

**LIFE SCIENCES
GRADUATE RECRUITMENT
SYMPOSIUM:
BLAST OFF YOUR SCIENTIFIC CAREER!**

FEBRUARY 4TH-6TH 2026

HOWDY!

WELCOME

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.

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.

CONTACT:



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SCHEDULE OF EVENTS

**2:00 -
4:30 PM**

Graduate Student Poster Presentations

4141 James A. Baker III Pavilion

**4:30 -
5:45 PM**

Graduate Student Oral Presentations

4141 James A. Baker III Pavilion

**6:00 -
6:45 PM**

Keynote Speech by Erin Winick Anthony

**Science Communicator,
Founder of STEAM Power Media**

4141 James A. Baker III Pavilion

**7:00 -
8:30 PM**

Banquet & Mingle

RSVP only

4141 James A. Baker III Pavilion

OUR TEAM



Ximena Paez

Chair of the Life Sciences Graduate
Recruitment Symposium
Associate Department Head for Administration,
Department of Nutrition



Tera McAdoo

Graduate Program Coordinator,
Department of Biochemistry & Biophysics



Serina DeSalvio

Assistant Chair of the Life Sciences Symposium
Program Coordinator, Department of Nutrition



Irving Valdez

Program Coordinator, Office of Graduate
Studies, College of Medicine



Ruben Valdez

Program Coordinator, Office of Graduate
Studies, College of Medicine



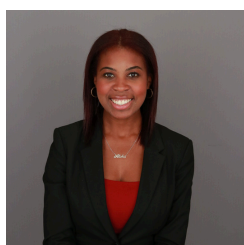
Isabel Caballero

Program Manager, Interdisciplinary
Graduate Program in Genetics & Genomics



Raquel Granados Aguilar

Program Coordinator & Graduate
Academic Advisor, Interdisciplinary
Faculty of Toxicology Graduate Program



Amanda Joseph

Program Coordinator, Interdisciplinary
Graduate Program in Genetics & Genomics



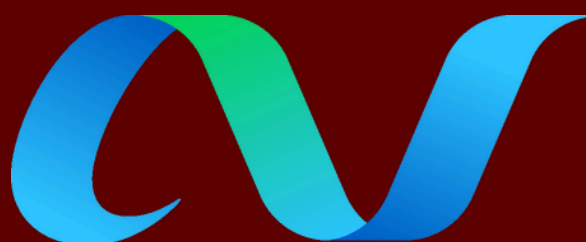
Sylvia Bernal Jones

Graduate Program Coordinator, Texas
A&M Institute for Neuroscience

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OUR BIGGEST SUPPORTER

Dr. Fuhui Tong

**Associate Provost and Dean
of the Graduate and
Professional School**



Dr. Fuhui Tong is the Associate Provost and Dean of the Graduate and Professional School. Her responsibilities include providing strategic, scholarly and financial leadership of the school, bolstering the university's identity as a research institution in a manner analogous to our peers, and supporting the enhancement of all graduate and professional programs to build and sustain excellence. Dr. Tong joined Texas A&M in 2007 and is a Professor in the Department of Educational Psychology.

Before serving as Interim Associate Provost and Dean from 2022-24, Dr. Tong served as Department Head and Doug Palmer Endowed Chair of Educational Psychology. Working collaboratively with her team, Dr. Tong has led the department to elevated academic excellence and recruited talented faculty and staff, along with \$22 million in new funding to support research and graduate education. During her tenure, graduate applications and enrollment of underrepresented minority graduate/professional students saw considerable gains. Dr. Tong has also co-directed an \$80 million system-approved research center where a diverse group of graduate students acquired research and publication experiences. Students advised by Dr. Tong have become local and global leaders in PK-16 settings.

KEYNOTE SPEAKER



**Erin
Winick
Anthony**

**Science Communicator,
Founder of STEAM Power Media**

Erin Winick Anthony is a maker, writer, science fashionista, and science communicator. She is the founder of science communication company STEAM Power Media which works to share the creativity in science and engineering, and help others tell their science stories. Her clients include Astrolab, Hearst Media Production Company, Zero G, Space Center Houston, the International Ocean Discovery Program, and more. Erin has a B.S. in mechanical engineering and more than 10 years experience working as a STEM communicator. Erin previously worked as a science communicator for the International Space Station, serving as a storyteller for the science conducted aboard the orbiting laboratory, and a reporter for MIT Technology Review. Erin is also a competitive pinball player and ranked in the top women pinball players in the world. Erin spends her weekends hiking, writing, 3D printing, playing with her cats, listening to Broadway cast albums, and scrolling through TikTok.

ORAL PRESENTERS

Nutrition

Angelica Michelle Burgman

Title: The impact of hyperpalatable ultra-processed foods on caloric intake, weight, and related cardiometabolic risk factors: a systematic review of randomized trials

Victoria Martinez

Title: The impact of phthalate mixtures in diabetes via epigenetic pathways

Honghui “Amber” Chang

Title: Glycemic Variability in Adolescents with Polycystic Ovary Syndrome (PCOS)

Toxicology

Kaylyn Dinh

Title: Forever chemicals and infant health: biomonitoring in breast milk and development of clay-based strategies for mitigation

Aidan P. Holman

Title: DyeSPY-LINK: A Novel Framework for Comparing Dyed Hair Samples in Forensic Casework

Wai Ning Tiffany Tsui

Title: Ligands For Orphan Nuclear Receptors NR4A1 and NR4A2 as Non-Hormonal Therapeutic Targets in Endometriosis

Neuroscience

Jordan Cook

Title: Antiphasic Circadian Rhythms in Dopamine Release Tune Reward Signaling

Jessica A. Bryan

Title: Targeting noradrenergic signaling to mitigate bone loss after spinal cord injury

Erika Marks

Title: Cognitive Impairment Resulting From Spinal Cord Injury: Causes and Treatments

ORAL PRESENTERS

Genetics & Genomics

Anestacia S. Robinson

Title: Circadian dynamics of the tumor microenvironment in triple negative breast cancer

Charles Mitchell

Title: A new approach for parkinson's treatment: neuroprotective nanomaterials alleviate amyloid burden, reduce cellular stress, and extend lifespan

Tyler Chan

Title: Genomic modification of the stable fly for sterile insect technique

Biochemistry & Biophysics

Kelly Risch

Title: Characterizing the role of conformational entropy in integral membrane protein folding and function

YuChen Yang

Title: Understanding how protein aggregate structure impacts disaggregation by molecular chaperones

Kiryl Zhaliaska

Title: Dag lactones as selective chemical probes of the $\text{pkc}\delta$ c1b domain

Medical Sciences

Michael Woolley

Title: Repurpose an FDA-approved antibody using DARPIn-scaffolded Bridge Protein

Tamara Natour

Title: Norepinephrine is a Novel Regulator of T helper 17 Polarization

Samantha Beevers

Title: Therapeutic strategy to target metastasis and plasminogen activation-mediated tumor microenvironment remodeling in cholangiocarcinoma

*Here at A&M, the science
competes at the highest level.*

- Dr. David Threadgill

University Distinguished Professor, Head of the Department of Nutrition, Texas A&M University



Poster Numbers

Poster Number	Name	Poster Title
1	Lauren Gladwell	Epigenetic Research in Obesity: From Bench Science to Bioinformatics
2	Emanuele Baldassarri	Genetic regulation of diet-induced thermogenesis in mice
3	Zahra Esmaeilinezhad	Changes in surrogate cardiometabolic risk factors in children and adults that may represent minimal important differences in target disease endpoints: scoping reviews and expert consensus
4	Chinanu Gubor	Nutrient analysis of dishes from independently owned Hispanic restaurants in east-central Texas: comparison with dietary benchmarks
5	Wen Jiang	Inhibition of HO1 by Small Molecules Improves Metabolic Disease
6	"DK" Diana Kolb	Menstrual & Reproductive Health and PCOS in U.S. Young Adult Females: A Descriptive Analysis from NAYAN
7	Hannah Lamar	Dietary fat and fiber intake, bile acid isoforms, and emergence of PCOS in post-menarcheal adolescents
8	Woody Liu	Myeloid GHSR reprograms cardiac macrophages to alter heart function in aging
9	Yuyang Lu	Macrophage Osr1 Controls the Fibroinflammatory Niche in Adipose Tissue
10	Deisy Ramos	Impacts of benzyl butyl phthalate (BBP) in adipose tissues via epigenetic pathways
11	Shan Xu	Tranquil: a bacterial therapeutic that attenuates autoimmune demyelination by reprogramming the neuroinflammatory microenvironment
12	Srijita Basak	Understanding the role of membrane lipids in PKC1 activation in <i>S. cerevisiae</i>
13	Abhishek Bastiray	Conformational switching of SARS-CoV-2 ORF9b enables hijacking of host Tom70

14	Alexandria Kemp	Advancing Fragment-Based Drug Discovery Using Reverse Micelle Nuclear Magnetic Resonance
15	Mason Kretiv	Orally Administered pDARPin for the Local Pan-Neutralization of TcdB Toxin in the Intestines
16	Evan Kurtz	Analysis of Small Signaling Peptides in Sorghum bicolor: Integrating Phylogeny and Gene Expression to Characterize Roles in Stem Development
17	Brianna Martin	GTP hydrolysis triggers membrane remodeling by AMPH-1
18	Noah Sherer	Molecular mechanism by which SARS-CoV-2 Orf9b suppresses the Tom70-Hsp90 interaction to evade innate immunity
19	Jadon Sitton	Fatty acids alter the toxicity of islet amyloid polypeptide aggregates in a length and saturation dependent manner
20	Dylan Suriadinata	Bridging protein TTR-53 mediates lipid signaling for cell corpse clearance
21	YuChen Yang	Understanding how protein aggregate structure impacts disaggregation by molecular chaperones
22	Bo Zhou	Resolving the 22q11.2 deletion using CTLR-seq reveals chromosomal rearrangement mechanisms and individual variance in breakpoints
23	Zara Akbari	Differential Effects of Intravenous and Neuron-Specific miR-20a-3p Delivery on White Matter Integrity and Cognitive Recovery After Stroke in Middle-Aged Female Rats
24	Roshni Babu	Neural stem cell-derived extracellular vesicle therapy slows down tau accumulation and associated neuropathology in ps19 mice
25	Kayli Colitts	Sex Differences in Spinal Cord Transcriptomics Following Noxious Stimulation and Lidocaine Administration in Contused Rats
26	Casey Delaney	Cerebello-basal ganglia networks and behavior across adulthood and aging
27	Yufei Huang	Early-Stage Corticostriatal Circuit Hyperactivity Impairs Cholinergic Function and Cognitive Flexibility in an Alzheimer's Model
28	Faith Lewis	Is cortical hyperexcitability after traumatic brain injury driven by cortical or thalamic excitation?

