protein expression to find proteins that significantly correlate with AUD diagnosis, EC_{50}, and pEC_{50} for further analysis in Gene Ontology Pathways. Our results directly measuring the activity of native human $GABA_ARs$ complexes found a trend of lower affinity for GABA in AUD compared to controls ($p = 0.072$, t-test) and no significant differences between ion current responses at any of the test GABA concentrations. These results suggest that GABA receptors in a subset of AUD subjects require higher GABA concentration for activation than those of controls. Furthermore, AUD samples demonstrate significant proteomic and transcriptomic differences when compared to control samples that may lend to structural differences and functionally compensatory changes to GABA receptors in AUD samples.

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Poster 20

Structure-Based Screening and Pharmacological Targeting Reveals a Binding Pocket in the Nav1.6/GSK3β Complex

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Background:
The voltage-gated Na+ channel Nav1.6 channel is abundantly expressed in neurons where it plays a pivotal role in action potential initiation and propagation. Research from our laboratory has identified a role of glycogen synthase kinase 3β (GSK3β) in governing Nav1.6 channel function. Intriguingly, we found that GSK3β's influence extends beyond phosphorylation of the channel's intracellular C-terminal domain (CTD), and involves a scaffolding role mediated by protein-protein interactions (PPI) between the Nav1.6 CTD and the kinase itself. However, the molecular determinants of this interaction and its potential independent modulation from kinase enzymatic activity are not well understood. In this study, we employed AlphaFold2 (AF2) to predict the structure of the Nav1.6/GSK3β complex. We aimed to characterize critical PPI sites and identify druggable binding pockets capable of modulating the Nav1.6/GSK3 complex without affecting kinase activity. This research holds promise for selectively targeting the Nav1.6/GSK3β complex with potential implications for therapeutic interventions in various central nervous system conditions.

Hypothesis/Goals:
Our hypothesis is that the Nav1.6/GSK3β complex has druggable binding pockets suitable for small molecule regulation independent of the kinase enzymatic activity.

Methods: We use in silico techniques, including AF2 structure predictions, virtual screening, molecular docking, and ADMET assessment, along with initial structural-activity relationship studies.

Results: Our molecular docking analysis revealed a significant pocket centered around GSK3β's axin-binding domain (ABD). Through an optimized structure-based screening targeting the ABD, we identified three top candidates (900-382, 302-560, 667-498) based on docking scores and SAR analysis. Notably, one candidate exhibited significantly higher predictions and promising characteristics positioning it as a promising lead molecule for modulation of the Nav1.6/GSK3β complex. This research may lead to selective neuromodulators with applicability for a wide array of neurodegenerative and neuropsychiatric disorders linked to GSK3β dysfunction.

Conclusions: These studies provided invaluable information on the importance of the Nav1.6/GSK3β modulators in disease that could guide new efforts in neurotherapeutics development.

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Poster 21

Longitudinal Blood Transcriptomic Changes in Hospitalized Mood Disorder Patients with Suicidal Ideation

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Suicide is a complex multifactorial event resulting from an interaction between biological and psychosocial factors. Identifying the molecular basis of suicidal ideation (SI) may provide targets for the development of novel treatment strategies and help better identify patients at risk of suicide. We aimed to longitudinally characterize the transcriptomic dynamics of peripheral blood mononuclear cells (PBMCs) in hospitalized patients with mood disorders admitted due to acute SI. We hypothesize that improvement of SI is associated with significant changes in blood gene expression and blood cell composition. We recruited forty-two patients with mood disorders, ages 18 or older, hospitalized with SI as a significant aspect of their presentations (Beck Scale for Suicidal Ideation (BSS) > 4). All subjects provided blood samples upon admission (T1) and immediately before discharge (T2). Bulk and single-cell RNA sequencing was performed at the two time points in N=15 and N=3 patients with significant improvement of SI (> 50% of reduction in BSS scores between T1 and T2), respectively, using Illumina 2x150bp sequencing and 10X Genomics Chromium™ 3' gene expression system. Paired analysis compared gene expression between T1 and T2 with correction for multiple comparisons. The levels of different blood cell types were estimated with transcriptomic data by cell-type deconvolution analysis. Twenty-six patients showed significant improvement of SI during hospitalization (mean (SD) BSS scores were 17.8 (8.31) at T1 and 1.0 (4.75) at T2). 'Non-improvers' had a mean (SD) BSS score of 21.3 (6.36) at T1 and 16 (7.75) at T2. No demographic or clinical variables at baseline were different between the two groups of patients. Three genes were differentially expressed between T1 and T2 (FDR=0.10) in patients that showed SI improvement, including *ZNF704* (logFC=1.70), *STMN1* (logFC=0.49), and *DDIT4* (logFC=0.67). Changes in BSS significantly correlated with changes in ZNF704 expression (r=0.597, p=0.019). The levels of B-cells and monocytes were significantly down-regulated at T2 compared to T1, while the levels of natural killer (NK) and T-cells were up-regulated ($p < 0.05$ for all). Specifically, the levels of the classical CD14+ monocytes were downregulated while the levels of the non-classical CD16+ monocytes were downregulated ($p < 0.05$) alongside SI improvement. SI symptom improvement could not be predicted by demographic and clinical variables at baseline, but was associated with significant blood transcriptomic changes. Symptom improvement was associated with major blood cell changes and specific changes in monocyte subtypes.

