Thirteenth Annual Arkansas Undergraduate Summer Research Symposium

at the University of Arkansas for Medical Sciences

I. Dodd Wilson Education Building, Lecture Hall, Room 126
University of Arkansas for Medical Sciences
Little Rock, Arkansas
Wednesday, July 29, 2026
8:30 AM – 4:00 PM

Pick up registration materials between 7:30 AM – 8:30 AM

UAMS GRADUATE SCHOOL
UNIVERSITY OF ARKANSAS FOR MEDICAL SCIENCES

UAMS COLLEGE OF MEDICINE
DEPARTMENT OF BIOCHEMISTRY AND MOLECULAR BIOLOGY
UNIVERSITY OF ARKANSAS FOR MEDICAL SCIENCES

Arkansas INBRE
IDeA Network of Biomedical Research Excellence

NATIONAL INSTITUTES OF HEALTH

SIGMA XI

MCE MedChemExpress

Thirteenth Annual Arkansas Undergraduate Summer Symposium Program

All events will take place in the I. Dodd Wilson Education Building

7:30 – 8:30 am	Pick up registration materials at the Registration desk / Validate parking *(Atrium)*
	(Kathy Carlson/Kahla Robinson/Veronica Overton)
	Arrival and poster set-up
8:30 am	Welcome and Opening Remarks *(Lecture Hall 126)*
	Grover P. Miller, PhD
8:45 – 10:15 am	Oral Platform Session I / Odd Talks T1, T3, T5, T7, T9, T11 *(Lecture Hall 126)*
	Session I Chair: Katie Ryan, PhD
10:15 – 10:30 am	Break
10:30 – 11:30 pm	Poster Platform Session I – Odd-numbered posters *(Atrium)*
11:30 – 12:15 pm	Lunch (*free for registrants only*) (*115 A & B)*
	& Finding your future in Graduate level research (*115 A & B/Atrium)*
12:15 – 1:15 pm	Poster Platform Session II – Even-numbered posters *(Atrium)*
1:20 – 2:50 pm	Oral Platform Session II / Evan Talks T2, T4, T6, T8, T10, T12 *(Lecture Hall 126)*
	Session II Chair: Alicia Byrd, PhD
2:50 – 3:00 pm	Break
3:00 pm	Introduction of the speaker *(Lecture Hall 126)*
	Grover P. Miller, PhD
3:05 – 3:45 pm	"Discovering Pathways: Decoding the Networks of Life" *Drew Jones, Ph.D. Assistant Professor* *Biochemistry and Molecular Pharmacology* *NYU Langone Health*
3:45 pm	Student recognition and Closing remarks.
	Alicia Byrd, PhD and Grover P. Miller, PhD

Poster set-up: Each poster has been assigned a number that corresponds to a specific poster board.

Participants should put up posters prior to 8:30 am if possible.

Oral Platform Session I: Each speaker will have 12 minutes followed by 3 minutes for questions.

Poster Platform Session I: Those students who have been assigned a poster board with an ***ODD number*** are requested to stand by their posters from **10:30 am to 11:30 am.**

Lunch: Lunch will be served in Room 115 A/B (*lunch is free for registrants only)*

Poster Platform Session II: Those students who have been assigned a poster board with an ***EVEN number*** are requested to stand by their posters from **12:15 pm to 1:15 pm.**

Oral Platform Session II: Each speaker will have 12 minutes followed by 3 minutes for questions.

Organizer: Grover P. Miller, PhD

Organized and Sponsored by the Department of Biochemistry and Molecular Biology at the University of Arkansas for Medical Sciences, along with the Graduate School, the Center for Molecular Interactions in Cancer (P20 GM152281), and the Arkansas INBRE program, supported by grant funding from the National Institute of General Medical Sciences (P20 GM103429).

Special thanks to the Departmental staff who have made this meeting possible:
Kathy Carlson, Kahla Robinson, and Veronica Overton

We also thank Joseph Utecht in the Department of Otolaryngology Center for Hearing Health Equity for helping put together this program.

Upcoming Events

Arkansas INBRE 2026

The 2026 AR INBRE conference will be held on Friday, November 6, and Saturday, November 7, at Embassy Suites in Rogers and on the University of Arkansas campus.