29	Jordan Mar	Assessing time-of-day-dependent learning and memory in mice using Novel Object Recognition test
30	Lizzy Olsen	Structural Neuroplasticity of Motor Circuits Following Loss of Motor Neuron Subsets
31	Alexis McAlister	MIF/CD74 Inhibition Reveals Sex-Specific Immune Responses in Mice After Traumatic Brain Injury
32	Nora Glenae	Acute igf-1 treatment improves locomotor recovery but not affect in spinally injured male rats
33	Lauren Pitts	Vagal innervation paradoxically regulates psychological trauma-induced inflammation
34	Kofi Owusu-Ansah	Peptide lv deficiency exacerbates lipopolysaccharide-induced retinal inflammation in a sex-dependent manner
35	Savannah Ruffino	Traumatic brain injury impairs subpopulation-specific plasticity towards reduced LTP and excessive LTD within the barrel cortex
36	Samantha Sweck	Circadian regulation of LC-MPFC dynamics underlying spontaneous behavior
37	Kennedy Coleman	Implications of the structural conformation of BOSR in <i>Borrelia burgdorferi</i>
38	Charlotte Heide	Influencing Immune System Composition and Function with Diet
39	Duminduni Hewa Angappulige	Spatial immunoprofiling reveals NACC1-driven immunosuppressive tumor-infiltrating lymphocytes in triple-negative breast cancer
40	Joseph Hoppe	Diabetic factor endothelin-1 accelerates human retinal microvascular endothelial cell invasion via ETB receptor/rho kinase signaling
41	Maralyce Martinez	Intestinal epithelial stem cell treatment following spinal cord injury to repair gut dysbiosis and neurological deficits
42	Mary Dickson-Amagada	SSRI-associated weight gain in adult women: a scoping review
43	Chelsea Page	Biomarkers to differentiate menopause transition stages: implications for clinical trajectories and treatment
44	Manshi Patel	Differences in colonic cellular composition and pathway enrichment after chronic spinal cord injury using single-cell sequencing

45	Kaylin Pickle	Post-stroke cognitive-affective impairment in middle-aged acyclic rats is attenuated by ovariectomy
46	Maddie Reeves	Linear Plasmid 36 and its role in Type I IFN upregulation in the mammalian host
47	Cristobal Rodriguez	A critical role for S100B secretion by substantia nigra pars compacta astrocytes in accelerating 6-hydroxydopamine induced Parkinsonism in mice
48	Brittany Shapiro	Borrelia burgdorferi BosR-mediated post-transcriptional regulation
49	Mariajose Tarot	Contribution of rickA to actin-tail formation in Rickettsia rickettsii
50	Hussain Alcassab	Vivo morpholino antisense inhibitor of dkk-1 in canine osteosarcoma reduces cell viability and dkk-1 levels
51	Emmarie Alexander	Characterizing genomic ancestry in a hybrid cat breed
52	Andres Barboza	Seqdef: an R package for phylogenetically weighted genomic prioritization across the tree of life
53	Isabella Childers	One of a kind: the unusual Y chromosome of the Hoffmann's two-toed sloth
54	Sydney Christensen	Neuronal histone modification: a dual model for arousal and sleep regulation
55	Andrew Eastland	Analysis of maize temporal trait stability by functional model fitting
56	Samantha Foster	Regulating epithelial-to-mesenchymal transition-driven breast cancer metastatic capacity with molybdenum disulfide (mos2) nanoparticles
57	Brighton Garrett	Dietary determinants of hepatic retinoid variation in genetically diverse mice: foundations for a predictive model
58	Luca Henze	Intraspecies competition between uropathogenic and commensal E. Coli
59	Pei-Jung Hsin	Unraveling the beauty and complexity of chicken feather patterns: integrative genome-wide mapping and single-cell transcriptomics
60	Harpreet Kaur	Sim2 regulation of mitochondrial dysfunction in down syndrome
61	Bella Lawlar	Identifying molecular differences between flat and polypoid adenomas in colorectal cancer

62	William Leach	Defining erbb-independent and dependent modulators of colorectal cancer through the use of mouse genetics
63	Mayra Mendoza	Towards complete and accurate animal reference genomes: mapping unassigned scaffolds to chromosomes by oligo-fish
64	Doris Migliaccio	Exploring the interplay between social environments, ethanol exposure, and sleep in <i>Drosophila melanogaster</i>
65	Trevor Millar	Synten-Aware Evaluation of CRISPR-Cas9 PAM Conservation Across Divergent <i>Drosophila melanogaster</i> Genomes
66	Veronika Mojik	The role of orcokinin in nutrient homeostasis in <i>Drosophila melanogaster</i>
67	Charles Mitchell	A new approach for Parkinson's treatment: neuroprotective nanomaterials alleviate amyloid burden, reduce cellular stress, and extend lifespan
68	Natalie Wideman	Chaos in the genome: an educational video game project focusing on student interest in science
69	Ibrahim Alshammari	Quantitative estimates of toxicodynamic variability for new approach methodologies-based systemic safety toolbox using a population-based human in vitro model
70	Nikki Barlow	Leveraging ion mobility spectrometry-mass spectrometry (IMS-MS) for rapid exposure assessment of complex chemical mixtures
71	Gustavo Elizondo	Functional dissection of extracellular vesicle- and soluble factor-mediated responses to the airway epithelial secretome: an air-liquid interface platform for inhalation toxicology
72	Hayley Jesse	Optimization of endothelial network formation and immune-endothelial signaling using commercial microphysiological vascular devices
73	Zachary Kobs	From source to body: disparities in arsenic exposure from Texas water systems to national biomonitoring
74	Madison McFarland	Expanding clay-based remediation strategies to reduce aflatoxin m1 contamination in milk
75	Hannah Roe	ECHA Writes Back: The Reasons for Rejection of Read-Across in Compliance Check Decisions by the European Chemicals Agency

76	Alexandra Svetlik	Extracellular vesicles Mediate Inflammatory Signaling in Arsenic-Exposed Bronchial Epithelial Cells
77	Devin Teri	Method development for exposure assessment using passive sampling devices and ion mobility spectrometry-mass spectrometry
78	Tzu-Hsin Yen	Targeted lipidomics as a high-throughput new approach method for mechanism-based studies of hepatotoxicity in vitro

**THE IMPACT OF HYPERPALATABLE ULTRA-PROCESSED FOODS ON
CALORIC INTAKE, WEIGHT, AND RELATED CARDIOMETABOLIC RISK
FACTORS: A SYSTEMATIC REVIEW OF RANDOMIZED TRIALS.**

Angelica M. Burgman^{1*}, Zahra Esmaeilinezhad¹, and Bradley C. Johnston²

¹ Department of Nutrition, Texas A&M University, College Station, TX, USA

² Department of Epidemiology and Biostatistics, Texas A&M University, College Station, Texas,
USA

ABSTRACT

Ultra-processed foods (UPFs) are increasingly consumed worldwide. Using established Cochrane and GRADE review methods, we will investigate the causal role of food processing as defined by NOVA through a systematic review and meta-analysis of randomized controlled trials assessing higher versus lower UPF consumption for seven days or longer in children and adults. Outcomes include changes in total daily energy intake, body weight, blood pressure, serum lipids, glucose metabolism and participant-reported outcomes. We will investigate whether hyperpalatable foods as defined by Fazzino et al., 2019, rather than the degree of processing alone, are an effect modifier for adverse health effects associated with UPFs.

THE IMPACT OF PHTHALATE MIXTURES IN DIABESITY VIA EPIGENETIC PATHWAYS

Victoria Martinez^{1,2}, Sunil Venkategowda¹, Nitya Shree¹, Mahua Choudhury¹

¹Department of Pharmaceutical Sciences, Irma Lerma Rangel College of Pharmacy,
Texas A&M Health Science Center, College Station, TX, USA

²Department of Nutrition, Texas A&M University, College Station, TX, USA

Phthalates are widespread environmental endocrine disruptors which can influence metabolic regulation. Six major phthalates, commonly detected in humans, are defined here as phthalate mixtures (PM). Computational analyses demonstrate that PM can affect epigenetic factors leading to “diabesity”. To explore this, we examined the effect of PM in the presence or absence of high-fat diet (HFD) in C57BL/6 male mice. PM exacerbated the HFD induced metabolic aberration (body weight, glucose tolerance, and insulin resistance) as early as week 3. In conclusion, these findings suggest that PM exposure contributes to metabolic dysfunction and may exacerbate diabesity-related outcomes.

