TEXAS A&M UNIVERSITY

LIFE SCIENCES GRADUATE RECRUITMENT SYMPOSIUM

FEBRUARY 1-2, 2024

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HOWDY!

1 Welcome to the Texas A&M University Life Sciences Network

At Texas A&M University, six graduate programs in the Life Sciences have come together to form a network to facilitate cross-disciplinary collaborations, join their outreach and recruitment efforts, build a graduate student community, and bring diversity to the institution.
The Texas A&M Life Sciences Network (LSN) represents the premier Ph.D. programs in the life sciences that collaborate on recruiting, orientation, programming, and graduate student support. These programs provide exceptional opportunities to pursue a Ph.D. degree across a wide breadth of life science disciplines with some of the most stellar faculty and research programs on campus.

2 Prospective Graduate Students

Why Choose Texas A&M?
At Texas A&M University, graduate and professional students learn from faculty members who are respected experts at the top of their fields. They work together in state-of-the-art facilities to solve pressing global challenges. Through practical learning experiences, students gain the skills and knowledge needed to excel professionally in their chosen field.

3 Contact

lifesciences.tamu.edu

lifesciences@ag.tamu.edu

Schedule of Events

February 1st

Welcome Breakfast and Reveille Meet & Greet — 7:45- 8:30 am
Candidates only
Texas A&M Hotel and Conference Center Ballroom

Keynote Speech by Dr. Fuhui Tong — 8:30- 9:00 am
Interim Associate Provost & Dean of the Graduate & Professional School. *Candidates only*
Texas A&M Hotel and Conference Center Ballroom

February 2nd

Graduate Student Poster Presentations — 2:00 - 4:00 pm
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TAMU Research Cores and Student Resource Showcase — 2:00 - 4:00 pm
MSC Bethancourt Ballroom

Graduate Student Oral Presentations — 4:30 - 6:00 pm
Texas A&M Hotel and Conference Center Ballroom

Keynote Speech by Dr. Manu O. Platt — 6:00 - 6:30 pm
Senior Investigator, National Institute of Biomedical Imaging and Bioengineering
Texas A&M Hotel and Conference Center Ballroom

Banquet & Mingle. Appearance by The Singing Cadets — 6:30 - 8:30 pm
RSVP only
Texas A&M Hotel and Conference Center Ballroom

Our Team

Ximena Paez-Colasante, Ph.D.,
Chair, Life Sciences Graduate Recruitment Symposium

Associate Department Head for Administration,
Department of Nutrition

Biochemistry and Molecular Biophysics

Praveena Kanchupati, Ph.D.

Instructional Assistant Professor
and Graduate Program
Coordinator

Tera McAdoo

Graduate Program
Assistant

Our Team

Genetics and Genomics

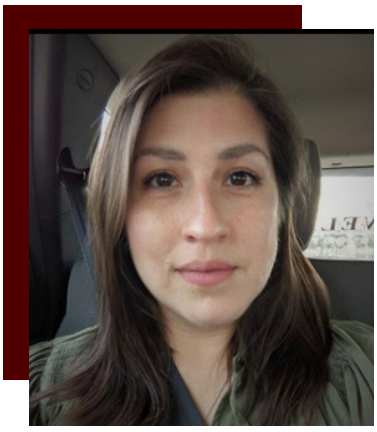
Isabel Caballero, Ph.D.

Program Manager

Tamara Ospina-Vega

Program Coordinator II

Medical Sciences



Fatima Bazan-Mota

Program Coordinator

Neuroscience

Sylvia M. Bernal Jones

Program Coordinator

Our Team

Nutrition

Tyler Fadal

Program Coordinator II

Jennifer Hancock

Program Coordinator II

Toxicology

Kayla Gibbs

Program Coordinator

Our Sponsors

TEXAS A&M UNIVERSITY

Graduate & Professional School

Division of Research

College of Agriculture & Life Sciences

School of Medicine

College of Arts & Science

School of Veterinary Medicine & Biomedical Sciences

Keynote Speaker

Dr. Fuhui Tong is Interim Associate Provost & Dean of the Graduate & Professional School and Professor of Bilingual/ESL education in the Department of Educational Psychology. Her research areas include program design and evaluation in bilingual/ESL education, language assessment, and the integration of language and literacy development with science learning among bilingual learners. She has authored and/or co-authored 120+ publications and has garnered over $90 million in extramural funding from the U.S. Department of Education, National Science Foundation, and Texas Education Agency. She joined the Graduate and Professional School in September of 2022.

Dr. Fuhui Tong

Professor, Department of Education
Interim Associate Provost & Dean of the Graduate & Professional School

Texas A&M University

Keynote Speaker

Dr. Manu O. Platt

Senior Investigator, NIBIB

Director, Center for Biomedical Engineering Technology Acceleration (βETA)

Head, Mechanics and Tissue Remodeling In Computational & Experimental Systems (MATRICES)

National Institute of Biomedical Imaging and Bioengineering

National Institutes of Health

Dr. Manu Platt received his B.S. in Biology from Morehouse College and a Ph.D. from Georgia Tech/Emory in Biomedical Engineering. After a postdoc at MIT, he returned to Georgia Tech/Emory Coulter Department of Biomedical Engineering where he advanced to full Professor.

His research program focuses on proteolytic mechanisms of disease, translational approaches to reduce strokes in people affected by sickle cell disease, and harnessing proteolytic networks and systems biology tools to predict disease progression. Integrated with research are mentoring goals of changing the look of the next generation of scientists and engineers through successful diversity programs he initiated. In 2023, Dr. Platt became the inaugural director of the NIH-wide Center for Biomedical Engineering Technology Acceleration (BETA Center), housed within the National Institute of Biomedical Imaging and Bioengineering (NIBIB). He is a Fellow of AIMBE, Fellow of BMES, The Root 100 in 2019, and AAAS Mentor Award in 2021.

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TAMU Resources Showcase Participants

Research Facility Cores

Texas A&M Institute for Genome Sciences and Society

- Texas A&M Preclinical and Phenotyping Core
- Molecular Genomics Core
- Bioinformatics Core

Microscopy Imaging Core

Flow Cytometry Core

Core Facilities - Division of Research

Student Resource Units

Texas A&M Graduate and Professional School

Career Center

Texas A&M Libraries

Counseling and Psychological Services (CAPS)

Graduate Student Government

Women in Science and Engineering (WISE)

Disability Services

BIOCHEMISTRY & MOLECULAR BIOPHYSICS

Poster Presentation Abstracts #1-22

1. PROBING AGONIST-DRIVEN TRANSLOCATION OF DIACYLGLYCEROL-SENSING C1 DOMAINS IN *S. CEREVISIAE*

Basak S.[1*], Katti S.[2], Green S.M.[2], Bankaitis V.A.[2], and Igumenova T.I.[1]

[1] Department of Biochemistry and Biophysics, Texas A&M University, College Station, Texas, USA
[2] Department of Molecular & Cellular Medicine, Texas A&M University, College Station, Texas, USA

ABSTRACT

C1-domains of Protein Kinase Cs are found to be regulated by sn-1,2-diacylglycerol (DAG). Among several modulators of PKC that have been reported, the two major classes we are interested in are phorbol esters and Bryostatins, showing drastic downstream effects upon C1-interaction. To address the effect of these modulators we expressed eGFP-C1 probes in yeast. In *S. cerevisiae*, significant pools of DAG are metabolically sequestered in vacuolar and plasma membranes, facilitating C1 domain probing. We show that two powerful PKC agonists, PDBu and Bryostatin-1, produce quantifiable C1-domain localization patterns, suggesting their agonistic effects.

2. RESHAPED ENERGY LANDSCAPE BY STRAIN-SPECIFIC MUTATION IN NON-STRUCTURAL PROTEIN 1 OF INFLUENZA A VIRUS CAN ALTER LONG-RANGE EPISTASIS

Iktae Kim[1], Alyssa Dubrow, Bryan Zuniga, Noah Sherer[1], Abhishek Bastiray[1]*
Pingwei Li[1], Jae-Hyun Cho[1]

[1] Department of Biochemistry and Biophysics, Texas A&M University, College Station Texas, USA

ABSTRACT

Non-structural protein 1 (NS1) is a multifunctional virulence factor of Influenza Virus A (IAV). NS1 interacts with a number of host proteins such as p85β of PI3K, TRIM25 and CPSF30, to suppress host innate immune responses. Despite high sequence and structural homology, NS1 protein of different IAV strains have exhibited strain-specific functions. Although NS1s share the

same binding residues for p85β, their binding energetics differ because of hidden epistatic interactions with strain-specific mutations. We demonstrate that the highly conserved core interface of NS1 to p85β, is invariable in conformation and dynamics. However, the rim interface residues surrounding the core residues have variable conformational dynamics. We find that strain-specific mutations altered the conformational energy landscape of NS1 through long-range epistatic interactions between strain-specific mutations and rim interface residues.

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3. THE DISCOVERY OF ENZYMES REQUIRED FOR BIOSYNTHESIS OF THE HS1 CAPSULAR POLYSACCHARIDE OF *CAMPYLOBACTER JEJUNI*

Ronnie Bourland[1*], Frank Raushel[§]

[1]Department of Biochemistry, Texas A&M University, College Station, TX, USA
[§]Department of Chemistry, Texas A&M University, College Station, TX, USA

ABSTRACT

Campylobacter jejuni is a gram-negative pathogenic bacterium commonly found in poultry and is the leading cause of gastrointestinal infections in the United States. *C. jejuni* possesses extracellular carbohydrate-based capsular polysaccharides composed of repeating units of monosaccharides bound via glycosidic linkages that contribute to bacterial colonization and pathogenicity. Serotype 1 (HS1) contains 13 different genes required for the production and presentation of the CPS. The full-length HS1_11 protein was expressed, purified, and characterized. The kinetic parameters for HS1_11 was determined through HPLC and NMR experiments with a turnover rate of 13 s^{-1} when saturating both CTP and L-glycerol-3-phosphate. HS1_09 is a multidomain protein with two predicted catalytic functions and required truncations to separately characterize each domain.

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4. EXPLOITING *BACILLUS SUBTILIS* MOBILIZATION BY *STREPTOMYCES SVICEUS* AS A PATH TO NATURAL PRODUCT DISCOVERY

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ABSTRACT

Chemical signals exchanged within microbial communities provide insight into innovative chemistry. Utilizing interactions between two soil bacteria, *Bacillus subtilis* and *Streptomyces*

venezuelae, our lab has developed an assay for novel antibiotic discovery. We found subinhibitory concentrations of chloramphenicol, a protein synthesis inhibitor synthesized by *S. venezuelae*, induce mobilization in *B. subtilis* colonies. *Streptomyces sviceus* also induces mobilization in *B. subtilis* but does not produce any known protein synthesis inhibitors. Therefore, *S. sviceus* produces an unidentified antibiotic or a metabolite inducing mobilization through an alternate mechanism. I aim to identify this unknown inducer metabolite and determine its mechanism of action.

5. CHARACTERIZATION OF THE ENZYMES INVOLVED IN ARABINOSE BIOSYNTHESIS IN THE CAPSULAR POLYSACCHARIDE OF *CAMPYLOBACTER JEJUNI*

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ABSTRACT

Campylobacter jejuni infection can result from poorly prepared poultry and possesses a capsular polysaccharide (CPS), which contributes to its diversity and pathogenicity. The different serotypes of *C. jejuni* each contain unique modified sugars that make *C. jejuni* pathogenic. Serotypes HS:15 contains L-arabinose within the CPS and also has four sugar modifying genes in the CPS gene cluster. HS-15.18 catalyzes the double oxidation of UDP-glucose to UDP glucuronic acid. A decarboxylase (HS15.19) removes the carboxyl to produce UDP-xylose. An epimerase (HS15.17) interconverts UDP-xylose with UDP-arabinose. The fourth enzyme in the pathway isomerises UDP-arabinopyranose with UDP-arabinofuranose.

6. A ZEBRAFISH KNOCKOUT OF COPPER TRANSPORTER CTR1 REVEAL AN ESSENTIAL REQUIREMENT OF COPPER IN ORGANISMAL DEVELOPMENT

Natalie M. Garza[1*], Andrew J. Latimer[2], Miriam C. Stein[1], Alexandra C. Jordan[1], Jonathan D. Gitlin[2] and Vishal M. Gohil[1]

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ABSTRACT

Copper is an essential micronutrient that acts as a catalytic cofactor for enzymes critical for human health. Recently, mutations in the copper importer Ctr1 have been shown to cause a fatal infantile disorder. We have constructed the first viable vertebrate model of Ctr1 deficiency in zebrafish to study its role in organismal development. Ctr1KO zebrafish exhibit hypoactivity of cuproenzymes resulting in stunted growth and reduced survival. Treatment with copper-ionophore elesclomol reverses these phenotypes. Our work uncovers a critical requirement of copper in zebrafish development and identifies elesclomol as an effective agent for treating copper deficiency caused by loss of Ctr1.

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7. DYSREGULATION OF RNA EDITING HELICASE 2 (KREH2) BY RNAI-SILENCING OR OVEREXPRESSION DISRUPTS EDITOSOME FUNCTION AND STABILITY

Joshua Meehan[1], Cody Goode[1*], Suzanne McDermott[2], Tyler Rodshagen[2], Laura McCleskey[1,] Zachary Goodall[1], Melanie Kuwahara[1], Neida Murillo[1], Zihao Yu[1], Addison Frese[1], Lanying Zeng[1], Jorge Cruz-Reyes[1]

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ABSTRACT

Trypanosoma brucei has a complex life cycle requiring adaptation to different hosts: procyclic form in insects and bloodstream form in mammals. Adaptation of the parasite during development includes extensive changes in mitochondrial U-indel editing. This process is catalyzed by ribonuclear protein complexes known the editosome. We previously showed that

RNAi-knockdown of KREH2, a key editosome protein, hinders full editing in all mRNA substrates examined in PCF and BSF. Furthermore, overexpression of KREH2 WT and dominant negative variants inhibit cell growth. Here we discuss new data examining these cell lines further, showing that KREH2 dysregulation disrupts editosome assembly.