More information will be provided in October 2026.

Sigma Xi

International Forum on Research Excellence
powered.by.Sigma.Xi
A Virtual Event
November 6–7, 2026

Talk Session I / odd number talks

8:45 am – 10:15 am

T1 **Elena Coleman**
Dr. Rupak Pathak[1]
"GPVI Deficiency Limits Total Body Irradiation-Induced Platelet–Immune Cell Crosstalk"
University of Arkansas for Medical Sciences[1]

T3 **Lillie Ballany**
Lillie Ballany[1], Joseph S. Beard[2], Alicia K. Byrd[2]
"Suppressor of Cancer Cell Invasion inhibits DNA unwinding by DNA Helicase B"
University of Arkansas - Fayetteville[1]
University of Arkansas for Medical Sciences[2]

T5 **Mia Canizares**
Mia Canizares[1], Somaya Y. Ibrahim[1]
"A novel target of chemotherapy-induced cardiotoxicity in valvular interstitial cells"
University of Arkansas for Medical Sciences[1]

T7 **Owen Bussell**
Owen Bussell[1]
"Recycling carbon dioxide into sustainable fuels with Mn-NHC electrocatalyst"
University of Central Arkansas[1]

T9 **Bailee Brinkley**
Bailee Brinkley[1]
"Investigating New Methods to Detect Gammaherpesviruses in vivo"
Arkansas Tech University[1]

T11 **Fariha Alam Bushra**
Fariha Alam Bushra[1], Md Rafikul Islam, Preeti Nagar, and Mohammad Rahman[1]
"Sense Oligonucleotide Pharmacology: A Potential Therapeutic Strategy to Correct Oncogenic Splicing"
University of Arkansas for Medical Sciences[1]

Poster Session I / odd number posters

10:30 am – 11:30 am

P1 **Benjamin Anderson**

Benjamin Anderson[1]

"Beyond Flat Cell Culture: 3D-Printed Villus-Crypt Scaffolds for Intestinal Modeling"

University of Arkansas - Little Rock[1]

P3 **Lily Butler**

Lily Butler[1], Nathan Avaritt[1], Dr. Allen Tackett[1]

"Pharmacologically activating ATF6 to enhance Immunotherapy response in Melanoma"

University of Arkansas for Medical Sciences[1]

P5 **Noah Canon**

Noah Canon[1], Joseph Bradshaw[1]

"[MnIII(TPP-HEP)Cl]: A Novel Water-Soluble MRI Contrast Agent for Brain Imaging"

Ouachita Baptist University[1]

P7 **Georgie Crain**

Georgie Crain[1], Kaley Haskins[1]

"Nutrition Assessment of Older Adults"

Ouachita Baptist University[1]

P9 **Willow Danielle**

Willow Danielle[1], Sandhya Krishnan[1], Mark Baillie[1]

"Social network analysis of STEM faculty communities in pedagogical learning experiences"

University of Arkansas - Little Rock[1]

P11 **Mason DeLaMater**

Dr. Mark Smeltzer[1], Dr. Nathanial Torres[1], Mason DeLaMater[1]

"Differences in the Staphylococcus aureus sarA/sigB/agr regulatory axis define alternative pathways in the pathogenesis of osteomyelitis"

University of Arkansas for Medical Sciences[1]

P13 **Praise Fabiyi**

Praise Fabiyi[1], Thanh Ha Vy Nguyen, Noemi Garcia Garcia, Emily Frances Cox, and Katie R. Ryan1[2]

"Rnd3 regulates metastatic hallmarks in lung adenocarcinoma through integrin Alpha 5 modulation"

University of Arkansas - Pine Bluff[1]

University of Arkansas for Medical Sciences[2]

P15 **Brooke Garlington**

Brooke Garlington[1], Rana M. Elbishbishy[2], Tamer S. Kaoud, Ruetaichanok Naowaphankun, Maria N. Cajimat, Dennis A. Bente, Ju-Hyeon Lee, Ramakirishna Edupuganti, Kevin N. Dalby, Joseph Oleniczak, Robert C. Chin, Mark Hickman