GLYCEMIC VARIABILITY IN ADOLESCENTS WITH POLYCYSTIC OVARY SYNDROME (PCOS)

Honghui “Amber” Chang^{*}, Heidi Vanden Brink

Department of Nutrition, Texas A&M University, College Station, TX, US

ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder that develops during adolescence and increases the risk of Type 2 Diabetes (T2D). Effective dietary interventions to mitigate PCOS and progression to T2D in adolescents remain undefined, owing in part to challenges when implementing lifestyle change during this developmental stage and a poor understanding of how diet affects PCOS progression. We will pilot continuous glucose monitors (CGMs) to measure the degree of glycemic variability in adolescents with versus without PCOS and will identify macronutrient compositions via mixed-meal tests associated with a lower glycemic response. Feasibility of CGMs will also be assessed.

UNDERSTANDING THE ROLE OF MEMBRANE LIPIDS IN PKC1 ACTIVATION IN *S. CEREVISIAE*

Srijita Basak^{1*}, Vytas A. Bankaitis² and Tatyana I. Igumenova¹

¹ Biochemistry and Biophysics, Texas A&M University, College Station, TX, USA

² Molecular and Cellular Medicine, Texas A&M University, College Station, TX, USA

ABSTRACT

PKC is a serine/threonine kinase which phosphorylates its substrates using ATP and is conserved in eukaryotes. Dysregulation of PKCs in mammals lead to diseases including neurodegenerative diseases and cancer, making it a valuable target for therapeutics. Yeast PKC isozyme, PKC1 is a conserved model which can be used to understand the mechanism and membrane signaling of the kinase, contributing to PKC-mediated therapeutic development. The aim of the study is to identify/assess the membrane lipids involved in interaction of the conserved regulatory domains of PKC1 in yeast. Understanding the lipid-interactions will provide insight into activation mechanism of the kinase.

CONFORMATIONAL SWITCHING OF SARS-CoV-2 ORF9B ENABLES HIJACKING OF HOST TOM70

Abhishek Bastiray^{1*}, Noah Sherer¹, and Jae-Hyun Cho¹

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

ABSTRACT

SARS-CoV-2 remains a global health challenge; however, the precise mechanistic basis of how SARS-CoV-2 evades the host immune response remains elusive. Open reading frame 9b (ORF9b), an accessory protein of SARS-CoV-2, is a key antagonist of the host innate immune response. ORF9b binds to the translocase of mitochondrial outer membrane (Tom70), to attenuate host interferon response. Interestingly, ORF9b is a fold switching protein which adopts a β -sheet rich dimeric structure but transitions to α -helical conformation upon binding Tom70. Using bio-layer interferometry (BLI) and nuclear magnetic resonance (NMR) spectroscopy, we provide key insights into the immune evasion strategy employed by SARS-CoV-2.

ADVANCING FRAGMENT-BASED DRUG DISCOVERY USING REVERSE MICELLE NUCLEAR MAGNETIC RESONANCE

Alexandria M. Kemp^{*}, Kyle Mimun, and Riley Schatz, Anthony C. Bishop, Thomas D. Meek, A. Joshua Wand

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

ABSTRACT

FBDD is advantageous over HTS in drug design. Binding subsites overlooked in HTS can be exploited by smaller compounds and inhibitors can be made by their linkage. FBDD does have some disadvantages due to weak binding nature of these compounds. An RM NMR approach is a powerful detection method for fragments. The procedures developed utilize technologies such as liquid handling, mechanized sample handling, programmed data processing, and automated data analysis. ¹⁵N TROSYs were collected as evidence of successful RM formation with new surfactants of a truncated enzyme construct designed to overcome challenges in NMR due to large molecular weight.

ORALLY ADMINISTERED pDARPIs FOR THE LOCAL PAN-NEUTRALIZATION OF TcdB TOXIN IN THE INTESTINES

Mason Kretiv^{1*}, Yu Zeng², and Zhilei Chen^{1,2}

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

² Microbial Pathogenesis and Immunology Department, Texas A&M University Health Science Center, Bryan, Texas, USA

ABSTRACT

Clostridioides difficile infection (CDI) is a colonic pathogen responsible for the most cases of infectious diarrhea and a 20% recurrence rate. TcdB toxin, a 270kDa virulence factor of CDI, has become a popular drug target as neutralizing it attenuates recurrence. However, its localization in the colon and strain specific variations make it challenging for drug discovery. We performed affinity and stability maturation of a protease-stable designed ankyrin repeat protein (pDARPin) library via Click Display to pan for protease stability and pan-TcdB Binding, with successful enrichment observed. Next steps will involve screening for individual protease-stable DARPins that pan-neutralize TcdB toxin.

Analysis of Small Signaling Peptides in *Sorghum bicolor*: Integrating Phylogeny and Gene Expression to Characterize Roles in Stem Development

Evan Kurtz^{1*}, Brian McKinley¹, and John Mullet¹

¹Department of Biochemistry and Biophysics, Texas A&M University, College Station, Texas, 77843

ABSTRACT

Small signaling peptides (SSPs) are critical regulators of plant growth, development, and responses to biotic and abiotic stress, yet their role in the C4 grass *Sorghum bicolor* is largely uncharacterized. 219 sorghum genes encoding peptides were assigned to 19 gene families. Expression of the 219 SSP encoding genes in sorghum organs, during stem development, and in stem tissues and cell types revealed distinct spatial, temporal and developmental patterns of expression. The results provide a foundation of information for analysis of SSP functions in sorghum that can be integrated to modulate traits important for production of sorghum crops.

GTP hydrolysis triggers membrane remodeling by AMPH-1

Wei Gai, Yuhang Wang, Brianna Martin, Junjie Zhang, Chavela M. Carr, and Hays S. Rye*

Department of Biochemistry and Biophysics, Texas A&M University, Texas, USA

ABSTRACT

Membrane-enclosed transport carriers return biological molecules from the recycling endosome to the plasma membrane using a poorly understood mechanism. guanine nucleotide regulated manner. We propose a model linking GTP binding and hydrolysis to the membrane binding and tubulation. GTP binding stabilizes interactions between AMPH-1 and the membrane through amphipathic, N-terminal alpha helices. In the GDP-bound state, these helices are repositioned to interact with the N-terminal helices of other homodimers, to form an oligomeric AMPH-1 lattice that tubulates the membrane, in preparation for carrier formation by membrane fission.

MOLECULAR MECHANISM BY WHICH SARS-CoV-2 ORF9B SUPPRESSES THE TOM70-HSP90 INTERACTION TO EVADE INNATE IMMUNITY

Noah Sherer^{1*}, Abhishek Bastiray¹, Xiao-Ru Chen¹, Trivikram Molugu¹, Gaya P. Yadav¹, Tatyana I. Igumenova¹, Jae-Hyun Cho¹

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

ABSTRACT

Understanding how SARS-CoV-2 evades the innate immune system is crucial for preventing future outbreaks. An accessory protein, Orf9b (open reading frame 9b), is a crucial antagonist of the antiviral response. Orf9b binds to Tom70 (translocase of mitochondrial outer membrane 70), preventing its interaction with Hsp90 (heat shock protein 90); thus, inhibiting the immune response. However, the exact molecular mechanism remains unclear. To elucidate the mechanistic basis of this mechanism, we employ single particle cryo-EM (cryo-electron microscopy) and ITC (isothermal titration calorimetry). Our results provide mechanistic insight into how SARS-CoV-2 disrupts the host innate immune response through Tom70 antagonization.

Fatty acids alter the toxicity of islet amyloid polypeptide aggregates in a length and saturation dependent manner

Jadon Sitton^{1*}, Davis Pickett¹, Andrew Hung¹, Roa Elsaigh¹, and Dmitry Kurouski^{1,2}

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

² Department of Biomedical Engineering, Texas A&M University, College Station, TX, USA

ABSTRACT

The aggregation of islet amyloid polypeptide (IAPP), a 37-amino acid hormone, has detrimental implications in type 2 diabetes (T2D). T2D affects over 30 million Americans, and incidence of T2D has drastically risen in younger populations over the past decade. While lifestyle and nutrition are linked to this rise, the exact molecular origin remains unclear. In this study, we demonstrate that fatty acids present in food and food supplements accelerate IAPP aggregation and increase aggregate toxicity. In *C. elegans* overexpressing IAPP, high dietary fatty acid content significantly shortens lifespan, suggesting dietary fatty acids can accelerate T2D onset and progression.

BRIDGING PROTEIN TTR-53 MEDIATES LIPID SIGNALING FOR CELL CORPSE CLEARANCE

Dylan Suriadinata^{1*}, Chinmay Phadke¹, Riley Harrison², Julia Frondoni², Gabriela S. Paredes-Davalillo², Ann M. Wehman^{1,2}

¹ Department of Biochemistry and Biophysics, Texas A&M University, College Station, Texas 77843, USA

² Department of Biological Sciences, University of Denver, Denver, CO 80210, USA

ABSTRACT

Phagocytosis of dying cells is essential for homeostasis and is mediated by engulfment pathways recognizing lipids. Transthyretin-like TTR-52 recognizes phosphatidylserine (PS) for apoptotic cell clearance in *Caenorhabditis elegans* but is not required for all dying cells. We demonstrate that *ttr-53* promotes polar body internalization. Fluorescently-tagged TTR-53 localizes to dying polar bodies, but its localization does not depend on PS. Deletion of *ttr-53* and PS synthase enhances the delay in polar body internalization, suggesting cells use both PS-dependent and PS-independent pathways to carry out phagocytosis. The existence of multiple lipid signals to recognize dying cells emphasizes its importance for normal physiology.

**UNDERSTANDING HOW PROTEIN AGGREGATE STRUCTURE IMPACTS
DISAGGREGATION BY MOLECULAR CHAPERONES**

YuChen Yang*, Daniel Shoup, Rajan Thapa and Hays Rye

Department of Biochemistry and Biophysics, Texas A&M University,
College Station, TX

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.