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8. EXPLORING THE EVOLUTIONARY SPACE OF RNA PACKAGING AND STRUCTURE IN ssRNA PHAGES

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ABSTRACT

ssRNA phages are bacterial viruses with short single-stranded RNA genomes. Solved structures of these phages display unique RNA-protein and RNA-RNA interactions between isolates. The contributions of these unique interactions to virion assembly and disassembly are largely unknown. Using structure-guided mutagenesis and single-particle cryo-EM, we intend to solve the structures of RNA-packaging mutants and define all RNA components necessary for successful assembly. Using this information, we will design custom RNA cargos for an ssRNA phage-based nucleotide delivery system.

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9. CARDIOLIPIN DEFICIENCY LEADS TO THE DESTABILIZATION OF MITOCHONDRIAL MAGNESIUM CHANNEL MRS2 IN BARTH SYNDROME

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ABSTRACT

Barth syndrome (BTHS) is an X-linked cardio-skeletal myopathy caused by mutations in the *TAFAZZIN* gene that disrupts cardiolipin remodeling in mitochondrial membranes. How cardiolipin deficiency gives rise to clinical symptoms in BTHS patients is not fully understood. To

this end, we performed a metallomics analysis of BTHS mitochondria and found a significant reduction in mitochondrial magnesium content. Consistently, we found a decreased abundance of mitochondrial magnesium channel, MRS2, in multiple BTHS models, including patient cardiac tissue. Our study unveils the critical role of cardiolipin in maintaining MRS2 stability and suggests that perturbation in mitochondrial magnesium could contribute to BTHS pathology.

10. DISCOVERY OF MECHANISM BASED PEPTIDOMIMETICS DUAL-TARGET INHIBITORS OF CYSTEINE PROTEASES SARS-COV-2 3CL-PROTEASE (M^{PRO}) AND HUMAN CATHEPSIN L INHIBITORS

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ABSTRACT

Human cathepsin L (hCatL) and SARS-CoV-2 Main protease (M^{Pro}) are cysteine proteases (CPs) involved in endosomal viral entry and viral replication of SARS-CoV-2 respectively. SARS-CoV 2 infection elevates the expression of hCatL, and overexpressed hCatL in turn accelerates the viral infection by forming a vicious cycle. These properties of both CPs make them promising therapeutic targets. The efficacy of the current FDA-approved drug nirmatrelvir is reduced with the arrival of new mutations within its molecular target M^{Pro} especially the mutations at the Gln recognizing S1 subsite which makes SARS-CoV-2 resistant to nirmatrelvir. In this project, we developed peptidomimetics with five new Gln mimics that inhibit both CPs (M^{pro} and hCatL). Peptidomimetics compounds with our new 2-Pyridonyl-Ala sidechain (structural chimera of Phe and Gln) inhibit both CPs at nanomolar concentrations. It establishes all crucial H-Bonding interactions with the S1 subsite of M^{pro} and is a close mimic of Phe that exerts potent inhibition for hCatL. In addition, these substrate-based inhibitors are engineered with new mild electrophilic centers that require high residence time in the active site and closer proximity to cysteine to form covalent adducts. Peptidomimetic compounds containing δ-lactols and cyclic imino-ketones are nanomolar inhibitors of hCatL and display at least 100-fold selectivity over its homologue hCatB. In fact, cyclic imino ketone formed a reversible michael adduct with only hCatL over hCatB and blocked SARS-CoV-2 in Vero-E6 and A549/ACE2 cells at nanomolar concentrations.

11. SMALL SIGNALING PEPTIDES REGULATE DEVELOPMENT IN *SORGHUM BICOLOR*

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ABSTRACT

Bioenergy Sorghum is a well-established bioenergy crop due to its long stem (3-5m), diploid genome, drought tolerance, and efficient C_4 photosynthesis. These traits allow Sorghum to sequester high amounts of atmospheric CO_2 in the form of complex carbohydrates like cellulose, starch, and sucrose which can be converted to bioethanol. Sorghum stems are the main organ harvested for biofuel production and its development is highly complex. Its regulation may be influenced by small signaling peptides. I use computational approaches to find small signaling peptide candidates in sorghum and test their function *in-vivo* via novel injection method.

12. REQUIREMENTS OF A SINGLE-STRANDED RNA BACTERIOPHAGE RNA-DEPENDENT RNA POLYMERASE

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ABSTRACT

Single-stranded RNA bacteriophages (ssRNA phages) are relatively simple viruses, 3-4 kb of positive-sense RNA, encoding four proteins: maturation, coat, replicase, and lysis proteins. The replicase protein is an RNA dependent RNA polymerase that has been shown to rapidly replicate RNA and induce a high mutation rate, assisting in their quasi-species status. Qβ's replicase has been extensively studied due to the possible applications of such a tool, but nothing has come to fruition. Here we determine the requirements of the broad host range ssRNA phage PRR1's replicase through biochemical assays to better understand how to utilize it as an evolutionary tool.

13. UNDERSTANDING THE ENDOSOMAL N-BAR AND EHD PROTEINS IN REGULATED MEMBRANE FISSION

Lauren Kustigian[1], Xue Gong[1], Wei Gai[1], Jirapat Thongchol[1], Brianna Martin*, Jason Puchalla[1], Junjie Zhang[1], Chavela M. Carr[1], and Hays S. Rye[1]
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ABSTRACT

Endocytic recycling maintains the composition of proteins and lipids at the plasma membrane, enabling eukaryotic cells to control material and signaling responses to their environment. Two major protein families implicated in this process, amphiphysins (N-BAR) and EHD, are known to play a role in membrane fission at the recycling endosome, but their mechanism of action remains elusive. Our work focuses on utilizing various biophysical, biochemical, and structural techniques to determine how the N-BAR and EHD proteins function, both independently and together, to create productive cargo carriers that transport cargo from the recycling endosome.

14. RECEPTOR PROTEIN TYROSINE PHOSPHATASES ARE REQUIRED FOR SENSORY AXON WIRING AND REGULATED BY POMT-MEDIATED O-MANNOSYLATION IN *DROSOPHILA*

Pedro Monagas-Valentin[1*], Robert Bridger[2], Ishita Chandel[1], Boris Novikov[1], Lance Wells[2] and Vlad Panin[1]

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ABSTRACT

Protein O-mannosylation is a unique post-translational modification required for proper neuromuscular development in animals. Deficiencies in *POMT1/2* lead to dystroglycanopathies, a group of congenital muscular dystrophies associated with neuromuscular abnormalities thought to be caused by defective modification of Dystroglycan. However, functional substrates of *POMT1/2* besides Dystroglycan remain largely uncharacterized. Using glycoproteomics and genetic approaches, we characterized POMT-dependent O-mannosylation of PTP69D. Our work suggests that O-mannosylation is essential for phosphorylation levels in *Drosophila*. These mechanisms are potentially conserved in mammals and may shed light on the involvement of POMT1/2 and RPTPs in human pathologies.

15. DEFINING HUMAN CATHEPSIN L: MECHANISTIC DETERMINATION THROUGH STEADY STATE AND PRE-STEADY STATE KINETICS

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ABSTRACT

Human cathepsin L (hCatL) is a member of the papain family of cysteine proteases. CatL is a lysosomal enzyme ubiquitous in cells and tissues, and its dysregulation has been associated with metastatic cancers and viral entry. To develop new classes of hCatL inhibitors, we seek to understand the catalytic mechanism of hCatL more fully in terms of the following questions: (1) what is the protonation state of the Cys-His catalytic dyad in the free enzyme form that binds both substrates and inhibitors? (2) For the double-displacement mechanism of cysteine proteases, is the acylation or de-acylation half-reaction of the mechanism rate-limiting?

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16. IDENTIFYING THE ROLE OF CONFORMATIONAL ENTROPY IN INTEGRAL MEMBRANE PROTEIN FOLDING AND FUNCTION

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ABSTRACT

Understanding the motions of amino acid side chains in proteins is crucial for a complete picture of their energetics, structure, and function. The conformational entropy manifested in sub-nanosecond motion can be a pivotal contribution to: thermodynamics of molecular recognition, protein function, and protein folding. This project will investigate the internal motion and conformational entropy of integral membrane proteins (IMPs). The change in conformational entropy upon binding of these IMPs to their biologically relevant ligands will be explored. This will chart completely unexplored territory of how the conformational entropy contributes to the thermodynamics of molecular recognition by integral membrane proteins.

17. MECHANISTIC BASIS OF THE INTERACTION BETWEEN NON-STRUCTURAL PROTEIN 1 OF INFLUENZA A VIRUS WITH TRIM25

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ABSTRACT

Influenza A viruses (IAVs) are negative-sense, ssRNA viruses responsible for many global health crises. One central virulence factor, non-structural protein 1 (NS1), plays important roles in suppressing the host's innate immune response such as through the inhibition of TRIM25; however, this interaction at the molecular level remains unclear. Here, we employ bio-layer interferometry (BLI) and size exclusion chromatography with multi-angle light scattering (SEC-MALS) to investigate the binding characteristics of TRIM25 with four NS1s of different strains. Our study is expected to provide mechanistic insight into the NS1-TRIM25 interactions—providing clues to how different NS1 strains evade the host immune response.

18. STRUCTURAL DYNAMICS OF RECEPTOR RECOGNITION AND PH-INDUCED DISSOCIATION OF FULL-LENGTH *CLOSTRIDIOIDES DIFFICILE* TOXIN B

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ABSTRACT

Clostridioides difficile secretes Toxin B (TcdB) as one of its major virulence factors, which binds to intestinal epithelial and subepithelial receptors, including frizzled proteins and chondroitin sulfate proteoglycan 4 (CSPG4). Here, we present cryo-EM structures of full-length TcdB in complex with the CSPG4 domain 1 fragment (D1401-560) at cytosolic pH and the cysteine-rich domain of frizzled-2 (CRD2) at both cytosolic and acidic pHs. These results reveal the structural dynamics of TcdB during its pre-entry step upon endosomal acidification, which provides a basis for developing therapeutics against *C. difficile* infections.

19. DEFINING MITOCHONDRIAL PROTEIN FUNCTIONS USING DEEP NEURAL NETWORKS

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ABSTRACT

Despite mitochondria's crucial role in cellular processes, the molecular function of many proteins remains unknown. We repurpose AlphaFold Multimer into a classification model, predicting 85% dimeric and 50% oligomeric pairs with a 0.002 false positive rate. Applying to the mitochondrial proteome, we screened ~640,000 binary combinations, identifying 2895 interactions among 1004 proteins. This links 85 uncharacterized mitochondrial proteins to known pathways. Experimental validation reveals Coa4 and Cmc2, poorly characterized proteins, play specific roles in cytochrome *c* oxidase assembly. Our work expands the utility of AlphaFold beyond the structural realm, providing a foundation for the complete understanding of mitochondrial protein function.

20. STRUCTURES OF ssRNA BACTERIOPHAGES AND INTERACTION WITH THEIR HOST RECEPTORS

Jirapat Thongchol[1,2,*] and Junjie Zhang[1,2]

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[2]Department of Biochemistry and Biophysics, Texas A&M University, College station, TX, USA

ABSTRACT

Positive-sense single-stranded RNA bacteriophages (ssRNA phages), discovered six decades ago, infect gram-negative bacteria via retractile pili. Using cryo-EM, we elucidated mature ssRNA phage structures (pepeviruses) infecting *Pseudomonas aeruginosa*. LeviOr01 and PP7, the only culturable pepeviruses, rely on the host type IV pilus (T4P) receptor. Unique among ssRNA phages, mature pepeviruses exhibit a near-icosahedral T=3 capsid with two Maturation (Mat) proteins. Interaction analysis reveals that pepevirus PP7 binds to a single pilin subunit on T4P, triggering T4P detachment upon entry. This study contributes to understanding the complex dynamics of ssRNA phage infections and leads to a re-evaluation of the structural simplicity of ssRNA phages.

21. TRACELESS AND EFFICIENT DELIVERY REAGENTS FOR GENE EDITING IN CENTRAL NERVOUS SYSTEM CELLS

Nathan R. Williams1*, Dr. Jason Allen[1], Dr. Theresa Sutherland[2], Dr. Kristina Najjar[1], Dr. Alfredo Erazo-Oliveras1, Dr. Jean-Philippe Pellois1

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ABSTRACT

The delivery of gene editing proteins into target cells is impeded by the degradation of those proteins in the cells' lysosomes. We have thus engineered a set of small peptides, based on the HIV-TAT peptide, which can assist the escape of desirable proteins from the endosomes of the cell before lysosomal degradation. These peptide delivery agents are also traceless, degraded by the target cells after assisting delivery. In order to optimize the activity of these delivery peptides and enhance gene editing efficiency in CNS cells, we determine here the influence of peptide hydrophobicity on delivery efficiency and toxicity.

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22. UNDERSTANDING HOW PROTEIN AGGREGATE STRUCTURE IMPACTS DISAGGREGATION BY MOLECULAR CHAPERONES

YuChen Yang[1] and Hays Rye[1]

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ABSTRACT

Protein aggregation is one of the most serious problems faced by living organisms. In humans, protein aggregation has been linked to severe diseases, such as type II diabetes, Alzheimer's, and Parkinson's diseases. All living cells use highly conserved networks of specialized proteins, known as molecular chaperones, to prevent and reverse protein aggregation. In our lab, we employ RuBisCO from the nitrogen-fixing proteobacterium *R. rubrum* and PepQ from *E. coli* as model proteins for chaperonin mediated protein disaggregation. My goal is to develop a detailed model of how molecular chaperones engage and dismantle aggregates and how aggregate structural differences lead to divergent responses to the molecular chaperone disaggregases.

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23. CHROMOSOMAL CLUES: RESOLVING CONTROVERSIAL PHYLOGENETIC RELATIONSHIPS USING SEX CHROMOSOMES

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ABSTRACT

Almost all living placental mammals possess XX/XY sex chromosomes, where only the Y contains male-specific genes. These genes are conserved across species, given their role in transcription, translation, and male sex development, yet bear unique evolutionary signatures within lineages. Due to difficulties in sequencing and assembling the chromosome, the Y has remained the only chromosome that has not been thoroughly scrutinized for phylogenetic inference. We present the first comprehensive phylogenetic analysis of the Y chromosome using over 60 mammals. Moreover, we demonstrate the utility of sex chromosomes in delineating controversial phylogenetic relationships that have been unresolved through autosomal-only analyses.