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Poster 22

Toll-Like Receptor 9 (TLR9) Inhibition Attenuates Chronic Stress-Induced Social Behavior Deficits

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Chronic stress is a major risk factor in the pathophysiology of many neuropsychiatric conditions. Toll-like receptors (TLRs) are pattern recognition receptors that are expressed by immune cells and play a critical role in stimulating innate immune system. Toll-like receptor 9 (TLR9), one of the nucleic acid-sensing TLRs, has been implicated in neuroinflammation. However, the role of TLR9 in chronic stress-induced behavioral deficits is not known. We investigated the potential of a TLR9 antagonist, ODN 2088 in attenuating chronic stress-induced social behavior deficits. **Methods** Male C57BL/6 mice (6 weeks old) were exposed to no stress (NS) or restraint stress (RS) for 21 days. ODN 2088 (i.p., 10 µg/mouse, Invivogen, San Diego, CA, US) or vehicle (saline) was administered every third day for 3 weeks. Following the stress procedure, the animals were tested using various behavioral tests. All the statistical analyses and graph preparation were performed using GraphPad Prism 9.0.0. $p < 0.05$ was considered significant. **Results**: NS-exposed mice spent more time in the chamber housing stranger mouse than the empty cage chamber in the social behavior test. However, RS-exposed mice showed no preference for either chamber indicating social behavior deficits. ODN 2088 treatment significantly attenuated RS-induced social behavior deficits (Time in chamber, Two-way ANOVA, chamber ($F(2,81) = 81.10$, $p < 0.0001$); ***$p < 0.001$ and ****$p < 0.0001$; $n = 10$ per group). We did not find any significant difference between the groups in any other behavioral tests. Our results demonstrate that TLR9 antagonism attenuates RS-induced social behavior deficits. **Conclusion**: Abnormalities in social cue identification, impaired social skills, and difficulties in maintaining social relationships are distinctive features of several psychiatric disorders including depression. In humans social behavior is predominantly controlled by the prefrontal cortex and our future studies will investigate the cellular pathways in the PFC mediating the neuroprotective effects of TLR9 antagonism on social behavior.

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Poster 23

Early-Life Exposure to Deltamethrin alters Brain Derived Extracellular Vesicle Content Leading to Disrupted Hippocampal Plasticity

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During development, the blood brain barrier is vulnerable to the lipophilic properties of Deltamethrin (DM) a type of commonly used pesticide. With the popularity of DM and other related pesticides in both agricultural and general public use, there is growing concern of links between developmental pyrethroid exposure and the incidence of neurodevelopmental disorders. Recent studies have linked pyrethroid exposure, to neurodevelopmental disorders including attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD). During development, DM exposure leads to neural circuit dysfunction, particularly in the hippocampus. A possible regulator of this dysfunction is brain derived extracellular vesicles (BDEVs), which are phospholipid nanovesicles that, based on their origin or size, include apoptotic bodies, micro vesicles, and exosomes. These molecules are involved in communication between cells and regulating the pathophysiology of several diseases. Studies have shown that exosomes can carry signaling information required to regulate neural circuit development, however their role in DM-induced neurotoxicity is yet to be explored. Here we hypothesized DM exposure during development alters the role of the biogenesis of BDEVs, which may lead to dysfunctional synaptic plasticity in the hippocampus.

Using a developmental exposure model, pregnant dams were exposed to 3 mg/kg/72 hours, equivalent to 1mg/kg/day, DM or vehicle through pregnancy and lactation. At post-natal day 30 BDEVs were extracted from both control and DM-exposed males and analyzed by transmission electron microscopy (TEM) to determine size and validated by western blot analysis. BDEVs were then labelled with a green, fluorescent membrane marker which was used to perform intracerebroventricular (ICV) stereotaxic injections in male WT mice that either got control or DM exosomes. After allowing 36 hours for distribution the brains were collected and sliced. Long term potentiation studies were performed by doing field excitatory post synaptic potential (fEPSP) from the striatum radiatum (CA1) of the hippocampus by stimulating Schaffer collaterals. Basal synaptic transmission was analyzed, and a stable baseline was obtained, which was followed by inducing long-term potentiation (LTP).

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Poster 24

Epigenetic GrimAge Acceleration and Cognitive Impairment in Bipolar Disorder

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Bipolar disorder (BD) has been previously associated with clinical signs of premature aging, including accelerated epigenetic aging in blood and brain, and a steeper age-related decline in cognitive function. However, the clinical drivers and cognitive correlates of epigenetic aging in BD are still unknown. We aimed to investigate the relationship between multiple measures of epigenetic aging acceleration with clinical, functioning, and cognitive outcomes in patients with BD and controls. Blood genome-wide DNA methylation levels were measured in BD patients (n = 153) and matched healthy controls (n = 50) with the Infinium MethylationEPIC BeadChip (Illumina). Epigenetic age estimates were calculated using an online tool, including the recently developed lifespan predictor GrimAge, and analyzed with generalized linear models controlling for demographic variables and blood cell proportions. BD was significantly associated with greater GrimAge acceleration (AgeAccelGrim, β=0.197, p = 0.009), and significant group-dependent interactions were found between AgeAccelGrim and blood cell proportions (CD4+ T-lymphocytes, monocytes, granulocytes, and B-cells). Within patients, higher AgeAccelGrim was associated with worse cognitive function in multiple domains (short-term affective memory (β=-0.078, p = 0.030), short-term non-affective memory (β=-0.088, p = 0.018), inhibition (β=0.064, p = 0.046), and problem-solving (β=-0.067, p = 0.034)), age of first diagnosis with any mood disorder (β=-0.076, p = 0.039) or BD (β=-0.102, p = 0.016), as well as with current non-smoking status (β=-0.392, p < 0.001). Overall, our findings support the contribution of epigenetic factors to the aging-related cognitive decline and premature mortality reported in BD patients, with an important driving effect of smoking in this population.