"Discovery of Novel Antiviral Drug Candidates for Hantavirus Infection"

University of Arkansas - Little Rock[1]

University of Arkansas for Medical Sciences[2]

P17 **Luke Garrett**

Luke Garrett[1], Robert Eoff[2], Amit Ketkar[2]

"Examining the impact of Rev1 interaction on DHX36 helicase activity"

Hendrix College[1]

University of Arkansas for Medical Sciences[2]

P19 **Sam Grose**

Sam Grose[1]

"Piloting a Biocube CURE Project"

Ouachita Baptist University[1]

P21 **Kaley Haskins**

Kaley Haskins[1], Georgie Crain[1]

"Comparison of the Body Mass Indices of Children Participating in a Summer Nutrition Education Program to Children Not Participating in a Summer Nutrition Education Program"

Ouachita Baptist University[1]

P23 **Murphy Hernandez**

Murphy Hernandez[1], Dr. Clay Jackson-Litteken[2]

"Role of Acinetobacter baumannii Regulators in Pneumonia Pathogenesis"

Harding University[1]

University of Arkansas for Medical Sciences[2]

P25 **Madison Holmes**

Madison Holmes[1], Sharon Hamilton[1]

"Optimization of Cross-Linked Pyranine-Loaded Electrospun Nanofiber Mats for Visual Wound pH Detection"

Ouachita Baptist University[1]

P27 **Jada-Lynn King**

Jada-Lynn King[1], Dr. Tudor Moldoveanu[1], Dr. Perry Caviness[1]

"Direct Activation of BCL-2 Protein, BAK, to Initiate Apoptosis in Cancer Cell Lines"

University of Arkansas for Medical Sciences[1]

P29 **Ava Lang**

Ava Lang[1], Prisha Warikoo[2], Mulu Tesfay[2]

"In Vitro Characterization of Recombinant VSV (VSV-Maraba-G)"

Arkansas State University[1]

University of Arkansas for Medical Sciences[2]

P31 **Georgia Liu**

Georgia Liu[1], Teresita Bellido[1], Kait Hapke[1]

"Role of the CYBA gene in diabetes-induced bone disease"

University of Arkansas for Medical Sciences[1]

P33 **Avery Maxwell**

Avery Maxwell[1]

"Developing eDNA tools to identify the geographical distribution of endangered madtoms in Arkansas"

Ouachita Baptist University[1]

P35 **Maci McJunkins**

Maci McJunkins[1], Sharon Hamilton[1], Sara Hubbard[1]

"A Toxic Tale: An XRF Analysis of Arsenic in Victorian Bookbindings"

Ouachita Baptist University[1]

P37 **Lydia Morales-Castillo**

Lydia Morales-Castillo[1], Sonja Ramirez[1]

"Optimization of the Purification of the TraI Helicase"

Lyon College[1]

P39 **Akshath Narravula**

Akshath Narravula[1]

"Dose and Time Dependent Disruption of Intestinal Barrier Genes by a Prohibited Antifungal"

NCTR[1]

P41 **Joan Obi**

Joan Obi[1], Bethany Paxton[2], Jesus Delgado-Calle[2]

"Investigating the Role of Notch3 in Dormant Multiple Myeloma Cells."

University of Arkansas - Fayetteville[1]
University of Arkansas for Medical Sciences[2]

P43 **Jessica Offordum**

Jessica Offordum[1], Shimaz Hoque[2], Mojnu Miah[2]

"Investigating Histone Modification Changes At FAM60A regulated gene promoters"

Grambling State University[1]
University of Arkansas for Medical Sciences[2]

P45 **Zhychaeus Powell**

Zhychaeus Powell[1]

"TUNEL-Positivity in Acute Kidney Injury:"

Philander Smith University[1]

P47 **Javier Ramirez Santamaria**

Javier Ramirez Santamaria[1]

"Comparative Study of Bipyridine and Mebim-pic Ligands in Homogeneous Rhenium Catalysts for Enhanced Captured CO_2 Reduction"

University of Central Arkansas[1]

P49 **Andrea Richards**

Andrea Richards[1], Leonard Harris[1]