24. STRAIN-SPECIFIC DIFFERENCES IN THE EFFECT OF MATERNAL DIETARY VITAMIN A INTAKE ON THE PRENATAL HIGH-FAT DIET INDUCED OBESITY IN MICE

Marianny Alvarado-Gonzalez[1*], Samuel Rosean[2], Simone Sidoli[3], Younkyung Kim[4], Loredana Quadro[4] & Masako Suzuki[15]

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ABSTRACT

Obese pregnancies in underserved communities often suffer from micronutrient deficiency due to food insecurity, which can lead to health risks for mothers and offspring later in life. To assess the effect of dietary vitamin A (VA) and fat intake on liver proteome and retinoid status and its genetic contributions, we performed a dietary intervention on three mouse strains and liver proteome analysis to identify differentially expressed proteins between diets. We also measured circulating and stored retinoid statuses. Our goal is to elucidate the mechanisms of how maternal dietary intake and genetic background influence offspring phenotypes later in life.

25. CONSEQUENCES OF IPA ISOFORM LOSS IN MULTIPLE MYELOMA PATHOPHYSIOLOGY

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ABSTRACT

Multiple myeloma (MM) is an incurable blood based cancer of plasma cells with a host of symptoms that rapidly reduce patients' quality of life. An emerging hallmark of MM involves reduced intronic polyadenylation (IPA) usage, causing the dysregulation of isoform expression in the malignancy. To better understand the consequences of the loss of IPA isoforms, we are probing the function of the IPA isoform of Ikaros (IKZF1), a transcription factor with known roles in MM pathophysiology, by engineering MM lines to examine whether the isoform has tumor suppressor activity.

26. EXPLORING THE EVOLUTIONARY DYNAMICS OF ACHIASMATIC MEIOSIS

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Crossing over during meiosis is crucial for genetic diversity. In species with chromosomal sex determination, reduced recombination between sex chromosomes occurs through multiple mechanisms, one of them being achiasmatic meiosis. Sexually antagonistic variation, where alleles benefit one sex over the other, was traditionally thought to drive these changes. However, recent explanations consider epistatic differences, haploid and diploid selection variations, and the reduction of aneuploid gametes as factors influencing achiasmy. The evolution of reduced recombination plays a vital role in sex chromosome divergence. We investigate the evolutionary dynamics of achiasmatic meiosis through an integrated methodology employing computer simulations and mathematical models.

27. EXPLORING A SINGLEMINDED-2S / SIRTUIN 3 INTERACTION IN THE REGULATION OF MITOCHONDRIAL DYNAMICS OF ER+ BC

Hannah Carter[1,3*], Steven Wall[2], Garhett Wyatt[1], Jessica Elswood[1], Lilia Sanchez[1], Kelly Scribner[1], Elizabeth Wellberg[1], Weston Porter[1]

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ABSTRACT

Characterizing the mechanisms underlying the regulation of mitochondrial dynamics are critical to understanding normal development and breast cancer in the mammary gland. Mitochondrial homeostasis is maintained through an intricately controlled cycle of fusion and fission. Our previous studies have highlighted the significance of Singleminded-2s (*SIM2s*), a member of the bHLH/PAS family, in regulating mitochondrial dynamics during the progression of estrogen receptor-positive (ER+) breast cancer. We have identified *SIM2s* as a tumor suppressor expressed in mammary epithelial cells, known to inhibit epithelial-mesenchymal transition (EMT) and metastasis. Loss of *SIM2s* leads to alterations in mitochondrial morphology and dynamics, characterized by decreased OPA1 expression and increased DRP1 levels, resulting in decreased mitochondrial fusion and elongation and increased fission. The knockdown (KD) of *SirT1* and *SirT3* in MCF7 WT cells via siRNA confers a downregulation of SIM2 expression. In the MCF7 SIM2KO cells, KD of *SirT1* and *SirT3* leads to the downregulation of OPA1 and DRP1. These results in combination with past studies and literature review lead us to hypothesize the presence of an interaction between SIM2 and SirT3 in the regulation of mitochondrial dynamics.

28. ELUCIDATING THE EVOLUTION OF THE RECOMBINATIONAL LANDSCAPE OF PLACENTAL MAMMALS USING COMPARATIVE GENOMICS

Isabella R. Childers[1,2*], Nicole M. Foley[1], Kevin R. Bredemeyer[1,2], and William J. Murphy[1,2]

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ABSTRACT

Despite the ability to sequence whole genomes, a prevailing issue of phylogenetics is how to accurately choose the correct sequences for inferring species relationships. Emerging studies have shown a negative correlation between local recombination rates and the frequency of the original branching events ("species tree"). We used the annotated genomes and recombination maps of four placental mammals (human, domestic cat, aardvark, and Hoffmann's two-toed sloth) to investigate how the recombination landscape is shaped through time. We found genes that are conserved in the lowest and highest recombining regions, which will be useful for future phylogenetic and adaptive gene studies, respectively.

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29. CRISPR INTERFERENCE (CRISPR-I) FOR SEQUENCE-SPECIFIC CONTROL OF GENE EXPRESSION IN *AEDES AEGYPTI*

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ABSTRACT

Genetic manipulation of disease vectors is of utmost demand with the onset of global temperatures rising, causing an increased burden on human health. To develop a CRISPR-I system in *Aedes aegypti*, we will use the identification of human transcriptional effectors to determine which human transcriptional repressors are orthologous. We will target the insect immune response pathway and measure the quantification of immune defense genes with qPCR.

By developing a CRISPR-I system in *Aedes aegypti*, sequence-specific repression can be achieved, which has a multitude of applications in vector biology for GPM use in modification of wild mosquito populations.

30. MITIGATING THE IMPACT OF PATERNAL BINGE DRINKING ON FETAL ALCOHOL SPECTRUM DISORDERS WITH ANTIOXIDANTS

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ABSTRACT

More focus has begun to shift towards how a man's preconceptual actions can manifest as symptoms in his offspring. Male C57BL/6J mice were treated with ethanol and/or antioxidants and subsequently mated to evaluate the interactions of the treatments. The presentation of fetal alcohol spectrum disorder symptoms was evaluated through qPCR, craniofacial linear morphometric measurements, and placental histology. Male health was also considered to determine the antioxidants' effectiveness through colorimetric assays and mitoQC reporter mice visualization. Results thus far show antioxidants to alleviate offspring's symptoms associated with paternal binge drinking.

31. COTTON CHRONOLOGY: CONVOLUTIONAL NEURAL NETWORK ENABLES SINGLE-PLANT SENESCENCE SCORING WITH TEMPORAL DRONE IMAGES

Aaron J. DeSalvio[1*], Alper Adak[2], Mustafa Arik[2], Seth C. Murray[2*], Oriana Garcia-Ramos[2], Serina M. DeSalvio[1], David M. Stelly[2]

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ABSTRACT

Senescence is a degenerative biological process that affects most organisms. Tracking time-series senescence data has previously required expert annotation and can be laborious for large-scale research. A convolutional neural network (CNN) was trained on unoccupied aerial system images of individual cotton (*Gossypium tomentosum*) plants. Using images from 14 drone flights capturing most of the senescence window, the CNN achieved 71.4% overall classification accuracy across six senescence categories. These results demonstrate that minimally pre-processed UAS images can enable translatable implementations of high throughput phenotyping using deep learning methods. This has applications for understanding fundamental plant biology and in plant breeding and genetics.

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32. GPX4 ALTERS IMMUNE RESPONSE AND LIPID OXIDATION IN MYCOBACTERIUM TUBERCULOSIS INFECTION

Lily M Ellzey [1,2*], Chi G Weindel[2], Aja K Coleman[2], Kristin L Patrick[1,2*], Robert O Watson[1,2*]

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ABSTRACT

Multiple lines of evidence demonstrate that mitochondrial ROS promotes necrotic cell death and poor disease outcomes in response to the important human pathogen *Mycobacterium tuberculosis*

(Mtb). However, the molecular mechanisms through which oxidative stress promotes Mtb pathogenesis remain unclear. We know that mitochondrial ROS accumulation fosters lipid oxidation and that cells repair this damage by expressing a reductase called glutathione peroxidase 4 (GPX4). We hypothesize that the balance between lipid oxidation and GPX4 activity is a key determinant of Mtb infection outcomes. Motivated by these foundational observations, we are working to understand how the accumulation of oxidated lipids influences mitochondrial homeostasis and licenses entry into different cell death modalities.

33. CONJUGATIVE TYPE IV SECRETION SYSTEMS ENABLE BACTERIAL ANTAGONISM THAT OPERATES INDEPENDENTLY OF PLASMID TRANSFER

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ABSTRACT

Interactions mediated by secretion systems are among ways in which bacteria interact with one another. We report a new antagonistic property of a type IV secretion system (T4SS) from plasmid RP4. The genetic determinants were scrutinized, and results suggested that the phenotype is independent of molecular cargo. Its effectiveness against varied Gram-negative bacteria and mixed bacterial populations was also demonstrated. Moreover, the phenotype is not limited to RP4, but was also observed in plasmid R388. Our results are the first demonstration of conjugative T4SS antagonism between Gram-negative bacteria on the genetic level and provides the foundation for future mechanistic studies.

34. TOWARD TELOMERE-TO-TELOMERE FELID GENOMES

Andrew J. Harris[1,2*], Leslie A. Lyons[3], Wesley C. Warren[4], Kendra Hoekzema[5], Evan E. Eichler[5,6], William J. Murphy[1,2]

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ABSTRACT

Advancements in genome assembly and long-read sequencing technologies have ushered in the era of telomere-to-telomere (T2T) genomes. We present results and analyses from near T2T assemblies for the domestic cat (*Felis catus*) and Geoffroy's cat (*Leopardus geoffroyi*) using a Safari cat F1-interspecific hybrid. Using PacBio HiFi and Oxford Nanopore "ultra-long" (> 100kb) reads, the genome assembler, Verkko, assembled 16/19 chromosomes from the domestic cat and 14/18 chromosomes from the Geoffroy's cat into a single sequence, with 11/19 and 5/18 being T2T, respectfully. We highlight similar results from a domestic cat x serval (*Leptailurus serval*) F1 hybrid trio and our ongoing progress toward generating T2T genomes across the entire cat family.

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35. GENETIC DIVERSITY AND THE CONTRIBUTIONS TO FASD

Samantha Higgins[1,3*], Sanat Bhadsavle[1], Destani Derrico[1], Alison Basel[1], Kara Thomas[1], Katie Scaturro[1], and Michael Golding[1] in collaboration with David Threadgill[2]

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ABSTRACT

Our genetic makeup influences the way we respond to environmental factors, such as the biological response to ethanol intake. Fetal Alcohol Spectrum Disorders (FASD) can be

developed from parental alcohol consumption. We employed a Simple Diversity Outcross (SDO) mouse model to investigate the influence of genetic contribution to FASD. We find that SDO mice consume ethanol and metabolize it more readily than the commonly used C57BL/6J strain. We have found that no major macro developmental or linear craniofacial defects are present in offspring sired by SDO paternal drinkers in contrast to what we see in offspring sired by C57BL/6J drinkers.

36. ALTERATIONS IN ELECTRON TRANSPORT CHAIN SUPER COMPLEXES ACROSS NORMAL MAMMARY GLAND DEVELOPMENT

Ramsey M. Jenschke*[1], Steven W. Wall[1], Jessica Epps[1], Lilia Sanchez[1], Monique Rijnkels[1], Weston Porter[1]

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ABSTRACT

Singleminded-2 (Sim2s) stabilizes mitochondrial respiratory chain super complexes (MRC SCs) in cancer cells. Sim2s also regulates mitophagy during mammary gland development, prompting an investigation into MRC reorganization and mitophagy in normal development. We show that the MRC undergoes structural changes during differentiation in vitro. Reduction of Sim2 altered the differentiation-associated changes in MRC, indicating the role of Sim2 in regulating the difference of mitochondria. *In vivo* images using Mitochondria Quality Control mice show higher levels of turnover in lactation, corresponding with peak differentiation and changes in MRC. This data shows that mitophagy is driven by the need for MRC SC stability.

37. GENOME-WIDE ASSOCIATION STUDIES REVEAL THE GENETIC ARCHITECTURE OF DEVELOPMENT AND YIELD-RELATED TRAITS IN NORTH AMERICAN MAIZE ACROSS ENVIRONMENTS AND YEARS

Fatma Ozair[1,3*], Seth C. Murray[2], and Alper Adak[2]

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ABSTRACT

Understanding the genetic basis of complex traits in crop models is important for biological discovery and crop improvement. The maize genome includes quantitative trait loci contributing to complex traits often investigated in a few environments. Here, field data collected through the Genomes-to-Fields project was analyzed to determine how genotype-by-environment interactions influence grain yield (GY), days to anthesis (DTA), days to silking (DTS), and plant height (PHT) using three testers across 30 diverse environments. We discovered 120 SNPs significantly associated with these traits using genome-wide association studies. Future work will identify candidate genes linked to these SNPs for further hybrid improvement.

38. HEMOGLOBIN BUFFERS T-LYMPHOCYTE MITOCHONDRIAL REDOX AND INFLAMMATION AFTER PSYCHOLOGICAL TRAUMA

Emily C. Reed[1,2,3*], Tatlock H. Lauten[1,2], Tamara Natour[1,2], Adam J. Case[1,2]

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ABSTRACT

Post-traumatic stress disorder (PTSD) causes heightened inflammation, but the mechanisms underlying this inflammation are unclear. Using a murine model of PTSD, we have shown psychological trauma alters T-lymphocyte inflammation and mitochondrial redox. Single-cell RNA sequencing identified hemoglobin-alpha 1 (Hba-a1) as highly upregulated in T-lymphocytes after psychological trauma, but its function in these cells is unknown. Using wild-type and Hba-a1 knockout mice, we investigated the function and regulation of Hba-a1 in T-lymphocytes after psychological trauma. Overall, loss of Hba-a1 causes a pro-inflammatory and potentiated redox environment in T-lymphocytes, suggesting a potential buffering effect against the molecular pathophysiology caused by psychological trauma.