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Poster 43

The Psychiatry of Cluster Headache: A Systematic Review and Meta-Analysis of Psychiatric Disorders and Cluster Headache

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Sponsored by: Dr. Antonio Teixeira
Supported by: Dr. Lijin Jose, Dr. Thiago Macedo e Cordeiro
Key Words: Cluster Headache, Depression, Anxiety, Psychiatric

Cluster headaches (CH) are a group of primary headache disorders lasting from 15 and 180 minutes located at the periorbital, supraorbital, and temporal regions with autonomic symptoms like rhinorrhea and lacrimation. They typically start between the ages of 20 and 40 year and have a prevalence of approximately 1 in 1000 of the population worldwide. Despite the chronic nature of the condition, the severity of the episodes and the related socioeconomic impact, the prevalence of psychiatric disorders in people with CH has not been well documented.

An electronic systematic review was done using the following databases: PubMed, PsycINFO, and Embase. There were 761 duplicates removed with a total of 1,208 records needing to be screened for title and abstract review. A total of 1,190 records were excluded after title and abstract review based on lack of fulfillment of inclusion and exclusion criteria. In the end, 18 articles were assessed for eligibility based on a full-text review. Of these 18, 11 articles were excluded. After a reference review, 2 articles were included in the study, leaving a total of 9 articles used in the review and meta-analysis. A random effects model was used in our meta-analysis due to the high heterogeneity in between studies and conducted using R. Quality of each study was assessed using the Joanna Briggs Institute's (JBI) critical appraisal tool.

To evaluate prevalence of depression in CH, seven studies were used. The weighted prevalence of CH and depression found was 25%. To evaluate prevalence of anxiety in CH, six studies were used. The weighted prevalence of CH and anxiety found was 18%. To evaluate risk of depression in CH and healthy controls, six studies were used. Each study showed a statistically significant increase in risk of depression in CH patients. The odds ratio of developing depression was 4.10 and statistically significant. To evaluate risk of anxiety in CH and healthy controls, four studies were used. Two of the four studies found statistically significant increased risk in developing anxiety. The odds ratio of developing anxiety was 2.85 and statistically significant.

Poster 25

Sex Differences in the Behavioral Effects of Fentanyl

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Aim. Fentanyl (FEN) is a synthetic opioid and highly effective mu-opioid receptor agonist. Additionally, FEN contributes to opioid use disorder (OUD) and overdose which continues to harm the U.S. population. Previously, sex differences in behavioral responses to morphine (MOR) have been reported. Yet, sex differences in the effects of FEN are not well understood. We now examine whether there are sex differences in behavioral responses to FEN. Findings would provide evidence of uniquely altered responses to FEN in females compared to males. **Methods.** Separate sets of male and female Sprague-Dawley rats were tested for: 1) locomotor activity in an open field (30-m) after FEN administration (0.1 mg/kg; SC), 2) schedule-controlled responding under a fixed-ratio 15 (FR15) schedule of food pellet delivery (30-m) 30-m after administration of several doses of FEN (0.003-0.1 mg/kg) and MOR (0.3-10 mg/kg), 3) self-administration of FEN solution (0.003-0.05 mg/kg/ml) delivery (30-m) under an FR3 schedule. **Results.** No sex differences were found in distance traveled during the locomotor activity assessment. Schedule-controlled responding rates decreased across FEN doses but there were no sex differences despite females showing higher response rates at baseline. During maintenance of self-administration, active lever presses and dipper presentations showed significant Sex X Dose effects (Ps<0.05) and a trend in Sex X Dose effects for inactive lever presses. **Conclusions.** FEN did not alter activity effects differentially between sexes. These results are similar to previous findings in MOR behavioral responses in rats. Further analysis of the lever pressing and drug seeking during extinction and reinstatement is ongoing to assess whether there are sex differences in persistence of responding for FEN.

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Poster 26

The Impact of Probiotics on Treating Autism Spectrum Disorder

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Autism Spectrum Disorder (ASD) is a developmental and intellectual disability that affects social interaction and communication. It can cause repetitive behavior, sensory anomalies and limited interests, gastrointestinal symptoms, along with other symptoms. The cause of ASD remains unknown. It has been suggested that genetics plays a big role, along with epigenetic factors that can contribute to the development of this disorder. Recently it has come to light that there are important differences in the microbiota composition of individuals with ASD in comparison to neurotypical controls. These changes produce differences in metabolite profiles, intestinal hyperpermeability and gastrointestinal symptoms, all of which contribute to the physiopathology of ASD. Probiotics, which are beneficial microorganisms that support gut health, have garnered attention for their potential impact on various aspects of human well-being, including neurological and psychological conditions such as ASD. The investigation into the effects of probiotics on the treatment of ASD has unveiled a promising avenue in the field of therapeutic interventions. The objective of this systematic review is to determine the impact of probiotics on individuals with ASD. We have used PubMed and Google Scholar to identify observational studies published before August 21st, 2023, including clinical case studies, clinical trials, or preclinical studies. The Preferred Reporting Items for Systematic Reviewers and Meta-analysis (PRISMA) reporting guideline was followed. The literature search yielded 15300 publications, of which 16 studies met the inclusion criteria, ASD patients were given probiotic supplementation were included. Across the majority of the studies analyzed, the results consistently pointed to significant improvements in gastrointestinal symptoms, a decrease in repetitive behaviors, improved communication and social skills, and overall higher test scores due to probiotic intervention. It is important to note that the studies we analyzed had certain limitations, including disparities based on age groups, diversity on the specific probiotic strains administered, intervention duration, and various other contributing factors. While research in this area is still relatively young and the exact mechanisms are not fully elucidated, preliminary findings have shown promise. In conclusion, the exploration of probiotics as a potential treatment for ASD holds significant promise, given the emerging understanding of the gut-brain connection and the role of gut microbiota in neurological conditions. While preliminary findings are encouraging, further research is essential to determine the specific strains, dosages, and mechanisms through which probiotics could effectively mitigate ASD symptoms and improve the quality of life for individuals on the spectrum.