RESOLVING THE 22Q11.2 DELETION USING CTRLR-SEQ REVEALS CHROMOSOMAL REARRANGEMENT MECHANISMS AND INDIVIDUAL VARIANCE IN BREAKPOINTS

Bo Zhou^{1*}

¹ Biochemistry and Biophysics, Texas A&M University, Texas, U.S

ABSTRACT

The 22q11.2 Deletion Syndrome (22q11DS) is the most common human microdeletion disorder. By resolving the exact sequence configurations in 22q11DS, this study solves a longstanding problem in human genetics. Offering insights into the high prevalence of 22q11DS in the population, this study shows that different 22q11DS patients have distinct deletion breakpoints and deletion-sequence configurations and that all patients analyzed in this study have a transposon sequence at the exact breakpoint location. CRISPR-targeted long-read sequencing (CTRLR-Seq), a universally applicable method for resolving large, challenging genomic regions and their associated rearrangements, is presented.

DIFFERENTIAL EFFECTS OF INTRAVENOUS AND NEURON-SPECIFIC miR-20a-3p DELIVERY ON WHITE MATTER INTEGRITY AND COGNITIVE RECOVERY AFTER STROKE IN MIDDLE-AGED FEMALE RATS

Zara Akbari^{1,2*} and Farida Sohrabji^{1,2}

¹ Texas A&M Institute for Neuroscience, Texas A&M University, College Station, TX, USA

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ABSTRACT

Our previous studies show that microRNA-20a-3p (miR-20a-3p) is a promising therapeutic candidate for ischemic stroke, but the precise mechanism and optimal delivery strategy remains unclear. Intravenous (IV) administration of miR-20a-3p in middle-aged female rats after stroke led to robust recovery, improving both acute sensorimotor outcomes and long-term cognition. This was accompanied by preservation of forebrain white matter tracts and reduced secondary neurodegeneration, as evidenced by callosal, internal capsule and anterior commissure volume and ventricular symmetry. Our previous studies also showed that IV mir20a-3p localized to neurons. In the present study, we investigated whether neuron-specific expression of miR-20a-3p would yield similar neuroprotective effects. Using a Tet-inducible AAV vector containing mir20a-3p or a scrambled oligo under the neuron-specific enolase (NSE) promoter, we delivered the construct stereotactically to the striatum. Five weeks later, stroke was induced via middle cerebral artery occlusion (MCAo) followed by ip administration of doxycycline (a Tet analog) to induce expression of the construct. Stroke impaired sensorimotor function in the acute phase, which was attenuated by neuron-specific mir20a-3p enrichment. In the chronic phase, stroke impaired spatial learning strategies as tested with Barnes maze, which was attenuated by neuronal miR20a-3p, but did not improve recall. Histopathology showed that stroke reduced the volume of the ischemic hemisphere and enlarged ventricular volume, which was not attenuated by neuronal mir20a-3p. Anterior commissure, corpus callosum and internal capsule volumes all showed a significant reduction in the left (ischemic) hemisphere of scrambled oligo treated stroke rats, while in the miR20a-3p enriched stroke group anterior commissure volume was preserved but not corpus callosum and internal capsule. These findings suggest that broader neuroprotection from IV delivery may result from miR-20a-3p engagement with multiple cell types, including astrocytes and microglia targets, not reached by neuron-restricted expression. Overall, our results suggest systemic drug delivery maximizes neuroprotection in stroke recovery. Future studies will focus on determining the cellular target(s) necessary for therapeutic efficacy of post-stroke interventions.

Supported by RFAG042189 to FS; AARF-21-849749 to DS.

NEURAL STEM CELL-DERIVED EXTRACELLULAR VESICLE THERAPY SLOWS DOWN TAU ACCUMULATION AND ASSOCIATED NEUROPATHOLOGY IN PS19 MICE

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ABSTRACT

Tauopathy, typified by increased tau hyperphosphorylation within neurons and neuroinflammation, leads to cognitive dysfunction. Since extracellular vesicles shed by human induced pluripotent stem cell-derived neural stem cells (hiPSC-NSC-EVs) carry diverse miRNAs/proteins that can inhibit tau phosphorylation and neuroinflammation, this study tested the effects of intranasal administration of hiPSC-NSC-EVs in four biweekly doses, commencing at 3 months of age, on cognitive function at 5-6 months of age in female PS19 mice. Compared with the vehicle-treated group, the hiPSC-NSC-EVs-treated group displayed improved cognitive function, reduced tau hyperphosphorylation and neuroinflammation, and enhanced hippocampal neurogenesis. Thus, hiPSC-NSC-EVs therapy has promise in treating tauopathies.

Funding: Supported by a grant from the National Institute on Aging (1RF1AG074256-01A1 to A.K.S.)

SEX DIFFERENCES IN SPINAL CORD TRANSCRIPTOMICS FOLLOWING NOXIOUS STIMULATION AND LIDOCAINE ADMINISTRATION IN CONTUSED RATS

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ABSTRACT

Previously we have demonstrated that noxious stimulation following spinal cord injury (SCI) promotes disruption of the blood spinal cord barrier, increases hemorrhage, and induces deficits in locomotor function in male rats. Baine et al. (2022) observed that estrous cycle stage at the time of injury may be implicated. To elucidate the sex differences in pain-induced changes, male and female animals were subjected to a T11 moderate contusion injury. One day later, animals received 6 minutes of noxious stimulation or restraint for an equivalent duration. Animal hindlimb locomotor function was assessed before and after stimulation. Three hours following stimulation, a 1 cm section of spinal cord centered around the lesion was collected, and samples were used for bulk RNA sequencing. Principal component analysis revealed two principal components accounting for 30.92% and 14.80% of the variance, primarily driven by sex and stimulation conditions, respectively. Key pathways differentially affected by sex and/or noxious stimulation include factors involved in complement and coagulation cascades, cytokines and chemokines involved in immune response, and pathways associated with central sensitization. Another experiment with a similar design was conducted where animals received an epidural infusion of lidocaine or its vehicle immediately prior to nociceptive stimulation. This approach allows us to parse out the transcriptional changes resulting from nociceptive stimulation alone versus those associated with the pain-induced increases in hemorrhage. This large data approach provides novel insight into the mechanisms behind noxious stimulation-induced disruption of the BSCB as well as sex differences that modulate these effects.

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CEREBELLO-BASAL GANGLIA NETWORKS AND BEHAVIOR ACROSS ADULTHOOD AND AGING

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ABSTRACT

The cerebello-basal ganglia (CB-BG) circuit is key to understanding brain aging as it has been implicated in modulating cognitive, motor, and reward-related functions. We reviewed baseline CB-BG resting state networks associated with cognitive and motor circuits using an ROI-ROI approach. 138 participants between the ages of 35- 86 completed motor and cognitive batteries then underwent resting state fMRI. We have shown that this network contains motor and cognitive subcircuits and has altered connectivity associated with age, sex, progesterone, and a Stroop task. This investigation of CB-BG networks represents an essential step in increasing knowledge of age-related cognitive and motor declines.

EARLY-STAGE CORTICOSTRIATAL CIRCUIT HYPERACTIVITY IMPAIRS CHOLINERGIC FUNCTION AND COGNITIVE FLEXIBILITY IN AN ALZHEIMER'S MODEL

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ABSTRACT

Cognitive flexibility deficits arise early in Alzheimer's disease (AD), yet the circuit mechanisms remain unclear. We show that 5xFAD mice exhibit early impairments in instrumental reversal learning. Electrophysiology revealed abnormalities in mPFC neurons, the mPFC-to-dMSN pathway, and both dMSNs and cholinergic interneurons in the dorsomedial striatum. Sustained chemogenetic inhibition of the mPFC-to-DMS circuit reduced cortical A β accumulation, normalized glutamatergic transmission in the mPFC and DMS, restored striatal acetylcholine levels, and rescued reversal learning deficits. These findings identify a hyperactive mPFC-to-DMS circuit driving early cognitive inflexibility. Targeting this circuit may offer a therapeutic strategy to preserve cognitive function in early AD.

IS CORTICAL HYPEREXCITABILITY AFTER TRAUMATIC BRAIN INJURY DRIVEN BY CORTICAL OR THALAMIC EXCITATION?

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ABSTRACT

Following traumatic brain injury (TBI), sensory hypersensitivity is common in patients, and animal models recapitulate this phenotype. However, the underlying mechanisms remain largely unknown. To address this gap, we assessed synaptic and network activity in the somatosensory cortex one-week following TBI in mice using two-photon calcium imaging combined with electrophysiological recordings. We found an increased number of excitatory neurons recruited following sensory stimulations after TBI, demonstrating amplified cortical excitability at the network level. This increased responsiveness was associated with increases in both synaptic inhibition and neuronal intrinsic excitability – together potentially driving post-TBI hyperexcitability in the sensory cortex.

ASSESSING TIME-OF-DAY-DEPENDENT LEARNING AND MEMORY IN MICE USING NOVEL OBJECT RECOGNITION TEST

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ABSTRACT

Rodent behavioral assays are usually scored manually, which is slow and variable. Using machine learning tools (DeepLabCut for pose estimation and BehaviorDEPOT for behavior classification), we built a semi-automated pipeline to score the Novel Object Recognition task. After tuning parameters, automated exploration scoring closely matched manual scoring. Applying the pipeline to mice tested during either the dark or light phase showed that mice preferred the novel object in both conditions, and their locomotion and exploration were stable across the 10-minute test. Overall, the study validates automated NOR scoring and suggests that object preference and exploratory behavior are consistent across circadian timepoints.

STRUCTURAL NEUROPLASTICITY OF MOTOR CIRCUITS FOLLOWING LOSS OF MOTOR NEURON SUBSETS

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ABSTRACT

When neural circuits are damaged, the remaining neurons can exhibit structural neuroplasticity to form new synapses and restore function. Using *Drosophila melanogaster* larvae, we investigated the structural remodeling capacity of surviving motor neurons (MNs) with distinct intrinsic properties following perturbation of neighboring MNs. Complete denervation of muscle subsets induces extensive axonal sprouting in surviving tonic firing MNs, restoring muscle contraction and larval locomotion. In contrast, phasic firing MNs show minimal axonal remodeling, demonstrating that tonic MNs have a greater intrinsic capacity for structural neuroplasticity. Unlike physical MN loss, silencing did not trigger axonal sprouting, indicating that full muscle denervation is a prerequisite for compensatory axonal remodeling.