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39. MICROBE INTERACTION UNVEILED THROUGH CLICK CHEMISTRY

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ABSTRACT

Synthetic multicellular systems offer potential in regulating multi-component microbial communities, understanding biofilm formation, and facilitating therapeutic delivery. Our investigation focuses on elucidating the interactions between single-celled eukaryotic

microorganisms and bacteria through click chemistry, with the objective of gaining insights into the advantages of nutrient exchange and the potential for redesigning community functions. Specifically, we used the strain-promoted azide-alkyne cycloaddition (SPAAC) - a commonly used reaction in click chemistry for linking molecules and peptides. This method resulted in the bacteria strain binding with single-celled algae, forming a single layer of bacteria around them.

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40. SINGLEMINDED2S IMPACTS MITOPHAGY AT ER-MITOCHONDRIA CONTACTS TO PROMOTE MAMMARY GLAND DIFFERENTIATION

Lilia Sanchez[1]*, Jessica Epps[1], Scott Pearson[1], Cole McQueen[1], Steven Wall[1], Elizabeth Wellberg[1], H. Ross Payne[1], Rola Barhoumi[1], Monique Rjinkels[1], Weston Porter[1]

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Investigating the regulatory mechanisms and crosstalk between organelles during cell differentiation is essential for understanding both normal mammary gland development and breast cancer etiology. We show singleminded2s (SIM2s) is novel mitophagy receptor and loss of SIM2s disrupts mitochondrial homeostasis and inter-organelle communication to the detriment of functional mammary gland development. We anticipate SIM2s' role is through directly interacting with mitophagy components at ER-mitochondrial contacts. Insights from our studies will contribute to knowledge of ER-mitochondrial contacts as a platform for homeostatic signaling, specifically during during normal development and cancer progression, which can be exploited for therapeutic purposes.

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41. NOVEL PROTEIN DNA CIRCUIT FOR THE DETECTION OF SHIGA TOXIN

Benjamin Thomas[1*], Rudo Simeon, Caitlyn Mutchler[2], and Zhilei Chen[3]

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3 Department of Microbial Pathogenesis and Immunology, Texas A&M School of Medicine, College Station, Texas, USA

ABSTRACT

Shiga toxin producing E. coli (STEC) is a foodborne pathogen that infects over one million individuals globally each year. Although early intervention has proven key in mitigating the risk

of developing hemolytic uremic syndrome (HUS) due to STEC infection, the need for lab tests (i.e., mass-spectrometry, ELISA) significantly delays the STEC diagnosis and treatment. Building upon a panel of biologics with high affinity to Shiga toxin (Stx2), this study developed a novel protein-DNA circuit for rapid (<2 hour) and isothermal detection of Stx2. We believe our assay can be extended to the diagnosis of other antigens in a plug-and-play format.

42. SIM2s MEDIATION OF STING-MEDIATED IMMUNITY DURING MAMMARY GLAND DEVELOPMENT

Ramiah Vickers[1*], Garhett Wyatt[1], Lilia Sanchez[1], Jessica Epps[1], A. Phillip West[1,2], Monique Rijnkels[1], Weston Porter[1]

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ABSTRACT

Instances of postpartum breast cancer (PPBC) are on the rise due to women deferring childbirth until their late 30s. These tumors are more advanced and generally have poor prognoses as patients are ~ 3x more likely to develop metastasis. Given its highly aggressive nature and lack of beneficial therapeutic treatments, our lab is interested in investigating novel drivers of PPBC initiation. Using in vitro and in vivo models we have identified a relationship between SIM2, STING and mitochondrial ROS that is of interest in future investigations for breast cancer development following lactation.

MEDICAL SCIENCES

Poster Presentation Abstracts #43 – 63

43. THE *MYCOBACTERIUM TUBERCULOSIS* SECRETED PROTEIN RV1075C MANIPULATES HOST HISTONE METHYLTRANSFERASES TO PROMOTE INFECTION

Aja K. Coleman[1*], Allison Wagner[1], Haley M. Scott[1], Robert O. Watson[1], and Kristin L. Patrick[1]

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ABSTRACT

Mycobacterium tuberculosis (Mtb) is one of the most infectious and deadly pathogens worldwide. An *in silico* screen identified a putative nuclear localization signal in the Mtb protein Rv1075c. We confirmed that Rv1075c traffics to the macrophage nucleus and biochemically associates with chromatin. We also found that expression of Rv1075c hyperinduces expression of *Ifnb1* and *ΔRv1075c* Mtb fail to induce early type I IFN responses during infection of primary murine macrophages. Together, our data argues that Rv1075c plays a critical role in helping Mtb establish a favorable niche by promoting a pro-Mtb gene expression program during macrophage infection.

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44. BORRELIA BURGDORFERI BB0473, A PUTATIVE MULTIDRUG AND TOXIN EFFLUX PROTEIN, IS IMPORTANT FOR MAMMALIAN INFECTIVITY

David Gafford-Gaby[1*], Sourav Roy[2], Brandon L. Garcia[2], and Jenny A. Hyde[1]

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[2] Department of Microbiology and Immunology, Brody School of Medicine, East Carolina University, Greenville, NC, USA

ABSTRACT

Borrelia burgdorferi, the causative agent of Lyme disease, is a bacterial pathogen that must dynamically adapt to its surroundings by acquiring nutrients and disposing of waste within diverse host environments in order to survive and cause disease. This work investigates the role of a predicted borrelial multidrug and toxin efflux (MATE) transporter in maintaining this metabolic balance in *Borrelia burgdorferi* and supporting survival in its mammalian hosts and tick vectors. Such studies allow us to describe how *Borrelia burgdorferi* maintains its internal homeostasis and adapts to metabolic challenges, providing critical information on the mechanisms of Lyme disease pathogenesis.

45. TRIM14 REGULATES MITOCHONDRIA HOMEOSTASIS DURING MYCOBACTERIUM TUBERCULOSIS INFECTION

Cory Mabry[1*], Aja Coleman[1], Chi Weindel[1], Kristin Patrick[1], Robert Watson[1]

[1] Department of Microbial Pathogenesis and Immunology, Texas A&M University, Bryan, TX, USA

ABSTRACT

Mycobacterium tuberculosis (Mtb) has evolved to suppress macrophage apoptosis. TRIM (tripartite motif-containing) proteins are key regulators of innate immunity and cell death. TRIM14 acts as a scaffold between the kinase TBK1 and STAT3 to increase STAT3 activity. STAT3 is a transcription factor, but recent studies have revealed STAT3 regulates mitochondria health. In the absence of TRIM14, STAT3 is not properly targeted to mitochondria. The lack of STAT3 in Trim14-/- primary macrophages is associated with significantly increased apoptotic cell death and altered mitochondria homeostasis. Our data supports a model whereby TRIM14 reduces apoptosis and maintains mitochondria homoeostasis in a STAT3-dependent manner.

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46. AN ADENO-ASSOCIATED VIRUS (AAV)-BASED TOOL TO SPECIFICALLY DAMAGE MTDNA IN ASTROCYTES WITHIN PRE-SPECIFIED REGIONS OF THE ADULT MOUSE BRAIN

Daniela A. Ayala[1]*, Anthony J. Matarazzo[1], Bonnie Seaberg[1], Mendell Rimer[1], Rahul Srinivasan[1,2].

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ABSTRACT

Astrocytes display robust mitochondrial Ca2+ influx in live dorsolateral striatum (DLS) slices, suggesting their active role in neuronal function. We proposed disrupting astrocyte mitochondrial function within specific brain regions triggers neuronal dysfunction and accelerated neurodegeneration. Using an AAV expressing the restriction enzyme PstI (Mito-PstI) under the astrocyte GfaABC1D promoter, targeted specifically to astrocytic mitochondria, we induced damage to astrocytic mitochondrial DNA (mtDNA). Mito-PstI introduction to mouse DLS, a Parkinson's disease (PD)-related region, resulted in altered mitochondrial function and structure, along with changes to astrocyte reactivity. This AAV tool offers valuable insights into neurodegeneration acceleration, particularly in the context of PD.

47. IMPACT OF RENAL LYMPHANGIOGENESIS ON RENAL PHOSPHATE HANDLING FOLLOWING KIDNEY INJURY

Thien T Phan[1], Heidi A. Creed[1], Dawson L. Weiss[1], Myles Vu[1], Andrea J. Reyna[1], Joseph M. Rutkowski [1*]

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Chronic phosphate (Pi) intake contributes to kidney damage. This study investigates the impact of renal lymphangiogenesis on Pi balance. Mice on a 2% Pi diet for 3 weeks or 2 months exhibit increase kidney lymphangiogenesis and elevated VEGF-C/D levels. KidVD mice, with enhanced renal lymphatic density, maintain serum and urinary Pi excretion comparable to wildtype mice on both chow and high Pi diets. In a Pi-driven CKD model, however, KidVD mice display increased Pi secretion post-injury, suggesting a potential role for kidney lymphatics in CKD recovery. The findings propose that renal VEGF-D overexpression and lymphangiogenesis may mitigate Pi retention following kidney injury, aiding in improved CKD outcomes.

48. HYPERTENSIVE STIMULI INCREASE BONE MARROW DERIVED MACROPHAGES IN VITRO AND IN VIVO

Hannah L. Smith[1*], Bethany L. Goodlett[1], Shobana Navaneethabalakrishnan[1], Brett M. Mitchell[1]

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ABSTRACT

Renal macrophage infiltration and inflammation are pivotal in hypertension (HTN). Mice with salt-sensitive or angiotensin II (AngII)-induced HTN have increased renal activated and/or pro inflammatory macrophages. Bone marrow-derived macrophages (BMDMs) grown in M-CSF were unaffected when treated with salt or AngII; however, both salt- and AngII-treated BMDMs cultivated in GM-CSF showed increased renal activated macrophages, pro-inflammatory M1 macrophages, and activated M1 macrophages. These data suggest that GM-CSF enhances macrophage responsiveness to HTN stimuli, contributing to HTN. Renal macrophages or GM CSF may be new therapeutic targets for HTN treatment.

49. BORRELIA BURGDORFERI BOSR-MEDIATED POST-

TRANSCRIPTIONAL REGULATION

Brittany L. Shapiro[1*], Prashant Jaiswal[1], Taylor Van Gundy[2], Sourav Roy[3], Brandon L. Garcia[3], Meghan Lybecker[2], Jenny A. Hyde[1], and Jon T. Skare[1]
Add an asterisk (*) to denote poster presenter

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The agent of Lyme disease, *Borrelia burgdorferi*, represents the most common vector-borne disease in the U.S.. To adapt to both infected mammals and the arthropod vector, dramatic changes in gene expression are required. The details of how *B. burgdorferi* carries this response out are still not clear. Recent data indicates that the borrelial regulatory protein BosR functions as a chaperone for small non-coding RNAs (sRNAs). We hypothesize that BosR-bound sRNAs recognize specific transcripts and provide an additional layer of post-transcriptional regulation needed for host adaptation. Characterization of BosR::sRNA::mRNA interactions should provide important insight into adaptive regulation operative in *B. burgdorferi*.

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50. GENETIC MODELING THE INTEGRATION OF BIOLOGICAL SEX, IMMUNITY, AND METABOLISM

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[1]Department of Cell Biology and Genetics, Texas A&M University School of Medicine, Bryan, TX, USA

ABSTRACT

Individuals in a population often show phenotypic variation in susceptibility to bacterial infection. Utilizing genetics and genomics, our lab's previous work in *Drosophila* highlighted that variation in infection susceptibility within populations is driven by energetic trade-offs, facilitated by immuno-metabolic signaling networks. We now show that biological sex and reproduction further shape immuno-metabolic responses to infection. Our preliminary data highlight that female reproductive tissues, part of a multi-tissue signaling network, influences immuno-metabolic responses and lipid-dependent energy resource allocation after bacterial infection. We hypothesize that sex-specific and mating-dependent changes in reproduction represent an evolutionary strategy to enhance population defenses against infection.

51. DEVELOPMENT OF A NOVEL NON-SECRETABLE FORM OF S100B AND ITS RELEVANCE TO PARKINSON'S DISEASE

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ABSTRACT

Parkinson's disease (PD) is a devastating neurodegenerative disorder with no known cure. PD is associated with an increase in secreted S100B, a protein ubiquitously expressed by astrocytes. We previously showed that extracellular S100B inhibits A-type voltage-gated potassium channels in cultured dopaminergic (DA) neurons, thereby pathologically increasing L-type voltage-gated calcium channel-mediated calcium fluxes. Here, we develop a novel adeno-associated virus that expresses non-secretable S100B with the goal of asking if S100B mediates pathological effects on DA neurons in vivo via an extracellular mechanism. This study has important implications for understanding the role of astrocytic S100B in the development of early PD.

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52. GUT METABOLITE TREATMENT REDUCES METABOLIC DYSFUNCTION AND PROMOTES FUNCTIONAL RECOVERY AFTER SPINAL CORD INJURY

Ashley J. Douthitt[1*] and Cédric G. Geoffroy[1]

[1]Department of Neuroscience and Experimental Therapeutics, Texas A&M University, School of Medicine, Bryan, TX, USA

ABSTRACT

Spinal cord injury (SCI) not only causes loss in motor function, but also disrupts neural circuitry and signals to vital organs, resulting in severe long-term complications outside of the central nervous system. Trauma to the spinal cord and disruption of cellular signaling results in alteration of the metabolite expression profile. The present study evaluates the therapeutic potential of microbiota-derived metabolites in a mouse model of thoracic SCI. Daily oral

treatments reduced local inflammation, reduced the onset of metabolic dysfunction, and promoted functional recovery. Overall, suggesting that metabolite supplementation may be an effective strategy to reduce secondary damages resulting from SCI.