Poster 27

The Neural Basis of Apathy in Parkinson's Disease: A Comparative Analysis of Brain Structure in PD Patients and Healthy Controls.

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Background: Apathy is one of the most important neuropsychiatric symptoms in patients with PD that is present in up to 70% of patients. The study of the neural basis of apathy is usually focused on structures involved in instrumental behavior, i.e. accumbens area, ventromedial prefrontal cortex (vmPFC), caudate nucleus, ventral tegmental area, substantia nigra, and amygdala. Research on apathy across various diseases suggests that disease-specific mechanisms involving additional brain structures may significantly contribute to apathy development. Methods: T1-weighted MRIs from 28 PD patients and 25 healthy controls (HC) were processed using FreeSurfer, extracting cortical surfaces and subcortical normalized volumes. For each group, these structural measures were combined with clinical and demographic variables: age, sex, Mini-Mental State Examination (MMSE), and Apathy Scale (AS), resulting in two data sets with identical variables. We built one model for each group using the AS score as the dependent variable. Using a leave-one-out cross-validation strategy, we trained Elastic Net models for each alpha value in the 0.05 to 0.95 range, selecting the one with the smallest cross-validation error per iteration. By averaging predictor coefficients across all models, we identified the mean coefficient for each predictor in both groups.

Findings: AS scores were associated with atrophy of brain structures in both PD and HC groups. Atrophy in brain structures like the corpus callosum were common among the groups. AS scores were associated with regions traditionally involved with instrumental behavior, i.e. accumbens area and caudate nucleus. AS scores in the PD group were associated with regions that are not traditionally involved with instrumental behavior, i.e. temporal cortex. The regions of interested associated with apathy in the PD group were predominantly cortical.

Discussion: Apathy is a transdiagnostic construct that is present in most neurodegenerative diseases and also psychiatric disorders (i.e. schizophrenia). The understanding of the neural basis of apathy can benefit patients across different disease settings. This study suggests that the neural mechanisms of apathy in PD may be related to specific components of PD's pathophysiology. Although PD is primarily associated with neurodegeneration in subcortical regions, e.g. substantia nigra pars compacta, the results from this study indicate cortical atrophy may play a bigger role in the development of PD- related apathy. Atrophy in the temporal lobe is consistent with the natural progression of PD according to the Braak stages.

Conclusion: While AS scores were linked to brain regions common to both PD patients and healthy individuals, certain structures were unique to those with PD. PD-related apathy was predominantly associated with cortical atrophy, especially in the temporal cortex.

Poster 28

Frustration-Mediated Demotivation for Cocaine in Nucleus Accumbens Shell GPR12 Knockdown Rats

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The orphan G-protein-coupled receptor (GPCR) GPR12 is a highly understudied protein, but one with tremendous therapeutic potential for neuropsychiatric conditions. Enhanced regional expression of GPR12 in the nucleus accumbens shell (shNAc) suggests highly specific function, however, research exploring the role of GPR12 in regulating neuronal activity and motivation-related behaviors is extremely limited. Similarly, pharmaceutical targeting of GPR12 is scarce since the mechanisms for its high constitutive activity and activation by endogenous and synthetic ligands remain unclear. Nonetheless, reports have demonstrated an involvement of GPR12 in regulating brain function via its high constitutive activity. Here, a multi-omics approach led us to identify GPR12 as a target of interest in the context of substance use disorders (SUDs). Firstly, *in vivo* manipulation of GPR12 expression in the shNAc resulted in increased frustration-related behavior and decreased motivation for cocaine compared to controls during operant tasks. Moreover, patch-clamp recordings revealed that shNAc knockdown of GPR12 resulted in decreased medium spiny neuron (MSN) intrinsic excitability, which has been associated with a protective behavioral phenotype in drug-taking in previous studies. Overall, our findings provide insights into the role of Gpr12 in regulating molecular signaling and neuronal excitability, connects these functions in the shNAc to a phenotype of frustration-mediated demotivation for cocaine taking, and collectively highlights its promising value as a tractable drug target for SUDs.

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Poster 30

Elucidating the Role of Ankyrins during Synapse Formation in the Outer Retina

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Defects in neural circuit formation and synaptogenesis have been implicated in many psychiatric disorders, and yet the developmental etiology that leads to aberrant circuitry remains poorly understood. Specific scaffolding proteins, Ankyrins, are implicated in different psychiatric disorders such as bipolar disorder and schizophrenia. Elucidating the mechanism behind Ankyrin mutations and synaptic connectivity defects in the mammalian brain poses a challenge due to the billions of neurons and synaptic connections. A more appealing model is the mammalian retina, where different types of photoreceptors synapse selectively to distinct postsynaptic targets: horizontal cells and bipolar neurons. During development, photoreceptors first make contacts to horizontal cells (referred to as first synaptic contact) and then to bipolar neurons (i.e. second synaptic contact). Although the timing and patterns of connections have been well-described for photoreceptors, little is known about the early molecular events that coordinate the selective wiring of photoreceptors to their respective targets. Here, we investigate a new role for the cytoskeletal scaffolding proteins, Ankyrins in mediating the early developmental events involved in photoreceptor connectivity. We found Ankyrin-B (AnkB) and Ankyrin-G (AnkG) to be differentially expressed in both a spatial and temporal manner in the developing retina. AnkB is highly expressed in horizontal cells at early time points when the first synaptic connection between photoreceptors and horizontal cells is being established, whereas AnkG is expressed at later stages in bipolar neurons when the second synaptic connection is formed. Moreover, our initial data reveals that loss of AnkB and AnkG results in phenotypes consistent with synaptic connectivity defects between photoreceptors and their synaptic targets. Additionally, we also found impaired retinal responses in animals with disruption of AnkB and AnkG compared to controls. Taken together, we uncovered a new role for Ankyrins in wiring photoreceptors to their postsynaptic partners during development. Future work will focus on disseminating the developmental mechanisms as well as the cell-type specific requirements of Ankyrins in photoreceptor connectivity. This work will have broad significance as it may reveal new potential targets that can be used to elucidate the mechanism behind Ankyrin proteins and neuropsychiatric disorders.