MIF/CD74 INHIBITION REVEALS SEX-SPECIFIC IMMUNE RESPONSES IN MICE AFTER TRAUMATIC BRAIN INJURY

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ABSTRACT

Traumatic brain injury (TBI) triggers acute inflammation, mediated by cytokines like macrophage migration inhibitory factor (MIF) and its receptor CD74. While MIF/CD74 signaling in astrocytes has been described, its role in peripheral and central immune responses remains unclear. We investigated the effects of antagonizing MIF/CD74 signaling on the immune response to TBI in male and female WT mice. Brains were analyzed by flow cytometry one-day post-injury. Male TBI mice showed significantly increased peripheral immune cell infiltration in the injured hemisphere compared to sham. Ongoing analyses suggest differential T and B cell involvement. These findings indicate sex-specific neuroimmune responses to TBI.

ACUTE IGF-1 TREATMENT IMPROVES LOCOMOTOR RECOVERY BUT NOT AFFECT IN SPINALLY INJURED MALE RATS

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ABSTRACT

Approximately one-third of people with spinal cord injury (SCI) report long-term depression. Blood-gut-barrier (BGB) dysfunction may contribute to this development. Male Sprague Dawley rats served as sham or SCI subjects (T12) or naive controls, treated with insulin-like growth factor 1 (IGF-1) or vehicle. Consistent with prior work, one-third of SCI rats displayed depression-like behavior, despite significant improvement of locomotor recovery and weight gain. IGF-1 preserved ileal goblet cells at 3 days, coinciding with increased serum markers of BGB breakdown. Long-term protection of the BGB may be necessary for reducing depression, but acute protection appears to improve locomotor recovery after SCI.

Funding: Woodnext Foundation

VAGAL INNERVATION PARADOXICALLY REGULATES PSYCHOLOGICAL TRAUMA-INDUCED INFLAMMATION

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ABSTRACT

Psychological trauma survivors are more likely to have debilitating inflammation, and our work suggests dysregulation of the autonomic nervous system may underlie this phenomenon. By ablating the autonomic splenic nerve, psychological trauma-induced inflammation (PTII) was significantly attenuated. However, the central neural pathways regulating PTII remain unknown. Given the anti-inflammatory role of the vagus nerve, we hypothesized that vagotomy would increase PTII. Surprisingly, PTII decreased in vagotomized animals, yet behavior was not impacted. Our data have elucidated a novel role for the vagus nerve in mediating PTII, which suggests it may be a site for therapeutic intervention for psychological trauma survivors.

PEPTIDE LV DEFICIENCY EXACERBATES LIPOPOLYSACCHARIDE-INDUCED RETINAL INFLAMMATION IN A SEX-DEPENDENT MANNER

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ABSTRACT

Peptide Lv (PLv) is an endogenous secretory peptide essential for retinal homeostasis. Its deletion causes early neural retinal thinning, decreased retinal light responses, and alters retinal vascular density. Although PLv plays an anti-inflammatory role in macrophages, its role in retinal inflammation remains unknown. We examined the inflammatory activity of PLv in LPS-stimulated cultured retinal cells and PLv-*null* mice, respectively. Using western blotting, PLv notably decreased NF- κ B P65 activation *in vitro*. Systemic LPS exposure causes sustained weight loss, deficits light responses and retinal edema in PLv^{-/-} males, indicating that PLv might protect against inflammation in a sex-specific manner.

TRAUMATIC BRAIN INJURY IMPAIRS SUBPOPULATION-SPECIFIC PLASTICITY TOWARDS REDUCED LTP AND EXCESSIVE LTD WITHIN THE BARREL CORTEX

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ABSTRACT

Learning and memory deficits are a major and persistent consequence of traumatic brain injury (TBI), yet how TBI alters synaptic and circuit plasticity in the intact, awake brain remains poorly understood. Using two-photon calcium imaging in barrel cortex of awake adult female mice, we demonstrate that TBI does not uniformly abolish plasticity but instead shifts its balance: synapses that would normally potentiate show attenuated strengthening, while highly active neurons are biased toward excessive depression. This subpopulation-specific reweighting of plasticity may underlie a circuit-level mechanism for impaired sensory processing and learning after TBI.

CIRCADIAN REGULATION OF LC-MPFC DYNAMICS UNDERLYING SPONTANEOUS BEHAVIOR

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ABSTRACT

Circadian rhythms in the brain arise from molecular clocks that regulate physiology and behavior across the 24-hour cycle, yet their role in neural circuits supporting complex spontaneous behavior is poorly understood. The locus coeruleus (LC) and medial prefrontal cortex (mPFC) form a bidirectional circuit critical for executive control, but whether this network is circadian regulated is unknown. Using long-term fiber photometry, we recorded calcium dynamics from LC noradrenergic terminals in the mPFC across multiple days in freely behaving mice. We found robust ~24-hour rhythms in LC-mPFC activity that persisted in constant darkness. Additionally, we disrupted *Cry1/2* in LC neurons to assess alterations in homecage behavioral organization.

**IMPLICATIONS OF THE STRUCTURAL CONFORMATION OF BOSR IN
BORRELIA BURGENDORFERI**

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ABSTRACT

Borrelia burgdorferi undergoes dynamic regulation to adapt to the mammalian host, partially reliant on the borrelial oxidative stress regulator (BosR). This unique metalloregulatory protein alters the *B. burgdorferi* transcriptional response through not fully understood mechanisms. This study investigates conformational structure of the protein in differing environments, in potentially distinct oligomeric forms, allows for BosR to differentially regulate gene expression. Mutated BosR strains have been generated to investigate the role of the protein structure status in global gene regulation and the pathogenic lifecycle. Our data suggests a novel regulatory mechanism used by BosR relative to other bacterial metalloregulatory proteins.

INFLUENCING IMMUNE SYSTEM COMPOSITION AND FUNCTION WITH DIET

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ABSTRACT

Genetics and lifestyle, including diet, can influence an individual's health and affect infection susceptibility. The genetically diverse Collaborative Cross mouse model previously enabled us to identify genetic loci correlated with *Salmonella enterica* Serotype Typhimurium infection outcomes. We aimed to incorporate common human diets in our diverse mouse model to modulate immune parameters and function. Genetically distinct mouse cohorts were adapted to a specialized diet for 4 weeks. Blood, spleens, and femurs were taken for analysis and generation of bone marrow-derived macrophages. It was determined that peripheral immune profiles and macrophage bacterial clearance were influenced by diet in a genetics-dependent manner.

SPATIAL IMMUNOPROFILING REVEALS NACC1-DRIVEN IMMUNOSUPPRESSIVE TUMOR-INFILTRATING LYMPHOCYTES IN TRIPLE-NEGATIVE BREAST CANCER

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ABSTRACT

Triple-negative breast cancer (TNBC) is characterized by a highly immunogenic yet functionally suppressed tumor microenvironment (TME), marked by heterogeneous populations of tumor-infiltrating lymphocytes (TILs). Nucleus accumbens-associated protein 1 (NACC1) is a transcriptional regulator with emerging roles in cancer progression and immune modulation; however, its contribution to immune regulation in TNBC remains poorly defined. In this study, we employed CODEX spatial immune profiling to analyze TNBC patient tissues. Spatial analysis revealed increased infiltration of CD8+ and CD4+ T cells within TNBC tumors, accompanied by regulatory T cell-mediated immunosuppression. Together, tumor-intrinsic NACC1 maintains immunosuppression despite robust immune infiltration in TNBC.

Key words: NACC1, TNBC, TME, CODEX

DIABETIC FACTOR ENDOTHELIN-1 ACCELERATES HUMAN RETINAL MICROVASCULAR ENDOTHELIAL CELL INVASION VIA ET_B RECEPTOR/RHO KINASE SIGNALING

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ABSTRACT

Endothelin-1 (ET-1) is significantly elevated in diabetic patients and animal models of diabetes, however its impact on pathological angiogenesis in proliferative diabetic retinopathy remains unclear. We used an established three-dimensional model of sprouting angiogenesis to investigate whether ET-1 promotes human retinal microvascular endothelial cell (HRMVEC) invasion and to elucidate the underlying mechanism. Using pharmacological inhibitors and RNA interference, we show that ET-1 activates the ET_B receptor and Rho kinase signaling to enhance overall HRMVEC invasion but does not impact the density of invading cells. The goal of these experiments is to understand how ET-1 contributes to pathological retinal angiogenesis.

INTESTINAL EPITHELIAL STEM CELL TREATMENT FOLLOWING SPINAL CORD INJURY TO REPAIR GUT DYSBIOSIS AND NEUROLOGICAL DEFICITS

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Abstract

Over 15 million people worldwide are affected by spinal cord injury (SCI). Following injury, SCI patients are at higher risk for gut dysbiosis, depression, and cognitive decline. Prior work has shown that after SCI the gut epithelial barrier is compromised. We hypothesize that the SCI-induced gut dysbiosis allows for translocation of inflammatory factors to the brain, to induce depression and cognitive dysfunction. This study uses intestinal epithelial stem cells as a potential treatment to restore the gut epithelium and prevent neurological changes following injury.