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53. AUTONOMIC REGULATION OF T-LYMPHOCYTE INFLAMMATION AFTER PSYCHOLOGICAL TRAUMA

Tatlock H. Lauten[1*], Emily C. Reed[1], Tamara Natour[1], Adam J. Case[1]

[1]Department of Psychiatry and Medical Physiology, Texas A&M University, College Station, Texas

ABSTRACT

Post-traumatic stress disorder (PTSD) is a psychiatric condition associated with systemic inflammation, heightened sympathetic activity, and increased cardiovascular disease risk. Physiological changes suggest immune system disturbances, however, precise mechanisms connecting PTSD to immunity remain unclear. Using a murine PTSD model, repeated social defeat stress (RSDS), that induces systemic inflammation such as interleukin 17A (IL-17A) and 22 (IL-22) from T-lymphocytes. Splenic denervation eliminates this inflammation, suggesting an autonomic role. Sympathetic inhibition during RSDS reduces blood pressure and heart rate, and mice lacking adrenergic receptors show diminished systemic levels of IL-17A and IL-22, supporting the sympathetic role in T-lymphocyte inflammation post trauma.

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54. ELUCIDATING THE ROLE OF GLUTAMATE DELTA-1 RECEPTOR (GluD1) IN PROTEIN KINASE C (PKC) DEPENDENT SYNAPTIC PLASTICITY IN THE DORSAL STRIATUM

Pooja Shree Chettiar[1*] and Shashank Dravid[1]

[1] Graduate Program in Medical Sciences, Department of Psychiatry and Behavioral Sciences, Texas A&M Health Science Center, College Station, Texas, USA

ABSTRACT

Protein Kinase C (PKC)–dependent synaptic plasticity is critical for learning and memory, but its mechanisms in the dorsal striatum remain elusive. Using brain slice electrophysiology, we observed plastic changes in NMDA receptor response upon PKC activation in the dorsal striatum

that were opposite to classical PKC-dependent plasticity in other brain regions (depression vs potentiation). These effects were GluD1-dependent since they were significantly diminished in GluD1 KO. We developed novel peptide molecules to replicate GluD1-dependent PKC-induced NMDA receptor plasticity and found that their local delivery into the dorsal striatum modulated cognitive flexibility and motor learning. These results identify a novel form of plasticity in neural circuits relevant to neuropsychiatric disorders.

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55. CHRONIC PAIN INTERVENTIONS: THERAPEUTIC TARGETING OF GLUTAMATE DELTA-1 RECEPTOR (GluD1) – CEREBELLIN-1 (Cbln1) SIGNALING AND AUTOPHAGIC PATHWAYS

Pooja Shree Chettiar[1*], Kishore Kumar S. Narasimhan[1], Siddhesh Sabnis[1] and Shashank Dravid[1]

[1] Graduate Program in Medical Sciences, Department of Psychiatry and Behavioral Sciences, Texas A&M Health Science Center, College Station, Texas, USA

Synaptic plasticity in central amygdala and spinal cord underlies chronification of pain but the underlying mechanisms are still poorly understood. We discovered that chronic pain models exhibit downregulation of glutamate delta 1 receptor (GluD1) and cerebellin 1 (Cbln1) signaling, leading to increased AMPA receptor activity and persistent pain. Both local injection into the brain and systemic intravenous administration of recombinant Cbln1 (rCbln1) provides sustained pain relief demonstrating therapeutic efficacy. Finally, our results suggest downstream regulation of autophagic signaling by GluD1 which regulate plasticity in chronic pain. These findings identify a promising strategy for chronic pain treatment by modulating central GluD1-Cbln1 signaling.

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56. ASTROCYTIC NMDARS IN THE DORSAL STRIATUM CONTROL FINE MOTOR SKILLS

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ABSTRACT

Recent studies have uncovered the expression and behavioral impact of astrocytic NMDA receptors, yet their role in regulating neural circuits remains unclear. We explored this by deleting

the GluN1 subunit from astrocytes in the AldhCreGluN1$^{flox/flox}$ model, observing significant impairments in fine motor skills. This impairment was also evident when astrocytic GluN1 was selectively ablated in the dorsal striatum. Electrophysiological studies revealed that the blocker DQP-1105, targeting GluN2C/2D receptors, reduced miniature excitatory postsynaptic currents, indicating that astrocytic NMDARs are crucial for basal neurotransmission regulation. Additionally, intranuclear DQP-1105 injections in dorsal striatum of wild-type mice impaired their fine motor skills, highlighting the receptors' significance in motor control.

57. THE DYNAMICS OF LNCRNA NEAT-1 AND THE PARASPECKLE IN REGULATING THE INNATE IMMUNE RESPONSE

Kaitlyn S. Armijo[1]*, Sikander Azam[1], Chi G. Weindel[1], Morgan J. Chapman[1], Alice Devigne[2], Schinichi Nakagawa[3], Testsuro Hirose[4], Susan Carpernter[2], Robert O. Watson[1], Kristin L. Patrick[1]

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ABSTRACT

Phase-separated particles can form and maintain a boundary for compartmentalization and sequestration, regulating select biomolecules in or out of their microenvironment (Ismail et al., 2021). Nuclear paraspeckles are phase-separated particles have been shown to respond to cellular stress (Bond & Fox, 2009; West et al., 2016). While activation of this innate immune gene expression requires redistribution of transcription factors and RNA processing machinery, the mechanism through which this reorganization occurs remains unknown. We hypothesize that nuclear membraneless organelles (MLOs) like paraspeckles help the nucleus regulate the transcription of innate immune genes in response to pathogen sensing in macrophages.

58. IMPLICATIONS OF THE BOSR REDOX STATE IN *BORRELIA BURGDORFERI*

Kennedy Coleman[1*], Prashant K. Jaiswal[1], Sourav Roy[2], Brandon L. Garcia[2], Jon T. Skare[1], & Jenny A. Hyde[1]

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ABSTRACT

Borrelia burgdorferi undergoes dynamic regulation to adapt to the mammalian host that is in part reliant on the borrelial oxidative stress regulator (BosR) for survival. This unique metalloregulatory protein is known to bind DNA and alter the B. burgdorferi transcriptional response through mechanisms that are not fully understood. This study investigates conformational function of the protein in oxidized and reduced environments that allows BosR to differentially regulate gene expression. Our data suggest a novel regulatory mechanism used by B. burgdorferi BosR relative to other known bacterial metalloregulatory proteins.

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59. PATHOGENESIS OF DUCHENNE MUSCULAR DYSTROPHY IS ASSOCIATED WITH DECREASED LYMPH TRANSPORT AND INFLAMMATORY LYMPHANGIOGENESIS IN SKELETAL MUSCLE

Shedreanna Johnson[1*], Bhuvaneshwaran Subramanian[1], Akshaya Narayanan[1] Wei Wang[1], Joseph M. Rutkowski[1], Bonnie Seaberg[2], Alexandria Aceves[3], Sarah E. Frazier[3], Alexis Rutledge[3], Jay Griffin[3], Peter Nghiem[3], Mendell Rimer[2], Mariappan Muthuchamy[1]

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ABSTRACT

Duchenne Muscular Dystrophy (DMD), affecting 1 in 5000 males due to an X-linked mutation of the DMD gene, manifests progressive disability. The absence of functional dystrophin leads to sustained inflammation during muscle contraction, exacerbating tissue damage. Investigating D2.mdx mice and golden retriever muscular dystrophy (GRMD), micro-

lymphangiography revealed reduced lymph transport. Gadolinium-enhanced MRI confirmed diminished lymph transport in GRMD dogs. Isolated mouse flank lymphatic vessels exhibited decreased diastolic diameter, contractile frequency, and ejection fraction in 8-week-old D2.*mdx* mice. Elevated inflammatory markers and lymphatic markers in specific muscles, along with pathological lymphangiogenesis, indicate a link between compromised lymphatic function and DMD progression.

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60. THE ROLE OF BETA ADRENERGIC SIGNALING IN SPLENIC T-LYMPHOCYTE INFLAMMATION FOLLOWING PSYCHOLOGICAL STRESS

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ABSTRACT

Post-traumatic stress disorder (PTSD) exhibits heightened systemic inflammation and autonomic dysregulation. Our group has recapitulated the psychological trauma-induced inflammatory phenotype in a pre-clinical murine model of PTSD known as repeated social defeat stress (RSDS). Targeted splenic nerve denervation was able to completely prevent splenic T-lymphocyte inflammation after RSDS, suggesting the T-lymphocyte response to psychological trauma is neural derived as opposed to circulating. Both pharmacological blockade and genetic knockouts of beta-adrenergic receptors were insufficient to reverse the inflammation, and even potentiated it in some cases. Therefore, additional studies are warranted to understand the source that drives stress-induced T-lymphocyte inflammation.

61. SPLICING INHIBITOR PLADIENOLIDE B CAUSES DEFECTS IN INNATE IMMUNE GENE EXPRESSION

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ABSTRACT

Pladienolide B (PlaB), a potential cancer therapeutic, is a splicing inhibitor that blocks the activity of the splicing factor SF3B1. Motivated by recent research that links splicing inhibition with enhanced inflammatory gene expression, we set out to understand how PlaB impacts innate immune gene expression in macrophages. Using RNA-seq, we identified hundreds of innate immune genes whose transcript abundance is altered by PlaB treatment. This work demonstrates how the innate immune response is regulated at the level of pre-mRNA splicing and furthers our understanding of how PlaB may impact inflammation and the ability to fight infection in cancer patients.

62. DEFINING A ROLE FOR ATP CITRATE LYASE AS AN IMMUNE METABOLIC SENSOR

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ABSTRACT

ATP Citrate Lyase (ACLY), a metabolic enzyme which catalyzes the conversion of citrate to acetyl-CoA, functions canonically in the cytosol and noncanonically in the nucleus. Exploiting *in vivo* Drosophila genetics, our new findings demonstrate that ACLY relocates from the nucleus to the cytosol in specific immune cells after bacterial infection, driven by changes in citrate metabolism. Relocalization of ACLY likely shifts acetyl-CoA production from the nucleus, where it aids chromatin maintenance, to the cytosol in order to support energy production during infection. We propose that ACLY is an immune-metabolic sensor and a critical molecular regulator of infection responses.

63. UNDERSTANDING THE MECHANISM OF SEC14 MEDIATED LIPID EXCHANGE

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ABSTRACT

Phosphoinositide signaling represents a major intracellular signal transduction system in all eukaryotic cells. Phosphatidylinositol (PtdIns) transfer proteins (PITPs) are central regulators of phosphoinositide signaling from yeast to mammals. These proteins execute an unusual energy-independent lipid exchange cycle that is coupled to the activation of PtdIns 4-OH kinase to produce the essential lipid PtdIns-4-phosphate. Thus, understanding the PITP lipid exchange cycle is key to understanding how phosphoinositide signaling is regulated in cells. Sec14 represents the major yeast PITP and, through the use of ^{19}F NMR and X-ray crystallography, we probe the local conformational dynamics that occur during lipid exchange activity.

NEUROSCIENCE

Poster Presentation Abstracts #64 – 74

64. OLIGODENDROGLIAL EXPRESSION OF THE ALZHEIMER SUSCEPTIBILITY GENE BIN1 MODULATES NEURONAL TAU PATHOLOGY AND NEUROINFLAMMATION

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ABSTRACT

BIN1 (bridging integrator 1) is the second most common genetic risk locus for late-onset Alzheimer's disease (LOAD), the leading cause of dementia. We recently demonstrated that *BIN1* is highly enriched in oligodendrocytes (OLGs), in the uncompact regions of myelin sheaths along axons. Given that white matter dystrophy occurs pre-symptomatically in LOAD patients, our finding raises the possibility that OLG/myelin associated BIN1 may regulate neuronal health and the long-term integrity of axons. To investigate the involvement of oligodendroglial Bin1 in AD pathology we employed mice expressing human mutant Tau P301S and a conditional Cre-Lox-P system for *Bin1* deletion in OLGs.

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65. TARGETING NORADRENERGIC SIGNALING TO MITIGATE BONE LOSS AFTER SPINAL CORD INJURY

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ABSTRACT

At least 80% of people living with spinal cord injury (SCI) have osteoporosis or osteopenia, with loss of approximately 40% of trabecular bone volume below the level of injury in the first 2 years, leaving them at increased risk of fractures and significant post-fracture complications. Treating SCI-induced osteoporosis is an unmet medical need. We hypothesize that increased noradrenergic signaling after injury may underly SCI-induced osteoporosis. We have administered norepinephrine and targeted adrenoceptor subtypes to moderately injured rats and found that targeting adrenergic signaling may be effective for reducing bone resorption post-SCI.

66. HUMAN ELECTROCORTICAL DYNAMICS DURING VISUALLY GUIDED LOCOMOTION IN PROJECTED VIRTUALLY REALITY

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ABSTRACT

Human locomotion requires the integration of external environmental cues and internal sensory feedback when navigating complex environments. Our understanding of human brain processes during walking in the real-world is relatively limited, but immersive virtual reality provides the experimental control and nearly unlimited possibilities for creating immersive environments for studying human locomotor behaviors. Combined with three-dimensional motion capture, mobile high-density electroencephalography (EEG), and video-recorded eye tracking goggles, we were able to study human gaze behavior, electrical brain activity, and lower limb motions while human participants walked along a realistic path with virtual obstacles to step over. We identified contrasting human electrocortical dynamics during unobstructed treadmill walking compared to visually tracking and stepping over obstacles in the environment. These findings are crucial for belter understanding neuromotor control of human gait and balance, and factors that may increase fall susceptibility.

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67. CIRCADIAN MODULATION OF *IN VIVO* BASAL DOPAMINE RELEASE IN THE MESOLIMBIC PATHWAY

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ABSTRACT

The release of dopamine (DA) in the mesolimbic pathway is a necessary driver for motivated behaviors. Here, DA neurons in the ventral tegmental area (VTA) synaptically project onto the nucleus accumbens (NAc). Although DA is well studied in the context of reward, it is unknown how circadian rhythms in DA release influence DA dynamics. Using long-term fiber photometry

and virally-injected DA sensors into the NAc, we demonstrate in mice that baseline DA release is circadian and that reward-evoked DA peaks at dawn. Future work aims to investigate the role of stress in the rhythmic release of DA.