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Poster 31

Interacting Immediate and Long-Term Action Regulation with History of Medically Severe Suicide Attempt

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Suicidal ideation is variable and nonspecific, and the stakes are high for prediction of acute risk as most suicides occur on the first attempt. Additionally, survival of medically severe suicide attempts can have long-lasting medical and psychiatric consequences, including subsequent suicide or other premature mortality. Interactions between long-term mechanisms (latent susceptibility) and short-term mechanisms (acute crises) in immediate behavior dysregulation may underlie suicide risk. A group of 28 individuals with recent medically severe suicide attempts, serving as a proxy for patients at risk of suicide, were compared to individuals with similar demographic and diagnostic characteristics including a history of suicidal ideation, but no history of a suicide attempt. Both groups completed the Columbia Suicide Severity and Risk Scale (C-SSRS) and the Beck Scale for Suicidal Ideation-Current (SSI-C) and Worst (SSI-W). Psychiatric symptoms were assessed through the clinician-administered Schedule for Affective Disorders and Schizophrenia (SADS-C) and the self-rated Internal State Scale (ISS). The immediate memory task (IMT) was used to measure impulsivity. Bivariate comparisons used t-tests and Cohen's D effect sizes. Pearson's R measured correlations between clinical/behavioral variables and SSI-W. Path analysis determined whether effects of variables on suicide risk were mediated by, or independent of, SSI-W. Bayesian analysis with non-informative priors found that lifetime aggressive behavior, current depressive symptoms and alcohol use indirectly contributed to a higher SSI-W, while lower IMT response discriminability indirectly led to a higher SSI-W. Variables with effects on MSSA independent of SSI-W included IMT bias, current ISS activation, and lifetime cumulative adversity. Minimization or denial of childhood trauma increased MSSA independently of SSI-W, but also correlated negatively with SSI-W, indirectly reducing MSSA. Subjects with MSSA had increased cumulative stress. Bayesian modeling suggested that this was necessary but not sufficient for suicide risk; cumulative stress is associated with MSSA, but actual suicidal behavior was facilitated by the combination of cumulative stress with behavioral dysregulation. These results suggest a possible interaction between sensitization and behavior regulation with regard to medically severe suicide attempts. Future studies are needed to further characterize the dynamic between sensitization and behavioral regulation with the goal of preventative treatment for suicide risk, regardless of attempt history.

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Poster 32

Prefrontal Cortex Neuronal Activity Reflects Individual Differences in risk-Taking Behavior Following Morphine Conditioning in Rats

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Opioid use disorder is associated with impaired risk-related decision-making. However, it is unclear how repeated opioid exposure affects the brain to alter risk taking under motivational conflict. In a modified conditioned place preference protocol, rats injected with either saline or morphine were exposed to the side of the apparatus preferred least at baseline. Conditioning occurred over 10 alternating days (5 pairings in each side). Two days after conditioning, rats underwent a preference test immediately followed by a conflict test in which an aversive stimulus (cat saliva) was introduced in the side of the chamber previously paired with morphine injections. In the preference test, morphine-treated rats spent more time in the drug-paired side of the apparatus than did saline-treated rats. In the conflict test, saline group rats avoided the side of the apparatus containing cat odor. In contrast, rats in the morphine group continued to prefer the previously drug-paired side despite the presence of cat odor, demonstrating increased risk-taking behavior. K-means clustering uncovered two subsets of morphine-treated rats that exhibited either: *i)* enhanced place preference and persistent drug seeking during conflict (*risk-takers, RT*), or *ii)* moderate place preference and suppressed drug seeking during conflict (*risk-avoiders, RA*). Single-unit recordings from neurons in the prelimbic (PL) cortex, a region involved in decision-making and strategy shifting, revealed decreased firing rates upon acute morphine exposure. In contrast, on the final drug conditioning day, morphine failed to suppress neuronal firing rates, suggesting that PL neurons undergo adaptation to repeated morphine exposure. Recordings during the preference test identified PL neurons that exhibited significantly higher or smaller firing rates when animals were in either the paired or neutral side of the apparatus. Further analysis uncovered distinct populations of PL neurons that were either excited or inhibited when rats entered the drug-paired side of the apparatus. Interestingly, while cells inhibited during paired side entries in the preference test showed no response to paired side entries during the conflict test in saline-treated and RA rats, these inhibitory entry responses persisted in RT rats. Additionally, RTs showed a greater proportion of paired side entry-responsive neurons that altered their response type between the preference and conflict tests. Taken together, our results suggest that a loss of PL inhibition after opioid conditioning is associated with the formation of contextual reward memory. Furthermore, persistent inhibitory signaling and enhanced spatial remapping of the drug-associated context in PL during conflict may underlie increased risk taking following opioid exposure.