SSRI-ASSOCIATED WEIGHT GAIN IN ADULT WOMEN: A SCOPING REVIEW

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ABSTRACT

Although Selective Serotonin Reuptake Inhibitors (SSRIs) are first-line treatments for depression, weight gain remains a significant concern affecting adherence and cardiometabolic health. Evidence regarding female-specific weight trajectories, particularly concerning the menopause transition, is fragmented. This systematic scoping review aims to map SSRI-associated weight changes in adult women. Following PRISMA-ScR guidelines, we will search academic databases (2010–2024) to synthesize data on weight, BMI, and metabolic parameters across individual SSRIs. By analyzing hormonal influences and research gaps, findings will clarify the interplay between SSRIs and menopause, ultimately supporting informed shared decision-making in primary care.

BIOMARKERS TO DIFFERENTIATE MENOPAUSE TRANSITION STAGES: IMPLICATIONS FOR CLINICAL TRAJECTORIES AND TREATMENT

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ABSTRACT

Accurate differentiation of menopausal stages is critical, as hormonal transitions drive metabolic restructuring and the development of central adiposity. Studies show GLP-1 agonists show superior weight reduction in women, but they also disproportionately trigger higher rates of adverse drug reactions. Evidence suggests estrogen and GLP-1 share signaling pathways. This scoping review synthesizes hormonal, genetic, and metabolomic biomarkers to provide a diagnostic framework for menopause staging. Identifying these biomarkers is essential to unraveling how biological variability influences GLP-1 agonist outcomes, ultimately enabling safer, personalized therapeutic strategies during the vulnerable menopausal transition.

**DIFFERENCES IN COLONIC CELLULAR COMPOSITION AND PATHWAY
ENRICHMENT AFTER CHRONIC SPINAL CORD INJURY USING SINGLE-CELL
SEQUENCING**

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ABSTRACT

Spinal cord injury (SCI) results in an immediate primary injury as well as a host of secondary effects, such as chronic pain, cognitive impairment, and gut dysregulation. Gut dysregulation includes persistent changes in motility, barrier integrity, and chronic inflammation, but the regional cellular changes along the colon that underlie these effects are not well understood. In this study, proximal and distal colonic tissues 12 weeks after SCI are analyzed using single-nucleus RNA sequencing to characterize regional differences in cellular composition and pathway enrichment that contribute to these secondary gastrointestinal effects.

**POST-STROKE COGNITIVE-AFFECTIVE IMPAIRMENT IN
MIDDLE-AGED ACYCLIC RATS IS ATTENUATED BY
OVARIECTOMY**

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ABSTRACT

After menopause, women have higher stroke incidence and severity. In animal models, menopause is modeled by ovariectomy (OVX) of young rats or using reproductively senescent (RS; ≥ 10 mos) rats, both of which impair acute stroke outcomes. However, our recent studies show that OVX in RS rats, paradoxically, improves acute stroke outcomes and these persist in the chronic phase such as better hippocampal learning and social motivation. Furthermore, OVX in RS females increased levels of the neuroprotectant IGF-1 and suggests a mechanism by which removal of the aging ovary may improve stroke recovery.

LINEAR PLASMID 36 AND ITS ROLE IN TYPE I IFN UPREGULATION IN THE MAMMALIAN HOST

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ABSTRACT

Borrelia burgdorferi, the causative agent of Lyme disease, engages a wide variety of immune signaling pathways, some of which elicit type I interferon signaling in the mammalian host. This upregulation of type I IFN is associated with increased inflammation and severity of arthritis. There are very few borrelia genes which have been linked to increased inflammation. The severity of the disease is linked to strains which are classified by rRNA intergenic spacer types 1-3, with RST1 being the more inflammatory strain. Previous research has shown that the most variable region between these strains is in linear plasmid 36, specifically *bbk35-50*. Given this, we have made mutants in this region based on directionality to investigate their potential role in upregulation of type I interferon response in the mammalian host. Co-culture with mouse immortalized bone-marrow derived macrophages (iBMDM) and primary mouse embryonic fibroblast (MEF) have shown no difference in mutants when co-cultured with iBMDM but have shown significant differences when co-cultured with MEFs. These MEFs co-cultures highlighted the decreased type I IFN response with mutants' strains that are lacking all or part of the *bbk35-50*. Together, this data suggests that a subset of genes in this region is contributing to increased type I IFN response.

A CRITICAL ROLE FOR S100B SECRETION BY SUBSTANTIA NIGRA PARS COMPACTA ASTROCYTES IN ACCELERATING 6-HYDROXYDOPAMINE INDUCED PARKINSONISM IN MICE

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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-secreted 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.

Borrelia burgdorferi BosR-mediated post-transcriptional regulation

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ABSTRACT

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, including BasA, recognize specific transcripts and provide an additional layer of post-transcriptional regulation needed for host adaptation. Characterization of BosR::BasA::mRNA interactions will provide important insight into adaptive regulation in *B. burgdorferi*.

CONTRIBUTION OF RICKA TO ACTIN-TAIL FORMATION IN RICKETTSIA RICKETTSII

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ABSTRACT

Rocky Mountain spotted fever, caused by the obligate intracellular bacterium *Rickettsia rickettsii*, is the deadliest tick-borne disease in the US with mortality rates for untreated illness historically exceeding 20%. Spotted fever group *Rickettsia* coopt host-cell actin to achieve two forms of actin-based motility, early and late. Here we use a *rickA* transposon mutant and a polyclonal antibody against RickA to confirm that RickA contributes to early tail formation in *R. rickettsii*. However in contrast to data from *Rickettsia parkeri*, early and late tails are separate phenomena and early tail formation does not significantly contribute to virulence in a fever model.

VIVO MORPHOLINO ANTISENSE INHIBITOR OF Dkk-1 IN CANINE OSTEOSARCOMA REDUCES CELL VIABILITY AND Dkk-1 LEVELS

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ABSTRACT

High levels of Dkk-1 in human and canine osteosarcoma (cOS) is linked to more osteolytic lesions and higher chemotherapeutic resistance. We used 3 cOS cell lines, TOB, TOL, and TOT to determine Dkk-1 mRNA expression levels and response Dkk-1 exon 2 targeting vivo morpholino (C-DkkMo). TOL and TOT had the highest relative Dkk-1 expression levels and growth rates, which were focused on. Exposure to 2.5 μ M of C-DkkMo significantly reduced cell viability in both TOL and TOT, as well as reduced Dkk1 mRNA expression precipitously. Exposure to standard of care 65 μ M cisplatin demonstrated similar cell viability reduction as C-DkkMo exposure.

CHARACTERIZING GENOMIC ANCESTRY IN A HYBRID CAT BREED

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ABSTRACT

The Savannah is a hybrid cat breed created by crossing African servals with domestic cats. While the parental species are genetically distinct, Savannahs generally remain healthy, except that F1–F4 males are sterile. Male sterility appears linked to the amount of serval ancestry, suggesting genomic incompatibilities between servals and domestic cats. Using whole-genome sequences from 127 Savannahs (F1–F7), we identify candidate genomic regions linked to sterility and examine their variation with regards to parental species ancestry.

SEQDEF: AN R PACKAGE FOR PHYLOGENETICALLY WEIGHTED GENOMIC PRIORITIZATION ACROSS THE TREE OF LIFE

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ABSTRACT

As biodiversity genomics projects scale up, objective criteria are needed to maximize phylogenetic coverage while minimizing redundancy. We introduce SeqDef, a phylogenetically aware statistic and companion R implementation designed to quantify the marginal genomic value of taxa by weighting the availability of sequence resources in related species. We provide a mathematical justification, interfacing with community tools (ape, ggplot), and demonstrate SeqDef in a case study, combining IUCN Red List categories and NCBI assembly presence/absence to derive a conservation-aware sequencing priority ranking. Applied to this dataset, SeqDef identifies the Dwarf Gulper Shark (*Centrophorus atromarginatus*) as a top priority under our weighting scheme. SeqDef is modular and intended to be combined with other decision variables (cost, feasibility, trait coverage) to support strategic planning for biodiversity genomics programs such as the Earth BioGenome Project.

ONE OF A KIND: THE UNUSUAL Y CHROMOSOME OF THE HOFFMANN'S TWO-TOED SLOTH

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ABSTRACT

Placental mammal sex chromosomes are typically cited as an example of an evolutionarily stable system. One unusual sex chromosome system has been documented in Hoffmann's two-toed sloth (*Choloepus hoffmanni*). For instance, males have been found with Y/autosome translocations of various sizes. To conduct a thorough study of the genomic sequence and structure of the sloth's sex chromosomes, we sequenced and generated a haplotype-phased genome assembly of a male Hoffmann's two-toed sloth. This male possesses an unusual X_1X_2Y system in which the Y represents a Y+X+autosome fusion. This system provides a unique insight into the functional evolution of mammalian sex chromosomes.

NEURONAL HISTONE MODIFICATION: A DUAL MODEL FOR AROUSAL AND SLEEP REGULATION

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ABSTRACT

Characterizing arousal and sleep as distinct processes is essential to understand the mechanisms underlying sleep and circadian rhythms. To study this, we investigate neuronal post-translation modifications in *Drosophila*, focusing on the novel histone monoaminylation group of PTMs. We identified daily rhythmic patterns of monoaminylations and found that disrupting individual monoaminylations produced distinct effects on sleep and arousal behaviors. Based on these findings, we hypothesize that specific monoaminylations contribute to maintaining circadian rhythms by balancing arousal and sleep signals. Further investigation into the dynamics of these modifications and their impact on oscillatory gene expression will be key to uncovering sleep-arousal regulation.

ANALYSIS OF MAIZE TEMPORAL TRAIT STABILITY BY FUNCTIONAL MODEL FITTING

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ABSTRACT

Large crop datasets have allowed for the prediction of trait values and for the stability of traits across environments, but only for end-of-season traits, also known as terminal traits. This is because terminal traits are single values. Temporal traits, which vary continuously throughout the season, are harder to summarize, and thus harder to predict. This study uses a maize breeding dataset to develop a method that could estimate temporal trait values with similar predictive power to existing methods for terminal traits.