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68. Aβ Drives Corticostriatal Hyperactivity and Cholinergic Dysfunction to Impair Cognitive Flexibility in Early Alzheimer's Disease

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ABSTRACT

Alzheimer's disease (AD) is characterized by amyloid-beta (Aβ) accumulation and cognitive deficits, the mechanisms of which remain poorly understood. We discovered that 5xFAD mice exhibit reversal learning deficits in instrumental tasks. We observed hyperactivity in the medial prefrontal cortex (mPFC) neurons and their heightened outputs to striatal direct-pathway medium spiny neurons. This resulted in reduced cholinergic activity and acetylcholine release in 5xFAD mice. Prolonged chemogenetic inhibition of mPFC neurons reduced Aβ accumulation, normalized glutamatergic transmission, elevated striatal acetylcholine levels, and improved cognition in 5xFAD mice. Our results provide insights into the neural circuit mechanisms underlying Aβ-induced cognitive flexibility deficits in AD.

69. MITIGATING NEUROGENIC AND COGNITIVE CHANGES AFTER SPINAL CORD INJURY THROUGH ADMINISTRATION OF NATURALLY OCCURRING GUT METABOLITES

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ABSTRACT

Traumatic spinal cord injury (SCI) effects over 300,000 Americans, with about 18,000 new incidences each year. The most visible and well-studied result of SCI is paralysis, leaving countless other adverse changes after SCI affecting patient's quality of life yet to be studied in depth. One of these changes is SCI induced cognitive decline, with over 64% of patients experiencing a loss in cognitive function and an increased risk for dementia in aging. Another change in the system after SCI, is irregular expression of gut metabolites. Some of these metabolites are known to be neuroprotective, are therefore of interest in functional recovery. I propose that the mechanism of this cognitive decline is a decrease in adult hippocampal neurogenesis, and that through treatment with neuroactive gut metabolites locomotor and cognitive decline can be mitigated. There are currently no noninvasive clinical treatments for SCI, and in this preclinical test, and in this study, we show that oral treatment with naturally occurring gut metabolites can mitigate adult hippocampal neurogenesis changes post injury and improve cognitive outcomes.

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70. COMPENSATION AND NEUROTROPHIC SIGNALING IN *DROSOPHILA* AFTER MOTOR DEATH

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ABSTRACT

In injury, loss of neurons disrupts the circuit and leads to many adverse outcomes. Synaptic plasticity is a major mechanism that can restore lost function in a circuit. To determine how

different neuron populations contribute to circuit recovery, we will distinguish between the tonic and phasic firing motor neurons (MNs) in the *Drosophila* motor system. After neuron ablation, function recovery and synaptic plasticity will be evaluated by analyzing the crawling behavior and morphological changes in the dendrites and neuromuscular junctions (NMJs) of remaining motor neurons. *Drosophila* neurotrophic factor 1 (DNT-1) signaling, a homolog of BDNF, will be investigated as a mechanism for triggering post injury synaptic plasticity. Overall, this study will give us a deeper understanding of circuit recovery mechanisms mediated by synaptic plasticity.

71. DOWNREGULATION OF PEPTIDE Lv (PLv) ALTERS RETINAL FUNCTION AND STRUCTURE

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ABSTRACT

Peptide Lv, a small (~40 amino acids) endogenous secretory peptide, is expressed in various organs including the retina, vascular endothelium, and brain. PLv augments the protein expression and current densities of the L-type voltage-gated calcium channels in photoreceptors. Given the novelty of PLv, our present understanding of its diverse functions is still premature. Using electroretinogram, PLv$^{-/-}$ mice show smaller retinal light responses as compared to wildtype littermates. H&E staining depicts increased retinal thinning in PLv$^{-/-}$ mice. This highlights PLv's potential role in maintaining neuro-retinal integrity. Our goal is to investigate the longitudinal changes in the neurovascular retina associated with PLv-deficiency.

72. POST-INJURY DEPLETION OF NEUTROPHILS IMPAIRS LONG-TERM FUNCTIONAL RECOVERY IN A SEX-DEPENDENT MANNER AFTER SPINAL CORD INJURY

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ABSTRACT

Following spinal cord injury (SCI), an inflammatory cascade ensues that can impair long-term functional recovery. Neutrophils, the most abundant circulating leukocytes in humans, are the first immune cells to infiltrate and have long been considered to exacerbate tissue damage and functional deficits post-SCI. We found that antibody-mediated neutrophil depletion performed 1-day prior to SCI had no significant impact on locomotor, however, neutrophil depletion immediately post-injury significantly impaired long-term hindlimb locomotor recovery in male but not female mice. Collectively, our findings indicate a sex-dependent and temporally-restricted role for neutrophils in promoting long-term recovery following SCI.

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73. OPTOGENETIC INHIBITION OF HIPPOCAMPAL-PREFRONTAL PROJECTIONS FACILITATES FEAR EXTINCTION IN RATS

Samantha L. Plas[1,2*], Kennedi L. Crayton[2], Stephen Maren[1,2]

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ABSTRACT

The ventral hippocampus (VH) is a major source of excitatory input to the infralimbic cortex (IL) and is implicated in extinction learning and retrieval. Our lab demonstrated that VH projections to IL mediates renewal, a form of fear relapse. Further, we demonstrated that activation of VH→IL projections impair extinction retrieval. Electrophysiology revealed that excitatory VH projections

recruit IL inhibitory neurons to inhibit IL principal cell activity. Collectively, these suggest that excitatory VH→IL projections oppose extinction retrieval and undermine extinction learning. To test this, we used optogenetics to inhibit VH terminals in the IL during extinction of auditory fear conditioning.

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74. NEURONAL ACTIVITY IN THE THALAMIC NUCLEUS REUNIENS DURING THE CONDITIONING AND EXTINCTION OF FEAR IN MALE AND FEMALE RATS

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ABSTRACT

The nucleus reuniens (RE) is a thalamic structure that interconnects the medial prefrontal cortex and the hippocampus via bidirectional connections. We have recently identified a critical role for the RE in the extinction of fear memory. However, the learning-related responses of RE neurons during fear conditioning and extinction has not been performed. To address this, we recorded calcium transients and electrophysiological single-unit responses of RE neurons during both auditory fear conditioning, extinction, and extinction retrieval in male and female Long-Evans rats. Results reveal that there is heterogeneity in RE neuronal activity during extinction and that the RE plays an important role in suppressing conditioned fear responses.

NUTRITION

Poster Presentation Abstracts #75 – 84

75. TGF-β1 SIGNALING IMPAIRS METFORMIN ACTION ON GLYCEMIC CONTROL

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ABSTRACT

Type 2 diabetes (T2D) is marked by high blood sugar levels, commonly treated with Metformin, which lowers liver glucose production. However, T2D patients often develop Metformin resistance. Our study found that high Transforming Growth Factor Beta 1 (TGF-β1) levels in T2D disrupt Metformin's function by blocking AMPK phosphorylation. Reducing TGF-β1 improves Metformin's blood sugar control in obese, high-fat diet mice and in a mouse model resistant to hepatic insulin due to insulin receptor substrate deletion. Using LY2157299, a TGF-β1 inhibitor, also increases Metformin sensitivity. Thus, targeting hepatic TGF-β1 might enhance Metformin's efficacy, offering a potential combination therapy for T2D.

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76. EFFICACY AND SAFETY OF PROBIOTICS FOR THE PREVENTION OF CLOSTRIDIOIDES DIFFICILE INFECTION IN ADULTS AND CHILDREN: A COCHRANE SYSTEMATIC REVIEW AND META-ANALYSIS

Zahra Esmaeilinezhad[1], Nirjhar R. Ghosh[1], Bradley C. Johnston[1,2]

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2. Department of Epidemiology and Biostatistics, School of Public Health, Texas A&M University College Station, Texas, USA.

ABSTRACT

Probiotics when given with antibiotics may prevent Clostridioides difficile infection (CDI) and CD-associated diarrhea (CDAD). In an updated systematic review based on Cochrane Handbook methods, 8 new randomized clinical trials (RCTs) were identified on probiotics for CDI and CDAD prevention. Probiotics did not significantly reduce CDI. However, they reduced the risk of CDAD by 50% (38 RCTs), the more clinically relevant outcome. The absolute risk reduction was 1.5% (95%CI 0.38-0.64) based on moderate certainty evidence. We found no significant adverse events attributable to probiotics (37 RCTs). Overall, probiotics as an adjunct to antibiotics are safe and effective for CDAD prevention.

77. HEALTH RELATED VALUES AND PREFERENCES REGARDING SATURATED FAT INTAKE: A CROSS SECTIONAL MIXED-METHODS STUDY IN TEXAS

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ABSTRACT

Nutritional choices help prevent cardiovascular disease. In a cross-sectional survey with semi structured interviews, we explored participants' willingness to reduce saturated fat (SFA) intake after learning the potential impact of reduced SFA diets on the absolute risk reduction concerning heart attack based on a Cochrane review. Among 30 participants, 14 (46%) were unwilling to reduce their SFA intake, while 11 (37%) were somewhat willing and 5 (17%) were willing to reduce. Reasons for unwillingness included taste preferences and the low cost of SFA-rich foods. These results emphasize the importance of considering peoples' values and preferences when developing recommendations for SFA intake.

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78. EPIGENETIC REGULATION BY A LONG NONCODING RNA MIRNA CLUSTER IN METABOLIC DYSFUNCTION ASSOCIATED STEATOTIC LIVER DISEASE (MASLD)

Lauren Gladwell[1,2*], Sunil Venkategowda[2], and Mahua Choudhury [1,2]

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ABSTRACT

The concomitant burden of metabolic diseases is debilitating. More devastatingly, the prevalence of obesity, Type 2 Diabetes, and MASLD have only progressively increased. Genetics, diet, and exercise have been the main factors considered for this rise; however, the new frontier of epigenetics describes that there is more to the story. By altering the organization of chromatin, epigenetics links how the environment can impact gene expression

in a reversible and heritable nature. Our lab is the first group to discover that the long noncoding RNA DLEU2 and its hosted microRNAs miR15a/16-1 are involved in metabolic disease and may regulate chromatin modifications.

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79. LOSS OF OVARIAN HORMONE EXACERBATES DIET-INDUCED NAFLD

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ABSTRACT

Estrogen is a primary sex hormone which may be responsible for the prevalence disparity of NAFLD between females and males. In the present study, female wild type (WT) mice were subjected to either sham operation or ovariectomy (OVX), then fed a high-fat diet (HFD) together with age-matched male mice for 12 weeks. Liver steatosis and inflammation, as well as systemic insulin sensitivity were examined. By OVX and HFD feeding, we demonstrated that loss of ovarian hormone abolishes sex-based protective effects on diet-induced NAFLD in female mice. As such, enhancing estrogen actions may be viable for managing NAFLD in males.

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80. INTERMITTENT FASTING REGULATES MYELOID CELLS SYSTEMICALLY AND IN LIVER IN OLD MICE

Hyewon Han[1*], Da Mi Kim[1], Zeyu Liu[1], Hongying Wang, Jiyeon Noh[1], Yuxiang Sun[1]

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ABSTRACT

Intermittent fasting (IF) is known for its metabolic effect, however, its effect on immunity is unknown. In a 12-week study, we subjected both young and old mice to 24-hour IF. IF improved glucose tolerance and insulin sensitivity in both young and old. IF suppressed age-induced increase in pro-inflammatory monocytes in the circulation and pro-inflammatory polarization, as well as cytokine expressions in peritoneal macrophages. In peripheral tissues, only in the liver, IF decreased the age-induced increase in pro-inflammatory macrophages. In summary, IF promotes anti-inflammatory programming of myeloid cells in the circulation and liver of old mice but not in young mice.

81. GHS-R DEFICIENCY IN MACROPHAGE REPROGRAMS INNATE IMMUNE CELL POPULATION AND ALLEVIATES THE INFLAMMATION IN AGED MOUSE HEART

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ABSTRACT

GHS-R, pivotal in ghrelin signaling, intricately influences macrophage behavior. There is growing recognition that the innate immune system as a macrophage contributes to cardiac development, composition, and function. Therefore, we evaluated how GHS-R ablation in macrophages reprograms the innate immune cell population and affects the inflammation in the aged mouse heart. We found that GHS-R deficiency prompts a significant reduction in pro-inflammatory M1-like macrophages and a notable increase in anti-inflammatory M2-like macrophages while concurrently suppressing age-related pro-inflammatory gene expression. Our results suggest that GHS-R in macrophages is a potential therapeutic target for preventing aging-induced cardiac disease in the heart.

82. DAILY MANGO CONSUMPTION IMPACT COGNITIVE FUNCTION IN ADOLESCENTS: A RANDOMIZED, DOUBLE BLINDED, PLACEBO-CONTROLLED STUDY

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ABSTRACT

Based on previous studies, it was hypothesized that consuming pre-hydrolyzed mango gallotannins along with a probiotic will effectively reduce systemic inflammation and improve cognitive function in adolescent male and females. Healthy adolescent females and males consumed either 400g of mango treated with tannase plus one probiotic pill or 400g of mango

without tannase plus one placebo pill daily, for 60 days. Systemic cytokine levels were measured, and cognitive function was assessed using the NIH toolbox Dimensional Change Card Sort and List Sorting Working Memory tests. The present evidence illustrates trending associations between systemic inflammation, executive and cognitive functions.

83. INFLAMM-AGING IS ASSOCIATED WITH PRO-INFLAMMATORY PROGRAMMING OF INNATE IMMUNE CELLS IN THE COLON

Ji Yeon Noh[1*], Hongying Wang[1], Yuhua Z. Farnell[2], Xiao-Di Tan[3], Gus Wright[4], Andrew Hillhouse[4], Yuxiang Sun[1]

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ABSTRACT

Inflammaging is a chronic low-grade inflammation in aging, contributing to various age-associated diseases including inflammatory bowel disease (IBD). Gut hormone ghrelin is mainly produced by the gastrointestinal track, and it functions through its receptor Growth Hormone Secretagogue Receptor (GHSR). We hypothesize that GHSR in macrophages regulates gut barrier and experimental colitis in aging. Experimental colitis was induced in young and aged myeloid-specific GHSR knockout mice. GHSR inhibition in myeloid cells attenuated susceptibility to colitis and aging leaky gut in aging. Our results suggest that ghrelin signaling plays an important role in intestinal inflammation and barrier function in aging.