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Poster 33

Peptidomimetics Modulates the Scaffolding Function of GSK3β in the Nav1.6 Macromolecular Complex

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Signaling pathways regulating ion channel macromolecular complexes play a crucial role in fine-tuning neuronal activity and remodeling synapses. Yet, the molecular underpinnings of activity-dependent signaling mechanisms regulating these molecular complexes are still poorly understood. In recent studies conducted in the nucleus accumbens (NAc), we have shown that vulnerability to depression-like and stress-induced disorders induces a form of maladaptive plasticity consisting of hyperexcitability of medium spiny neurons (MSNs). Vulnerability in these cells is mediated by the increased interaction between glycogen synthase kinase 3β (GSK3β) and the C-terminal domain (CTD) of the voltage-gated Na+ channel Nav1.6. A decoy peptide mimicking the Nav1.6 segment interacting with the kinase or *in vivo* genetic silencing of GSK3β was found to be sufficient to prevent MSN maladaptive plasticity. Building on these results, we hypothesized that inhibition of the GSK3β/Nav1.6 protein-protein interaction complex could counteract the GSK3β-dependent maladaptive plasticity of MSNs. To that end, we developed a series of peptide-based probes capable of modulating the scaffolding function of GSK3β in the Nav1.6 channel complex. Molecular docking, split-luciferase complementation (LCA), intrinsic fluorescence, surface plasmon resonance (SPR), whole-cell patch-clamp electrophysiology in heterologous cells, and in the *ex-vivo* acute slice preparation as well as viral vector-based in vivo gene silencing were implemented for discovery and validation studies. Probe ZL141 was found to significantly inhibit GSK3β/Nav1.6 complex formation using the LCA and to bind to GSK3β using SPR. In addition, whole-cell patch-clamp recordings in HEK293 cells stably expressing Nav1.6 showed that ZL141 regulates peak current density, voltage-dependent activation, and steady-state inactivation curves as well as long-term inactivation of Nav1.6 in a GSK3β-dependent manner. Further studies for GSK3β *in vivo* genetic silencing and *ex vivo* slice recordings of MSN in the NAc support the mechanism of action of ZL141 observed in heterologous cells. These studies lay the groundwork for the development of novel neurotherapeutics based on the modulation of the non-enzymatic activity of GSK3β in the brain.

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Poster 34

Effects of Morphine Treatment on VEGF and Wound Healing in Opioid Use Disorder

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Opioids such as morphine are highly effective and addictive analgesics that are prescribed both for the management of acute pain in a hospital setting, as well as for chronic pain in an outpatient setting. Studies suggest that opioids disrupt wound healing through a variety of mechanisms, including suppression of the inflammatory response, reducing angiogenesis, and blocking recruitment of myofibroblasts. Fibroblasts play a key role in wound healing through production of collagen and facilitation of angiogenesis. One of the key growth factors mediating these processes is vascular endothelial growth factor (VEGF). In this study, we aimed to assess the effects of morphine treatment on wound healing in postmortem fibroblasts taken from a patient with opioid use disorder (OUD), using both an acute and chronic treatment protocol. Cell lines were treated with different concentrations of morphine for 24 hours in the acute arm and 168 hours in the chronic arm of the experiment. Levels of VEGF were measured by qPCR after treatment, and a scratch test was performed to directly measure the rate of wound healing. We hypothesized that cells treated with morphine would show reduced rates of wound healing, and that the impact of chronic treatment would be greater than the acute treatment. We found that while acute exposure to morphine had no visible effect on VEGF levels , chronic treatment produced a decrease in VEGF at low and medium doses , and increased levels at the higher dose. experiments using additional subjects to allow for statistical analysis. Future studies will also measure global gene expression through RNA sequencing to assess the effects of chronic morphine treatment on genes driving wound healing in patients with OUD. This study will lead to a better understanding of how opioids affect wound healing and will provide insight into the mechanisms underlying this process.

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Poster 36

Malignant Catatonia in a Patient After Stem Cell Transplant and its Complexities: Report from a Single Patient Encounter.

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Background: Catatonia in the medically ill is a potentially fatal comorbidity. As it is historically associated with psychiatric psychosis, catatonia is often missed in the medical setting (Oldham et al 2015). There are few cases of reported catatonia in cancer patients and typically they are seen in those with central nervous system disease (Smith et al 2012). For cancer treatments with high medical complexity and high rates of delirium, like during hematopoietic stem cell transplantation (SCT), reports of catatonia might be expected, but remain rare. The mainstay of treatment for catatonia is benzodiazepines and in severe cases electroconvulsive therapy (ECT). Here we aim to showcase the complexity of medical-psychiatric care in this patient population.

Case: A 66yo female with history of bipolar II disorder and myelofibrosis treated with matched unrelated donor stem cell transplant complicated by acute graft versus host disease was diagnosed with malignant catatonia after presenting with altered mental status 84 days post-transplant. Brain MRI revealed no significant findings. The patient had a long standing history of bipolar II stable on medications Development of malignant catatonia was likely multifactorial as the patient had no history of catatonia and was undergoing treatment for graft versus host disease (GVHD) with mediations that have been known to have neuropsychiatric side effects (tacrolimus and high-dose steroids). We utilized a high dose lorazepam drip resulting in need for intubation to protect patient airway. She was transferred to intensive care unit (ICU) on the 23rd day of hospitalization and remained under ICU care for 18 days for intubation and close monitoring of vitals. Meanwhile we planned to transfer the patient for ECT. Following stabilization of her vital signs on Lorazepam 0.25mg per hour drip she was eventually extubated. Lorazepam drip was tapered, and the patient was started on Amantadine via nasogastric (NG) tube as adjunct treatment. She eventually achieved full resolution of her catatonia. The length of hospitalization totaled 140 days. ECT was not utilized due to medical and situational barriers in arranging care.

Discussion: Since malignant catatonia has increased risk of morbidity and mortality, it is crucial to identify it early. Hence, including it in the differential for delirium is important regardless of prior psychiatric history. Our case highlights the value of close collaboration between medical and liaison psychiatry teams to help with possible early identification and various aspects of treatment planning including ECT.