REGULATING EPITHELIAL-TO-MESENCHYMAL TRANSITION-DRIVEN BREAST CANCER METASTATIC CAPACITY WITH MOLYBDENUM DISULFIDE (MO₂) NANOPARTICLES

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ABSTRACT

Epithelial-to-mesenchymal transition (EMT) enables cancer cell migration, invasion, and metastasis. While EMT has physiological roles, its activation in tumors promotes metastatic progression, making it a therapeutic target. This study investigates molybdenum disulfide (MoS₂) nanoparticles as a drug-free EMT inhibitor in breast cancer models. MoS₂ treatment reduced cell migration, focal adhesion proteins, TGFβ signaling, EMT marker expression, mammosphere formation, and clonogenicity in vitro. In vivo, MoS₂ injection decreased primary tumor growth and lung metastases. These findings demonstrate that MoS₂ nanoparticles inhibit EMT and metastatic potential, highlighting their promise as a non-toxic anti-metastatic therapy.

DIETARY DETERMINANTS OF HEPATIC RETINOID VARIATION IN GENETICALLY DIVERSE MICE: FOUNDATIONS FOR A PREDICTIVE MODEL

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ABSTRACT

Malnutrition affects many Americans, risking adverse outcomes from nutrients such as vitamin A (vitA), including deficiency or excess. Liver biopsies, the most accurate method for measuring vitA, are invasive. We aim to develop a machine-learning model using simplified diversity outbred (SDO) mice to predict hepatic retinoid levels noninvasively from serum metabolomics, reflecting genetic diversity. Initial findings show significant variation in retinoid levels and diet responses among strains. Using SDO mice fed two humanized diets, we're assessing phenotypic variation in response to the diets. This ongoing project will enable more personalized nutrition strategies, improving dietary guidance for at-risk populations.

INTRASPECIES COMPETITION BETWEEN UROPATHOGENIC AND COMMENSAL *E. COLI*

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ABSTRACT

Uropathogenic *E. coli* (UPEC) causes most urinary tract infections, many of which recur due to the same strain. While some recurrent infections arise from bladder reservoirs, the intestine is the primary UPEC reservoir, where it coexists with commensal *E. coli*. How UPEC competes in this niche remains poorly understood. My work examines strain-specific UPEC–commensal interactions under varying oxygen and media conditions. UPEC shows a competitive advantage under anaerobic conditions, while aerobic agar competitions reveal strain-dependent outcomes. Phenotypic profiling and fluorescence-based high-throughput screening enable identification of key competitive genes.

UNRAVELING THE BEAUTY AND COMPLEXITY OF CHICKEN FEATHER PATTERNS: INTEGRATIVE GENOME- WIDE MAPPING AND SINGLE-CELL TRANSCRIPTOMICS

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ABSTRACT

This study investigated the genetic basis of feather pigmentation by carrying out a Mendelian cross between red junglefowl (RJF) and domestic Silver Sebright chickens. Whole-genome sequencing for over 250 F2s were performed. Five large-effect loci were implicated, including three known pigmentation-related genes (*SOX10*, *GJA5*, and *MC1R*) and two previously undocumented signals on chromosome 1 and chromosome 2. To explore molecular addresses and regulatory pathways, single-cell RNA sequencing was done on active feather follicles from four body regions (breast, leg, dorsal, and hackle) in an adult male RJF. Twelve major follicular cell types and body-region specific expression patterns were identified.

SIM2 REGULATION OF MITOCHONDRIAL DYSFUNCTION IN DOWN SYNDROME

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ABSTRACT

Down syndrome (DS) is a complex disorder associated with metabolic abnormalities such as obesity, liver fibrosis, and hypotonia. Using DS mouse models crossed with *mito-QC* and *Sim2*^{+/-} mice at young and old ages, we investigated mitochondrial dysfunction in skeletal muscle and liver, focusing on the role of *Sim2*. Through immunofluorescence, transmission electron microscopy, and indirect calorimetry, we assessed mitophagy, mitochondrial morphology, turnover, and systemic metabolism. Our findings indicate mitochondrial dysfunction and changes in mitochondrial shape and turnover in DS, and that *Sim2* normalization partially improves metabolic regulation. Our aim is to investigate how *Sim2* dosage change alters mitochondrial functioning.

IDENTIFYING MOLECULAR DIFFERENCES BETWEEN FLAT AND POLYPOID ADENOMAS IN COLORECTAL CANCER

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ABSTRACT

Flat colorectal adenomas are precursors to carcinoma that are easily missed during screening, yet the mechanism for why some individuals develop flat lesions rather than polypoid remains poorly understood. Previous work used I/LnJ and KK/HIJ mice developing 90% and 20% flat polyps respectively and found that tumor samples were not strain specific, but the normal adjacent colon was strain specific. An F2 mouse population using QTL showed that Chromosomes 7 and 9 were significantly associated with flat polyp morphologies. Current work is using a 10% flat polyp mouse strain (FVB) to add to QTL data and further elucidate genetic modifiers.

DEFINING ERBB-INDEPENDENT AND DEPENDENT MODULATORS OF COLORECTAL CANCER THROUGH THE USE OF MOUSE GENETICS

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ABSTRACT

The ERBB family of receptor tyrosine kinases – EGFR (ERBB1/HER1), ERBB2 (HER2), ERBB3 (HER3), ERBB4 (HER4) – recognizes diverse ligand signals to regulate essential cellular signaling pathways including RAS/RAF/MAPK, PI3K/AKT, and JAK/STAT. Dysregulation of this network has been implicated in colorectal cancer (CRC) progression and treatment response. Our lab has demonstrated roles for the ERBB family members in resistance to anti-EGFR therapy, EGFR-independent cancer progression, and genetic modification of the disease. Here, we outline prior findings in the C57BL/6J mouse strain to guide investigations of ERBB-independent and -dependent mechanisms in the 129S1/SvImJ background.

TOWARDS COMPLETE AND ACCURATE ANIMAL REFERENCE GENOMES: MAPPING UNASSIGNED SCAFFOLDS TO CHROMOSOMES BY OLIGO-FISH

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ABSTRACT

Accurate assignment of genomic scaffolds to chromosomes remains a major challenge in animal genome assemblies, particularly for regions enriched with repetitive DNA. Here, we focused on improving the completeness and accuracy of the alpaca (*Vicugna pacos*) reference genome, VicPac4, by applying Oligonucleotide Fluorescence *in situ* Hybridization (Oligo-FISH). This approach combines bioinformatic probe design with hybridization to metaphase chromosomes, enabling precise localization of sequences that are difficult to assign computationally and the discovery and chromosomal localization of this previously uncharacterized satellite specific to South American camelids. Importantly, the methodological framework is broadly applicable to other domestic and wild species, particularly those with incomplete assemblies providing a scalable model for improving genome assemblies across diverse taxa.

EXPLORING THE INTERPLAY BETWEEN SOCIAL ENVIRONMENTS, ETHANOL EXPOSURE, AND SLEEP IN *DROSOPHILA MELANOGASTER*

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ABSTRACT

Social context is a regulator of behavior and health, yet its interaction with ethanol remains incompletely understood. Ethanol can alter sleep and behavioral state, while prolonged social isolation shifts ethanol's behavioral valence and sensitivity. We examine social isolation as a phenomenon that interacts with ethanol to influence behavioral sensitivity. Using *Drosophila melanogaster* as a genetically tractable model for conserved behavioral and circadian regulation, we identify reproducible differences in ethanol responsiveness driven by social state rather than exposure alone. Future work will use gene expression analyses, relating ethanol effects to molecular pathways mediating circadian regulation and stress from chronic social isolation.

SYNTENY-AWARE EVALUATION OF CRISPR-Cas9 PAM CONSERVATION ACROSS DIVERGENT *DROSOPHILA MELANOGASTER* GENOMES

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ABSTRACT

CRISPR/Cas9 guide RNA design relies on a single reference genome, assuming conservation of target sites across individuals. Mutations between divergent strains can disrupt or create protospacer-adjacent motifs (PAMs), altering Cas9 activity. Here, we evaluate PAM conservation between the *Drosophila melanogaster* ISO1 reference genome and a high-quality de-novo assembly of a Cas9 expressing strain. Whole-genome alignments were used to define primary synteny blocks, within which all candidate SpCas9 target sites were mapped across both genomes. By integrating synteny assignments with Cutting Frequency Determination (CFD) scoring, we quantify how genetic variation impacts predicted CRISPR efficacy, highlighting limitations of single reference-based guide design.

THE ROLE OF ORCOKININ IN NUTRIENT HOMEOSTASIS IN *DROSOPHILA MELANOGASTER*

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The *Drosophila* *OK* gene encodes two distinct peptide isoforms, *OK-A* and *OK-B*, which are generated by tissue-specific alternative splicing and are predominantly expressed in the CNS and the gut, respectively. Intriguingly, *OK-A* and *OK-B* share no similarity and hence are likely to signal to two different receptors. Despite strong evolutionary conservation of OKs across arthropods, their physiological functions remain poorly understood. Here we show that OKs play a regulatory role in the acquisition of glycogen stores.

We generated isoform-specific knockouts using CRISPR-Cas9 gene editing, to assess which of the two OK isoforms is involved in glycogen homeostasis and we have identified a novel role of gut-derived Orcokinin in glycogen homeostasis. Together with CNS- derived FMRFa, our studies indicate that both gut and brain signaling are critical to maintain glycogen levels across multiple tissues.