84. DIETARY VARIABILITY IN ADOLESCENT GIRLS OVER A TWO-YEAR PERIOD

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ABSTRACT

Diet quality variability in adolescents is not well understood. In a longitudinal study conducted across two sites (Kansas City, MO and Ithaca, NY), we assessed the Health Eating Index (HEI) and added sugar intake in 40 adolescents. Overall, mean HEI scores (<51) indicated poor diet quality across both sites. While both exhibited fluctuating added sugar intake, mean values decreased by 24 months. Notably, site-specific trends emerged. Kansas City adolescents experienced a qualitative decline in HEI (10.2%), suggesting a worsening dietary pattern. Conversely, Ithaca showed relative stability with a moderate decrease (3.2%). This suggests potential regional variations in adolescent dietary trajectories.

TOXICOLOGY

Poster Presentation Abstracts #85 – 98

85. BIPHASIC MODULATION OF STRIATAL CHOLINERGIC ACTIVITY BY DIRECT-PATHWAY NEURONS

Chen Ruifeng et al.

ABSTRACT

Unavailable

86. BIS-INDOLE DERIVED NUCLEAR RECEPTOR 4A (NR4A) LIGANDS ENHANCE TEMOZOLOMIDE CYTOTOXICITY IN GLIOBLASTOMA CELLS

Evan Farkas[1*], Keshav Karki[1], Gargi Sivaram[2], Jaclyn Reyes[2], Caitrina Kearns[2], Robert Rostomily[3], Andrei Mikeev[3], and Stephen Safe[1]

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ABSTRACT

Glioblastoma (GBM) is the most common form of adult brain cancer and the standard of care temozolomide (TMZ) and radiation therapy are subject to drug resistance. In-vitro studies with TMZ show that growth inhibition of human and mouse GBM cell lines can be in the low mM range. The orphan nuclear receptor 4A (NR4A) is highly expressed in GBM patients and their tumor growth is inhibited by a series of bis-indole derived compounds (CDIMs) that bind both receptors and act as inverse agonists. These CDIMS, synergistically sensitize tumor cells to TMZ treatment by regulating TMZ-resistance genes/gene products.

87. HAZARD CHARACTERIZATION AND GROUPING OF PFAS USING A COMPENDIUM OF HUMAN CELL LINES FORM DIFFERENT ORGANS

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ABSTRACT

Per- and poly-fluoroalkyl substances (PFAS) are widely used in manufacturing, thus are ubiquitous in environmental and human samples, yet there is a lack of data for majority of PFAS. We hypothesized that a compendium of human *in vitro* models can be used to identify groupings and potential cell-specific effects. We utilized a diverse set of 56 PFAS and tested them in concentration response using various *in vitro* models. Majority of PFAS were without effect, however, cell-specific responses were observed. We demonstrate that a panel of *in vitro* models can be used to rank and group PFAS to inform risk-based decision-making.

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88. DETECTING DIET-RELATED CHANGES IN MOUSE TISSUE COMPOSITION WITH RAMAN SPECTROSCOPY

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ABSTRACT

The rapid rise of metabolic syndrome in the U.S., emphasizes the inadequacy of national dietary guidelines and a need for new methods of studying diet. In this study, we employed Raman spectroscopy investigate diet-related changes in mouse tissue composition, hypothesizing that Raman could not only detect variations in response to dietary changes but also accurately predict the specific diet followed by each mouse over preceding months. Our study found that Raman could differentiate diets, detect nutritional deficiencies, and discern age-related changes in tissue composition. These findings lay the groundwork for future applications in metabolic studies and personalized health monitoring.

89. COMPARISON OF IN VITRO MODELS TO PREDICT AIRWAY TOXICITY FROM DIESEL EXHAUST PARTICULATE MATTER

Olivia Lampe et al.

ABSTRACT

UNAVAILABLE

90. PREDICTING RENAL CLEARANCE OF PFAS WITH A HUMAN KIDNEY PROXIMAL TUBULE TISSUE CHIP AND A NOVEL PHYSIOLOGICALLY-BASED KIDNEY MODEL

Hsing-Chieh (Candice) Lin[1*†], Courtney Sakolish[1†], Haley L. Moyer[1], Stephen S. Ferguson[2], Jason P. Stanko[2], Ivan Rusyn[1], Weihsueh A. Chiu[1]

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ABSTRACT

PFAS are a class of chemicals that have concerns for human health in part due to many of them having long half-lives. It is believed that many of these chemicals can be reabsorbed in renal proximal tubules, making it difficult for them to be excreted, but there are limited approaches to estimate renal clearance (CL_{renal}) of PFAS. In vitro models, coupled with physiologically-based in vitro-to-in vivo extrapolation (IVIVE), could potentially be a useful method. We developed human proximal tubule systems, by using Transwells without or with dynamic flow, to predict CL_{renal} of three PFAS: one with a long half-life (PFOS) and two with shorter half-lives (PFBS and PFHxA). Our predictions for human CL_{renal} of PFAS were correlated highly with available values from in vivo human studies. We conclude that our in vitro-in silico method can distinguish between PFAS with lower and higher clearances, and thereby help prioritize PFAS with greater potential for human health concern.

91. IMPROVING PROBABILISTIC RISK ASSESSMENT OF SUPERFUND PRIORITY CHEMICALS USING BAYESIAN BENCHMARK DOSE MODELING AND NEW APPROACH METHODOLOGIES (NAMS) FOR HUMAN POPULATION VARIABILITY

En-Hsuan Lu[1*], Lucie C. Ford[1], Ivan Rusyn[1], Weihsueh A. Chiu[1]
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ABSTRACT

Recent efforts to redefine traditional toxicity values as a probabilistic risk-specific dose reveal two key uncertainties in risk assessment: POD estimates and extent of human variability. To improve accuracy and precision of toxicity values, we integrated Bayesian benchmark dose modeling and NAM-based toxicokinetic and toxicodynamic data. Following the WHO/IPCS framework for probabilistic risk assessment, we derived refined estimates for the target human dose for magnitude of effect M and population incidence I (HD_{MI}) for 19 Superfund priority chemicals. Our approach yielded more protective HD_{MI} values for most chemicals, and the incorporation of chemical-specific NAM-based data increased precision of toxicity values.

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92. TRENDS AND DISPARITIES IN URINARY BLADDER CANCER INCIDENCE AND MORTALITY IN TEXAS AND THE US: 2006-2020

Kayla Morales*, Nishat Tasnim Hasan, Natalie Johnson, Taehyun Roh

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ABSTRACT

Urinary bladder cancer is the sixth most frequently diagnosed cancer in the US. Understanding the trends and disparities is crucial for effective prevention and intervention strategies. The objective of this study was to evaluate trends in the incidence and mortality of urinary bladder cancer, assess disparities across various characteristics, and compare trends between Texas and US populations. Our study highlights significant variations in urinary bladder cancer incidence and mortality rates in Texas and the US, emphasizing the importance of understanding the underlying causes and implementing targeted interventions to address these disparities.

93. EVALUATING MECHANISTIC UNDERPINNINGS OF ENVIRONMENTAL CHEMICAL EFFECTS ON FETO-MATERNAL INTERFACE USING A HUMAN ORGAN-ON-CHIP MODEL

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ABSTRACT

Preterm birth is linked to high risk of infant mortality and adverse health outcomes later in life. A number of hypotheses have been proposed to explain the etiology of preterm birth, including exposure to environmental contaminants. This study used a Fetal-Maternal interface organ-on-chip (FMi-OOC) model to test the hypothesis that polybrominated diphenyl ether 47 (PBDE-47), perfluorooctanoic acid (PFOA), and dichlorodiphenyltrichloroethane (DDT), chemicals linked to preterm birth in epidemiological studies, cause cell death and inflammation predisposing to preterm birth. This study provides important data using a FMi-OOC to support linkages between environmental exposures and fetal membrane-associated changes linked to preterm birth.

94. ADSORPTION AND DETOXIFICATION OF DEOXYNIVALENOL BY NATURAL CLAYS

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ABSTRACT

Deoxynivalenol (DON), an end-product of several Fusarium species, ranks among the foremost mycotoxins in cereal crops and is a prevalent contaminant in animal feed. Its contamination poses significant health risks to both humans and livestock. Edible clays exhibit promises as potential therapeutic agents in the diet of animals and humans, potentially mitigating short-term exposure and reducing the toxicity associated with DON. This study investigated the sorption mechanisms of DON onto the active surfaces of various clays and activated carbons. The safety and DON-detoxifying capacity of clays were affirmed through experiments involving DON-sensitive living organisms, *Hydra vulgaris* and *Lemna minor*.

95. USING *L. MINOR* AND *C.ELEGANS* TO VALIDATE THE EFFICACY OF SORBENT MATERIALS TO REMEDIATE REAL-LIFE SOIL SAMPLES

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ABSTRACT

To prevent toxicity to the ecosystem, the identification of hazardous soils and remediation is necessary. We used 2 organisms to test PAH-contaminated soil samples for toxicity. Reference materials that contained a mixture of priority pollutants served as positive controls. Results indicated that most samples cause <50% inhibition of growth parameters to the organisms. However, 3 samples caused severe inhibition of the measured parameters. The reference materials were the most toxic samples. The inclusion of sorbents decreased the toxicity of all samples. Our goal is to continue using these organisms as tools for biomonitoring and verifying the efficacy of remediation practices.

96. ASSOCIATION OF PDGF-BB WITH ARSENIC IN VITRO AND IN A SOUTH TEXAS POPULATION

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ABSTRACT

Exposure to arsenic (As) from drinking water is a worldwide public health concern where Texas is ranked number seven in the U.S. of people affected by arsenic contamination. Using a paired *in vitro* and human translational study of urine samples from participants in South Texas, we exposed uroepithelial cells to $NaAsO_2$ for 5-weeks to establish phenotypic endpoints. Platelet-derived growth factor (PDGF)-BB levels were significantly increased *in vitro*. Linear regression analysis showed associations between As levels from participants and urinary cytotoxicity and cytokine levels. Based on experimental and population-based data, PDGF-BB may be a biomarker of As effect reflecting low-level exposure.

97. A REFERENCE LIBRARY FOR SUSPECT SCREENING OF ENVIRONMENTAL TOXICANTS USING NONTARGETED ION MOBILITY SPECTROMETRY-MASS SPECTROMETRY ANALYSES

Devin Teri[1,2]*, Noor A. Aly[1,2], James N. Dodds[3], Alexandra C. Cordova[1,2], Erin S. Baker[1,3], Ivan Rusyn[1,2]

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ABSTRACT

Ion mobility spectrometry-mass spectrometry is an analytical technique that separates compounds based on mass-to-charge ratio and drift time, enabling the calculation of collisional cross section (CCS) values for molecular and isomeric distinctions. We utilized 4,000+ chemicals from the ToxCast Program to establish a comprehensive CCS database, which were analyzed via IMS-MS. CCS values were calculated using Agilent IM-MS Browser and manual verification. Approximately 50% of the chemicals were detected in at least one ionization mode, with CCS reproducibility within +/-1% Å². This database will be pivotal for high-throughput suspect screening, enabling rapid exposure and risk assessments of complex environmental samples.

98. A NEW APPROACH METHODS STRATEGY FOR RISK-BASED PRIORITIZATION OF PFAS

Han-Hsuan Doris Tsai[1,2], Lucie C. Ford[1,2], Zunwei Chen[1,2], Sarah D. Burnett[1,2], Allison N. Dickey[3], Fred A. Wright[1,3,4], Ivan Rusyn[1,2]

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ABSTRACT

Per- and polyfluoroalkyl substances (PFAS) comprise many chemicals that are persistent and some pose human health hazards. A strategy for PFAS grouping and prioritization is crucial. We evaluated whether structure-based grouping strategy can be substantiated using *in vitro* bioactivity data, and these data may be used for risk-based prioritization of PFAS, by assessing concentration response effects in human hepatocytes and cardiomyocytes for 26 structurally diverse PFAS. Few phenotypic effects were seen in hepatocytes, but negative chronotropy was observed for 8 PFAS. Substance- and cell-specific transcriptomic changes were pronounced, with little group-specific effects. Bioactivity and exposure ratio calculations indicated most PFAS had ratios>1. Our data challenges structure-based grouping approach for prioritization. Testing individual PFAS would be needed for decision-making. Our proposed strategy may be used for prioritization of PFAS.

BIOCHEMISTRY & MOLECULAR BIOPHYSICS

Oral (3MT) Presentation Abstracts #1-3

1. DECODING KILLER GENES

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ABSTRACT

Fiersviridae, small bacteriophages with ssRNA, typically possess three core genes and a single gene responsible for host lysis, leading to the release of viral progeny. Although the molecular mechanism of most single-gene lysis (Sgl) proteins remains unknown, the examination of type I Sgls has provided insights into crucial cell wall biosynthesis pathways targeted by antibiotics. Type I Sgls inhibit peptidoglycan (PG) synthesis, resembling cell wall antibiotics like fosfomycin and cycloserine, which abolish the supply of Lipid II, the universal substrate for PG biosynthesis. Studying novel Sgls provides an opportunity to deepen our understanding of current antibiotic targets or discover new ones.

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2. SMALL SIGNALING PEPTIDES REGULATE DEVELOPMENT IN *SORGHUM BICOLOR*

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ABSTRACT

Bioenergy Sorghum is a well-established bioenergy crop due to its long stem (3-5m), diploid genome, drought tolerance, and efficient C_4 photosynthesis. These traits allow Sorghum to sequester high amounts of atmospheric CO_2 in the form of complex carbohydrates like cellulose, starch, and sucrose which can be converted to bioethanol. Sorghum stems are the main organ harvested for biofuel production and its development is highly complex. Its regulation may be influenced by small signaling peptides. I use computational approaches to find small signaling peptide candidates in sorghum and test their function *in-vivo* via novel injection method.