Poster 37

Maternal Opioid Exposure Alters Murine Neurodevelopment

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More than 932,000 people have died since 1999-2020 from a drug overdose and more than 564,000 of those overdose deaths involved an opioid, synthetic or natural. In 2020 alone over 68,000 people died from opioid overdose, which is 8.5 times more than in 1999. From 2010 – 2017 there has been a 131% increase in maternal opioid related diagnoses. Methadone has been the mainstay in care for treatment of opioid use disorder (OUD), but since 2002 buprenorphine has been an approved medication for opioid use disorder (MOUD) and has become more accessible for treatment than methadone in recent years. Since buprenorphine is relatively new there have been no long-term studies on the effects of buprenorphine treatment on offspring when used during pregnancy to treat OUD. A recent rodent study by our team showed that maternal buprenorphine exposure altered neural progenitor cell proliferation and corticogenesis. In this study the model of MOUD developed in the Schlagal study was used to investigate the outcomes of offspring. An earlier embryological time point compared to the Schlagal study was investigated using immunohistochemistry. ImageJ and Imaris were used to quantify cell count and alterations in cortical zones and different brain regions. Opioid and buprenorphine exposure during pregnancy altered proliferation and cortical development compared to control. These experiments demonstrate the need to further investigate the effects of buprenorphine on offspring with the aim of achieving better outcomes for mothers and offspring.

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Poster 38

Microglial Type I Interferon Signaling Mediates Chronic Stress-Induced Social Behavior Deficits

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Major depression is a chronic illness, well characterized by a lack of energy, sadness, low mood, insomnia, and ultimately inability to enjoy life. Chronic stress is a major risk factor for developing depression. Chronic stress conditions induce neuroinflammation and synapse loss. However, the mechanism mediating chronic stress-induced neuroinflammation and synapse loss is poorly understood. Our previous findings from animal studies demonstrated microglial activation and type I interferon (IFN-I)-associated behavior deficits under chronic stress conditions. Microglia, the immune cells in the brain play crucial roles in inflammation and synapse pruning. Here we aimed to explore microglial IFN-I signaling as a link between chronic stress-mediated increased neuroinflammation and synapse loss. 6 weeks old male and female mice with IFN-I receptor deleted in microglia and their age-matched wild-type controls were subjected to 21 days of chronic unpredictable stress (CUS). Social interaction and Y-maze tests were performed to assess social behavior and cognitive functions, respectively. Microglial activation, neuroinflammation and synapse density were analyzed by flow cytometry and immunostaining in the prefrontal cortex and hippocampus, brain regions implicated in mood and behavior. Three-way ANOVA (sex X genotype X stress), followed by Bonferroni post hoc tests, were performed using GraphPad Prism 9.1.1. IFN-I receptor deletion in microglia significantly attenuated CUS-induced increases in neuroinflammation, synapse loss and social behavior deficits in mice. IFN-I is linked to neuropsychiatric disorders in various preclinical and clinical contexts. The present study identified a crucial role of microglial IFN-I signaling in compromising synapses and causing behavior impairment under chronic stress conditions. Targeting microglial IFN-I pathway represents a promising therapeutic option, especially for patients with an elevated immune profile, as seen in many depressed subjects.

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Poster 39

The Relationship Between ADHD-like Inattention and Anxiety During Treatment of Youth with Anxiety.

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The prevalence of attention-deficit hyperactivity disorder (ADHD) among individuals aged 3-17 ranges from 5% to 20% (Bitsko et al., 2019; Salari, et al., 2023; Zalsman & Shilton, 2016). The clinical profile for ADHD exhibits many symptoms that overlap with anxiety (e.g., restlessness, irritability, and concentration problems), often leading to a complex presentation at risk for misdiagnosis (American Psychiatric Association [APA], 2022). The presence of comorbidity with ADHD is common, including 25-50% of individuals with anxiety disorders (Koyuncu et al., 2022; Pallanti & Salerno, 2020). The comorbid condition is associated with more attentional problems and lower levels of social competence than either ADHD or anxiety alone (Celebi & Unal, 2021; Koyuncu et al., 2015). Whether diagnosed ADHD in youth with anxiety disorders represents true underlying ADHD pathology remains unclear. Accordingly, the present study aims to fill this gap. If anxiety truly contributes to ADHD-like inattention and hyperactivity/impulsivity, these symptoms should be expected to decrease following parent-based cognitive behavioral therapy (CBT) for anxiety. The first aim of the study assesses whether attention problems decrease following anxiety treatment. The second study aim is to evaluate whether baseline ADHD symptoms predict treatment outcome over time, measuring ADHD symptoms as a continuous variable. Data were collected from 62 youth with clinically significant anxiety and OCD between the ages of 7 to 17 years and their parents following parent-based CBT treatment. Each youth participant completed the clinician-administered *Pediatric Anxiety Rating Scale* (PARS), which assesses the frequency, severity, and associated impairment of anxiety symptoms. The *Swanson, Nolan, and Pelham—IV* (SNAP-IV) was completed by each parent, assessing the core symptoms of ADHD (inattention and impulsivity) of their children. At baseline, clinician-rated PARS was significantly positively correlated with both parent report of SNAP-IV inattention (r = .30, p<.01) and SNAP-IV impulsivity (r=.23, p<.05). Regarding post-treatment symptom severity, there were correlations among clinician-rated PARS and parent-rated SNAP-IV inattention (r=.31, p<.01) and clinician-rated PARS and parent-rated SNAP-IV impulsivity (r=.28, p<.01). A series of hierarchical simultaneous multiple regression analyses revealed that change in anxiety at the end of treatment was found to significantly predict ADHD-like inattention over time, where higher change scores predicted lower inattention severity (b = -.18, p = .05). This change was not significantly associated with ADHD-impulsivity. Regarding baseline ADHD symptoms as a predictor, the interaction between time and treatment group were non-significant. These results indicate that youth who exhibit clinically significant anxiety and ADHD-like inattention may see substantial improvements in their attention problems following treatment for anxiety. Consequently, clinical anxiety and inattentiveness may be closely linked to anxious feelings among youth. Clinicians may consider prioritizing anxiety issues that are faced before targeting inattentive-type ADHD.