A NEW APPROACH FOR PARKINSON'S TREATMENT: NEUROPROTECTIVE NANOMATERIALS ALLEVIATE AMYLOID BURDEN, REDUCE CELLULAR STRESS, AND EXTEND LIFESPAN

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ABSTRACT

Parkinson's disease (PD) is the fastest increasing neurological disorder and is characterized by the toxic build-up of alpha synuclein (α -syn) protein aggregates. Aggregates induce cellular stress, mitochondrial dysfunction, and eventual cell death. Transition metal dichalcogenide (TMD) nanoflowers (NFs) are 2D nanomaterials with innate biological properties that have shown an ability to increase cellular viability, reduce cellular stress pathways, upregulate mitochondrial biogenesis, and reduce amyloid burden. Administration into organismal models show no toxicity, an ability to significantly increase lifespan, and slow progression of α -syn aggregates. TMD NFs show potent promise in treating clinical PD as a neuroprotective therapeutic.

CHAOS IN THE GENOME: AN EDUCATIONAL VIDEO GAME PROJECT FOCUSING ON STUDENT INTEREST IN SCIENCE

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ABSTRACT

Chaos in the Genome is an educational video game designed to help improve middle and high school students' interest and enthusiasm towards science. Based on topics from biology and genetics, the video game will be a digital strategy-based, deck-building card game. Students will interact with a species' genome as it evolves, compete to outlive other players, and face challenges, like harmful environments. Currently, the game is in the playtesting stage using a paper prototype, focusing on assessing the mechanics of the game. The next step is to refine the paper prototype and begin outlining the first digital prototype.

QUANTITATIVE ESTIMATES OF TOXICODYNAMIC VARIABILITY FOR NEW APPROACH METHODOLOGIES- BASED SYSTEMIC SAFETY TOOLBOX USING A POPULATION-BASED HUMAN *IN VITRO* MODEL

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ABSTRACT

Next-Generation Risk Assessment (NGRA) frameworks leverage New Approach Methodologies (NAM) to support regulatory decision-making without animal testing. However, chemical-specific inter-individual variability largely relies on default uncertainty factors. This study tested a NAM-based approach to derive toxicodynamic variability factors (TDVF) using 131 human lymphoblastoid cells from European and African subpopulations across 53 chemicals. Eighteen chemicals showed effects below 300 μ M, yielding a median TDVF₀₅ of 3.8 [range 1-46], aligning with the default factor of 3.16. Genome-wide association study (GWAS) analysis identified xenobiotic metabolism genes underlying variability. This demonstrates that *in vitro* models can quantify inter-individual variability for NGRA, improving risk predictions.

LEVERAGING ION MOBILITY SPECTROMETRY-MASS SPECTROMETRY (IMS-MS) FOR RAPID EXPOSURE ASSESSMENT OF COMPLEX CHEMICAL MIXTURES

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ABSTRACT

Rapid and accurate analytical methods are essential for assessing complex chemical exposures, yet existing approaches are limited in efficiency and throughput. This study evaluated ion mobility spectrometry–mass spectrometry (IMS-MS) as a rapid alternative for characterizing chemical exposures in human serum. IMS-MS demonstrated enhanced speed, separation, and non-targeted capabilities, making it well-suited for monitoring complex chemical mixtures. Our goal is to apply IMS-MS methods to human serum samples to improve the characterization of mixed chemical exposures and strengthen biomonitoring and exposure assessment efforts.

FUNCTIONAL DISSECTION OF EXTRACELLULAR VESICLE- AND SOLUBLE FACTOR-MEDIATED RESPONSES TO THE AIRWAY EPITHELIAL SECRETOME: AN AIR-LIQUID INTERFACE PLATFORM FOR INHALATION TOXICOLOGY

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ABSTRACT

Airway epithelial cells are a frontline interface, orchestrating barrier defense and immunomodulation through intercellular communication. However, the contribution of extracellular vesicles (EVs) relative to soluble factors in response to inhaled toxicants remains poorly defined. We established an air–liquid interface platform using human bronchial epithelial (16HBE) cells to characterize secretome-mediated signaling following TNF- α exposure. Conditioned media was fractionated into EV-enriched and soluble factor pools and applied to naïve cells. Soluble factors elicited the strongest cytokine and transcriptional responses, whereas EV-enriched fractions induced minimal bioactivity. This platform enables functional resolution of epithelial signaling and extension to relevant inhalation exposures in disease pathogenesis.

OPTIMIZATION OF ENDOTHELIAL NETWORK FORMATION AND IMMUNE-ENDOTHELIAL SIGNALING USING COMMERCIAL MICROPHYSIOLOGICAL VASCULAR DEVICES

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ABSTRACT

Physiologically relevant vascular models are needed to study *in vitro* inflammatory responses to chemical exposure. Microphysiological systems (MPS) offer promising platforms but vary in their ability to support immune-vascular interactions. This project investigates endothelial network formation and inflammatory activation across two MPS: the IdenTX-40 chip and the Mimetas Organoplate®. Using endothelial progenitor cells and HUVECs, we examined how media composition and fibroblast support influence vessel stability and ICAM-1 expression. Due to the IdenTX-40 format, immune cell adhesion was assessed in the Organoplate® following inflammatory stimulation. Together, these complementary platforms support controlled modeling of vascular formation, inflammation, and immune cell trafficking.

FROM SOURCE TO BODY: DISPARITIES IN ARSENIC EXPOSURE FROM TEXAS WATER SYSTEMS TO NATIONAL BIOMONITORING

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ABSTRACT

Arsenic is a naturally occurring groundwater contaminant and a known human toxicant associated with cancer and other adverse health outcomes. Although drinking water regulations have reduced arsenic levels, it remains unclear whether exposure reductions have been equitable. This study integrates arsenic monitoring data from Texas public water systems with national biomonitoring data to evaluate temporal trends and disparities in exposure. While overall arsenic levels declined over time, higher exposures persisted among rural, socioeconomically disadvantaged, and socially vulnerable populations. These findings highlight persistent inequities in environmental exposure and emphasize the need for targeted public health interventions to reduce arsenic-related health risks.

EXPANDING CLAY-BASED REMEDIATION STRATEGIES TO REDUCE AFLATOXIN M1 CONTAMINATION IN MILK

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ABSTRACT

Exposure to aflatoxins through contaminated milk is a significant food safety and public health concern. Aflatoxin B1 (AFB1), ingested from mold-contaminated crops, is metabolized into aflatoxin M1 (AFM1), which is excreted into milk and contributes to dietary cancer risk. As climate variability increases aflatoxin production, effective mitigation technologies remain limited. This project investigates post-lactational clay-based remediation by adding calcium montmorillonite binders directly to contaminated milk. Preliminary in vitro studies show approximately 68% reduction of AFM1, supporting the potential of this approach for milk decontamination.

ECHA WRITES BACK: THE REASONS FOR REJECTION OF READ-ACROSS IN COMPLIANCE CHECK DECISIONS BY THE EUROPEAN CHEMICALS AGENCY

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ABSTRACT

The European Chemicals Agency evaluates chemical safety data under EU REACH regulations, requiring compliance checks on at least 20% of registrations. Analysis of nearly 2,800 compliance check decisions conducted through 2025 identified more than 30% (1,200) make use of read-across—a method that uses similar chemical data to make decisions chemical safety. Using 17 assessment criteria capturing structural, toxicokinetic, and toxicodynamic factors, each of the rejected read-across hypotheses were evaluated to determine the reasons for rejection. This systematic evaluation of rejection reasons provides clear guidance for improving future submissions and advancing New Approach Methods in toxicology.

EXTRACELLULAR VESICLES MEDIATE INFLAMMATORY SIGNALING IN ARSENIC-EXPOSED BRONCHIAL EPITHELIAL CELLS

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ABSTRACT

Exposure to arsenic from drinking water is a known carcinogen with an association to respiratory diseases following ingestion, especially to rural residents in South Texas. Extracellular vesicles (EVs) are cell-derived structures that represent biomarkers of exposure. Developing novel biomarkers is essential to identify susceptible populations. We exposed 16HBE cells to NaAsO₂ for 24-hours to establish mechanisms underlying inflammation at the gene and protein level. *IL-1β* was increased after 24-hours and IL-1β had an increased trend at 10μM. For the EV exposure, *IL-1β* was increased at 10μM. EVs isolated from exposed 16HBE cells may serve as novel biomarkers of respiratory inflammation.

METHOD DEVELOPMENT FOR EXPOSURE ASSESSMENT USING PASSIVE SAMPLING DEVICES AND ION MOBILITY SPECTROMETRY-MASS SPECTROMETRY

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ABSTRACT

Understanding personal exposure to environmental chemicals is critical. This study demonstrates the use of silicone wristbands as passive samplers combined with ion mobility spectrometry–mass spectrometry (IMS-MS) and a curated collision cross section (CCS) database to identify environmental chemicals. An IMS-MS workflow incorporating CCS values for over 2,000 compounds was developed and optimized using mock exposure studies and multiple ionization modes. The method was validated with wristbands worn by volunteers, showing alignment with expected exposures. This approach will be applied to real-world samples from a human cohort, including agricultural and hospital work environments, enabling scalable, confident detection of diverse contaminants.

TARGETED LIPIDOMICS AS A HIGH-THROUGHPUT NEW APPROACH METHOD FOR MECHANISM-BASED STUDIES OF HEPATOTOXICITY IN VITRO

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ABSTRACT

This study aimed to enhance the translational relevance of in vitro hepatotoxicity studies by incorporating mechanistic phenotypic readouts using a fast targeted lipidomic approach. A panel of hepatotoxicity-relevant lipids was analyzed in human hepatocytes exposed to multiple concentrations of fialuridine, bosentan, and chlorpromazine. Chlorpromazine and bosentan induced similar alterations in neutral lipid and phospholipid metabolism, suggesting membrane perturbation, whereas bosentan additionally increased the pro-apoptotic lipid ceramide. Although sensitivity to fialuridine was limited in the current design, suppression of ATP-dependent pathways was observed, consistent with mitochondrial toxicity. Overall, we provide a promising strategy for the mechanistic evaluation of in vitro hepatotoxicity.