CONFORMATIONAL ENTROPY IN ANTIBODY AFFINITY MATURATION

Weimin Tan[1]*, Taylor R. Cole[1], Anthony C. Bishop[1] and A. Joshua Wand[1]

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ABSTRACT

Antibodies are one of the most important components of our immune system and they are one of the fastest growing therapeutics. Antibodies go through a process called affinity maturation during an immune response to generate antigen-specific antibodies of high affinity. In drug discovery, therapeutic antibodies affinity maturation is carried out by directed evolution. The change in antibody flexibility and conformational change has been indicated to be significant for affinity improvement. We use Nuclear Magnetic Resonance (NMR) relaxation experiments to determine changes in antibody side chain and backbone flexibility and the corresponding contributions of conformational entropy to antigen binding. Our goal is to investigate the differences in conformational entropy in antibody during affinity maturation.

GENETICS & GENOMICS

Oral (3MT) Presentation Abstracts #4-6

4. FLOWER POWER AND GENETIC DIVERSITY: DEVELOPING METHODS FOR RECOMBINATION ANALYSIS IN COTTON

Serina M. DeSalvio

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ABSTRACT

Genetic diversity is vital to crop improvement. My research focuses on increasing the genetic diversity in cultivated cotton. I'm doing this by characterizing and manipulating recombination rates. Nearly half of the cotton genome is occupied by regions of both low recombination, and low genetic diversity. First, we are seeking to better understand the restraints on recombination in the cotton genome in post-meiotic tissue. After that, we can think about altering the recombination landscape through things like gene editing. This work will provide a foundation for future recombination studies and insights for how we can continue to improve cultivated cotton.

5. THE ROLE OF LIPID PEROXIDES IN *Mycobacterium tuberculosis* INFECTION

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ABSTRACT

During infection with pathogens such as Mycobacterium tuberculosis (Mtb), reactive oxygen species (ROS) accumulate. ROS can lead to the oxidation of other macromolecules, such as proteins, DNA, and lipids, damaging cellular components and impairing normal functions. Lipid peroxides pose a threat to cellular functioning due to the vital role lipids play in maintaining the integrity of the plasma membrane. Increased lipid peroxides are associated with poor outcomes in Mtb infection and with lytic cell death in Mtb-infected macrophages. When macrophages are infected with Mtb, we showed that oxidized lipids aggregate and colocalize with Mtb bacilli. Motivated by this novel finding, we are working to explore further how lipid oxidation influences host outcomes during Mtb infection.

6. UNDERSTANDING DNA DOUBLE-STRAND BREAK (DSB) REPAIR FOR A SELF-ELIMINATING TRANSGENE IN THE MAJOR DENGUE VECTOR, *Aedes aegypti*

Joseph S. Romanowski

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ABSTRACT

The *Aedes aegypti* mosquito is a human disease vector responsible for over 400 million dengue virus infections each year. *Aedes* genetic control approaches, such as homing gene drives, use cellular DNA double-strand break (DSB) repair mechanisms to achieve super-Mendelian inheritance of transgenes that modify or suppress populations. Safeguard mechanisms to remove gene drives, however, are not well reported. Here, we use CRISPR-Cas9 and Oxford Nanopore sequencing on a self-eliminating transgene to investigate the effects of DSB location and nuclease on the single-strand annealing DSB repair pathway, a mechanism capable of removing multi-kilobase gene drive transgenes from the *Aedes aegypti* genome.

MEDICAL SCIENCES

Oral (3MT) Presentation Abstracts #7 - 9

7. AN ADENO-ASSOCIATED VIRUS (AAV)-BASED TOOL TO SPECIFICALLY DAMAGE MTDNA IN ASTROCYTES WITHIN PRE-SPECIFIED REGIONS OF THE ADULT MOUSE BRAIN

Daniela A. Ayala[1]*, Anthony J. Matarazzo[1], Bonnie Seaberg[1], Mendell Rimer[1], Rahul Srinivasan[1,2].

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ABSTRACT

Astrocytes display robust mitochondrial Ca2+ influx in live dorsolateral striatum (DLS) slices, suggesting their active role in neuronal function. We proposed disrupting astrocyte mitochondrial function within specific brain regions triggers neuronal dysfunction and accelerated neurodegeneration. Using an AAV expressing the restriction enzyme PstI (Mito-PstI) under the astrocyte GfaABC1D promoter, targeted specifically to astrocytic mitochondria, we induced damage to astrocytic mitochondrial DNA (mtDNA). Mito-PstI introduction to mouse DLS, a Parkinson's disease (PD)-related region, resulted in altered mitochondrial function and structure, along with changes to astrocyte reactivity. This AAV tool offers valuable insights into neurodegeneration acceleration, particularly in the context of PD.

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8. THE *MYCOBACTERIUM TUBERCULOSIS* SECRETED PROTEIN RV1075C MANIPULATES HOST HISTONE METHYLTRANSFERASES TO PROMOTE INFECTION

Aja K. Coleman[1*], Allison Wagner[1], Haley M. Scott[1], Robert O. Watson[1], and Kristin L.

Patrick[1] [1] Department of Microbial Pathogenesis and Immunology, Texas A&M Health, Bryan, TX, USA

ABSTRACT

Mycobacterium tuberculosis (Mtb) is one of the most infectious and deadly pathogens worldwide. An *in silico* screen identified a putative nuclear localization signal in the Mtb

protein Rv1075c. We confirmed that Rv1075c traffics to the macrophage nucleus and biochemically associates with chromatin. We also found that expression of Rv1075c hyperinduces expression of *Ifnb1* and *ΔRv1075c* Mtb fail to induce early type I IFN responses during infection of primary murine macrophages. Together, our data argues that Rv1075c plays a critical role in helping Mtb establish a favorable niche by promoting a pro-Mtb gene expression program during macrophage infection.

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9. BORRELIA BURGDORFERI BOSR-MEDIATED POST-TRANSCRIPTIONAL REGULATION

Brittany L. Shapiro[1*], Prashant Jaiswal[1], Taylor Van Gundy[2], Sourav Roy[3], Brandon L. Garcia[3], Meghan Lybecker[2], Jenny A. Hyde[1], and Jon T. Skare[1]
Add an asterisk (*) to denote poster presenter

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The agent of Lyme disease, *Borrelia burgdorferi*, represents the most common vector-borne disease in the U.S.. To adapt to both infected mammals and the arthropod vector, dramatic changes in gene expression are required. The details of how *B. burgdorferi* carries this response out are still not clear. Recent data indicates that the borrelial regulatory protein BosR functions as a chaperone for small non-coding RNAs (sRNAs). We hypothesize that BosR-bound sRNAs recognize specific transcripts and provide an additional layer of post-transcriptional regulation needed for host adaptation. Characterization of BosR::sRNA::mRNA interactions should provide important insight into adaptive regulation operative in *B. burgdorferi*.

NEUROSCIENCE

Oral (3MT) Presentation Abstracts #10 - 12

10. BRAINSTEM-AMYGDALA INTERACTIONS LEAD TO PREFRONTAL CORTEX INHIBITION AND IMPAIR EXTINCTION LEARNING

Hugo Bayer[1,2*], Annalise Binette[1,2], and Samantha Sweck[1,2], James Hassell Jr.[2], Vitor Juliano[2,3], Kennedi L. Crayton[2], Carolina D. Munhoz[3], Stephen Maren[1,2]

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ABSTRACT

Extinction learning is the basis of exposure therapy, which is used to treat disorders such as post-traumatic stress disorder. Under stress, the amygdala suppresses the medial prefrontal cortex (mPFC), and this is thought to underlie extinction impairments. However, the upstream circuits that activate the amygdala under stress are not well understood. In this study we explored how norepinephrine projections from the locus coeruleus (LC) to the basolateral amygdala (BLA) promote extinction deficits under stress. We used chemogenetics, fiber photometry and immunohistochemistry and found out that LC activation and stress increases activation of BLA cells that project to the mPFC.

11. SEX DIFFERENCES IN NOVEL TRANSGENIC MICE WITH CONSITUTIVELY UPREGULATED B2* NEURONAL NICOTINIC ACTEYLCHOLINE RECEPTORS: IMPLICATIONS FOR PARKINSON'S DISEASE

Gauri Pandey[1,2*], Sara M. Zarate[2], Roger Garcia[2], and Rahul Srinivasan[1,2]

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ABSTRACT

Nicotine reduces Parkinson's disease (PD) risk, however, nicotine concentrations in tobacco users cannot activate neuronal nicotinic acetylcholine receptors (nAChRs), making this an unlikely mechanism for neuroprotection of dopaminergic (DA) neurons. We showed that nanomolar concentrations of the nicotinic ligand, cytisine chaperone β2-subunit-containing nAChRs out of the endoplasmic reticulum (ER), thereby reducing ER stress. Here, we create a transgenic mouse line with enhanced ER export of β2 nAChRs. β2-mutant mice demonstrated significant increase in Sec24D ER exit sites (ERES) within substantia nigra pars compacta DA neurons of only female mice. This was accompanied by reduced motor deficits in parkinsonian mice.

12. NEUTROPHIL EXTRACELLULAR TRAPS IN SPINAL CORD INJURY

Shelby K. Reid[1,2*], Miranda E. Leal[2], Megan A. Kirchhoff[2], and Dylan A. McCreedy[1,2]

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ABSTRACT

Neutrophils are a common immune cell that crosses the blood-spinal cord barrier within hours after spinal cord injury (SCI). Using ELISA and flow cytometry, we defined the timeline of neutrophil extracellular traps (NETs)—a neutrophil effector function—in a mouse model of SCI. To assess NETs' role in long-term recovery after SCI, we treated mice with an anti-NET therapeutic (human recombinant DNase I) and used behavioral assays and histochemistry to evaluate long term tissue damage and motor recovery. Our data indicates the therapeutic potential of targeting NETs after SCI to support functional recovery.

NUTRITION

Oral (3MT) Presentation Abstracts #13 - 15

13. EPIGENETIC REGULATION BY A LONG NONCODING RNA-MIRNA CLUSTER IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE (MASLD)

Lauren Gladwell[1,2], Sunil Venkategowda[2], and Mahua Choudhury [1,2]

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ABSTRACT

The concomitant burden of metabolic diseases is debilitating. More devastatingly, the prevalence of obesity, Type 2 Diabetes, and MASLD have only progressively increased. Genetics, diet, and exercise have been the main factors considered for this rise; however, the new frontier of epigenetics describes that there is more to the story. By altering the organization of chromatin, epigenetics links how the environment can impact gene expression in a reversible and heritable nature. Our lab is the first group to discover that the long noncoding RNA DLEU2 and its hosted microRNAs miR15a/16-1 are involved in metabolic disease and may regulate chromatin modifications.

14. GHRELIN SIGNALING IN MACROPHAGES AND INFLAMMATORY BOWEL DISEASE IN AGING

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ABSTRACT

Inflammaging is a chronic low-grade inflammation in aging, contributing to various age-associated diseases including inflammatory bowel disease (IBD). Gut hormone ghrelin is mainly produced by the gastrointestinal track, and it functions through its receptor Growth Hormone Secretagogue Receptor (GHSR). We hypothesize that GHSR in macrophages regulates gut

barrier and experimental colitis in aging. Experimental colitis was induced in young and aged myeloid- specific GHSR knockout mice. GHSR inhibition in myeloid cells attenuated susceptibility to colitis and aging leaky gut in aging. Our results suggest that ghrelin signaling plays an important role in intestinal inflammation and barrier function in aging.

15. RELEVANCE OF DIETARY INTAKE ON POLYCYSTIC OVARY SYNDROME (PCOS) RISK IN ADOLESCENTS

Joelle Sfeir1, April Bertomo[2], Gabriella Spinola[2], Seung-Yeon Shawn Ha[3], Heidi Vanden Brink[1]

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ABSTRACT

The relevance of nutrition during reproductive maturation is unknown, although is associated with pubertal timing and PCOS in adults. To address this, we conducted a cohort study to monitor changes in diet with reproductive maturation in 40 adolescents during the post-menarcheal years. Full analyses are underway, however preliminary results indicate that red meat intake within one year of menarche tends to predict menstrual irregularity ($p=0.05$) and hirsutism, a marker of hyperandrogenism in PCOS, is associated with lower legume ($p=0.02$), vegetable ($p=0.04$), and whole grain intake ($p=0.02$), independent of BMI. Dietary intake may contribute to the development of PCOS in adolescents.

TOXICOLOGY

Oral (3MT) Presentation Abstracts #16 - 18

16. BIS-INDOLE DERIVED NUCLEAR RECEPTOR 4A (NR4A) LIGANDS ENHANCE TEMOZOLOMIDE CYTOTOXICITY IN GLIOBLASTOMA CELLS

Evan Farkas[1*], Keshav Karki[1], Gargi Sivaram[2], Jaclyn Reyes[2], Caitrina Kearns[2], Robert Rostomily[3], Andrei Mikeev[3], and Stephen Safe[1]

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ABSTRACT

Glioblastoma (GBM) is the most common form of adult brain cancer and the standard of care temozolomide (TMZ) and radiation therapy are subject to drug resistance. In-vitro studies with TMZ show that growth inhibition of human and mouse GBM cell lines can be in the low mM range. The orphan nuclear receptor 4A (NR4A) is highly expressed in GBM patients and their tumor growth is inhibited by a series of bis-indole derived compounds (CDIMs) that bind both receptors and act as inverse agonists. These CDIMS, synergistically sensitize tumor cells to TMZ treatment by regulating TMZ-resistance genes/gene products.

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17. DETECTING DIET-RELATED CHANGES IN MOUSE TISSUE COMPOSITION WITH RAMAN SPECTROSCOPY

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ABSTRACT

The rapid rise of metabolic syndrome in the U.S., emphasizes the inadequacy of national dietary

guidelines and a need for new methods of studying diet. In this study, we employed Raman spectroscopy investigate diet-related changes in mouse tissue composition, hypothesizing that Raman could not only detect variations in response to dietary changes but also accurately predict the specific diet followed by each mouse over preceding months. Our study found that Raman could differentiate diets, detect nutritional deficiencies, and discern age-related changes in tissue composition. These findings lay the groundwork for future applications in metabolic studies and personalized health monitoring.

18. APPLICATION OF MOBILE MONITORING IN DIVERSE ENVIRONMENTS TO CHARACTERIZE HAZARDOUS VOLATILE ORGANIC COMPOUND MIXTURES

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ABSTRACT

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