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Poster 40

Glutamatergic Cerebellar Neurons Differentially Contribute to the Acquisition of Motor and Social Behaviors

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Insults to the developing cerebellum can cause motor, language, and social deficits. Here, we investigate whether developmental insults to different cerebellar neurons constrain the ability to acquire cerebellar-dependent behaviors. We perturb cerebellar cortical or nuclei neuron function by eliminating glutamatergic neurotransmission during development, and then we measure motor and social behaviors in early postnatal and adult mice. Altering cortical and nuclei neurons impacts postnatal motor control and social vocalizations. Normalizing neurotransmission in cortical neurons but not nuclei neurons restores social behaviors while the motor deficits remain impaired in adults. In contrast, manipulating only a subset of nuclei neurons leaves social behaviors intact but leads to early motor deficits that are restored by adulthood. Our data uncover that glutamatergic neurotransmission from cerebellar cortical and nuclei neurons differentially control the acquisition of motor and social behaviors, and that the brain can compensate for some but not all perturbations to the developing cerebellum.

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Poster 41

Elucidating the role of BetaII-spectrin in Dendritic Tiling

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Formation of precise neural circuits is essential for the functioning of the nervous system with defects linked to several neurodevelopment and psychiatric disorders. During development, newborn neurons integrate into existing neural circuits by developing dendritic branches. These dendritic branches are highly dynamic and adopt complex and diverse morphologies, influencing the function of these neurons. One mechanism responsible for this dendritic diversity is self-avoidance and tiling. In self avoidance, dendrites of the same neuron avoid one another and in tiling, dendrites from different neurons avoid each other. Such spacing mechanisms ensure that arbors maximize their spread across a territory while minimizing the redundancy with which the territory is innervated. Molecular studies have shown that cell adhesion molecules like Dscams and protocadherins prevent the overlapping of dendrites and act as mediators of self-avoidance and tiling. Loss of these proteins leads to dendritic outgrowth and severe overlapping of the arbors. Although several key cell adhesion molecules have been identified for their role in tiling, no cytoskeletal proteins have been studied. Spectrins are important cytoskeletal scaffolding proteins in the neurons required for anchoring membrane proteins and for forming and maintaining synaptic connections. Loss of spectrins has been linked to diseases like epilepsy, ADHD, autism spectrum disorders and several others. The complexity of neural circuits and lack of tools to study the neuron subtypes make the brain a difficult model system for research. The mammalian outer retina, on the other hand, is an excellent model to study the role of spectrins in early development and neural circuit formation. Retina neurons connect in precise synaptic layers and share general neuron and synapse feature with the brain, with a plethora of tools available to study neural circuit formation during development. The bipolar cells, consisting of 15 subtypes are housed in the outer retina and form a unique link connecting the inner and outer layers of the retina. Therefore, understanding the bipolar cell plasticity is an important objective for approaches aimed at neuronal regeneration and repair. Recently, it has been shown that loss of betaII-spectrin results in impaired synapse formation by the bipolar cells leading to lamination defects in the outer retina. Our experiments with the betaII-spectrin KO mice have shown dendritic sprouting in some rod bipolar cells. Further experiments are needed to show abnormal tiling defects in these mice. Using modern tools like reporter transgenic mouse lines that label a specific bipolar cell type and technologies like single cell sequencing, we plan to elucidate the role of betaII-spectrin in dendritic tiling. Understanding the role of these scaffolding proteins will shed light on the importance of the dendritic arbors in normal brain function and the consequences of altered dendritic branching in disease states and cognitive disorders.

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Poster 42

Feasibility of a Combined Electroencephalogram and Transcranial Magnetic Stimulation Protocol in Individuals with Cocaine Use Disorder

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There is an urgent need to identify novel treatments for cocaine use disorder (CUD), as pharmacological treatments have largely failed. Brain stimulation via transcranial magnetic stimulation (TMS) is a promising avenue with addressable issues for optimization. Identifying novel stimulation sites and neurobiological targets, as well as addressing feasibility and tolerability is essential in this clinical population. The current proof-of-concept pilot study assessed the feasibility of an acute TMS protocol (intermittent theta burst stimulation, 2x, 15 minutes apart) in 5 individuals with current CUD. We also assessed the preliminary effects of TMS to a novel site, the dorsomedial prefrontal cortex (compared to sham), on electroencephalogram (EEG) markers of reward functioning. We used a within-subjects crossover design where each participant completed self-report, safety, and EEG measures before and after active or sham stimulation (80% rMT) on separate days. Ten participants were screened and five participants qualified. All qualifying participants completed both TMS days and all assessments. All participants found the TMS to be tolerable. One participant asked for the intensity to be lowered. Two participants had a lingering headache that was alleviated by over the counter medication. Differences in reward processing were observed pre-post active TMS but not sham TMS. Specially, brain reactivity to losses was enhanced following TMS. Future directions including building upon the pilot design by adding a 3rd arm (comparing dorsomedial prefrontal cortex to traditional dorsolateral prefrontal cortex) and increasing the sample size. This work will contribute to our increasing knowledge of TMS for the treatment of CUD and other substance use disorders.

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