"Investigating the Impact of Microhomology-Mediated End Joining Repair in Fanconi Anemia Using Computational Modeling"

University of Arkansas - Fayetteville[1]

P51 **Kiah Rucker**

Kiah Rucker[1], Dr. Ping-Ching Hsu[2]

"Assessment of Urinary Heavy Metals Concentrations and Geospatial Exposure in the Arkansas Rural Community Health Study"

Philander Smith Univeristy[1]
University of Arkansas for Medical Sciences[2]

P53 **Ian Scarth**

Ian Scarth[1], Karen Beenken, PhD[2]

"Evaluating a DDM-Assisted Sample Preparation Method for Quantitative Proteomics of Melanoma Cells"

University of Hawai'i at Hilo[1]
University of Arkansas for Medical Sciences[2]

P55 **Dishant Sharma**

Dishant Sharma[1], Isabelle Miousse[2], Sarita Garg[2]

"Empagliflozin Reprograms Mitochondrial Metabolism and Redox Pathways in vivo"

University of Southern California[1]
University of Arkansas for Medical Sciences[2]

P57 **Akul Shrivastava**

Akul Shrivastava[1], Heather Downs[1], Dylan Gilbreath, Linda Larson-Prior[2]

"Using EEG-derived ERPs to Determine the Effect of Maternal Weight on Pediatric Neurodevelopment"

University of Arkansas for Medical Sciences[1]
Other, University of Arkansas for Medical Sciences[2]

P59 **Paige Spicer**

Paige Spicer[1], Dr. Sharon Hamilton[1]

"Optimization of Amoxicillin-Loaded Electrospun Nanofiber Mats for Controlled Drug Delivery to Prevent Wound Infection"

Ouachita Baptist University[1]

P61 **Godistruth Umeakuekwe**

Godistruth Umeakuekwe[1], Dr. Ryan Allen[2]

"Determining the Influence of Human Lipoproteins and Serum on Abscess Formation and Dispersal in GFP-Expressing Staphylococcus aureus U1-sarA"

University of Arkansas - Pine Bluff[1]
University of Arkansas for Medical Sciences[2]

P63 **Rylan Ward**

Rylan Ward[1], Mark Manzano[2], Prasanth Viswanathan[2]

"Investigating the Impact of Peroxisomal Gene Knockout on Murine gammaherpesvirus-68 (MHV68) Replication"

University of Central Arkansas[1]
University of Arkansas for Medical Sciences[2]

P65 **Jacee Wheat**

Jacee N. Wheat[1], Martin Morales[2], Robert L. Eoff[2]

"Inhibition of DNA polymerase kappa to improve antiproliferative effects of temozolomide on glioblastoma cells"

Hendrix College[1]
University of Arkansas for Medical Sciences[2]

P67 **Rhys Wolter**

Rhys Wolter[1], Dr. Robert Eoff[2], Dr. Qudes Al-Anbaky[2]

""Investigation of the cellular mechanism of action for a small-molecule inhibitor of DNA polymerase kappa""

Episcopal Collegiate School[1]
University of Arkansas for Medical Sciences[2]

P69 **Youtong Zhang**

Isabella Zhang[1], Benjamin H. May[1], Alicia K. Byrd[1]

"Identification of an inhibitor of HELB helicase"

University of Arkansas for Medical Sciences[1]

P71 **Chelsea Cliff**

Chelsea Cliff[1], Ava Lang[1], Prisha Warikoo[1]

"In Vitro Characterization of Recombinant VSV Expressing MMR Glycoproteins for Pancreatic Cancer Therapy"

University of Arkansas for Medical Sciences[1]

P73 **Bennett Dishongh**

Bennett Dishongh[1], Emily Frances Cox, Noemi Garcia Garcia, Thanh Ha Vy Nguyen, and Katie R. Ryan[2]

"CST4 as a Novel Regulator of Triple Negative Breast Cancer"

Hendrix College[1]
University of Arkansas for Medical Sciences[2]

P75 **Kaylee Francis**

Kaylee Francis[1], Dr. Robert L. Eoff[1], Dr. Amit Ketkar[1]

"REV1 facilitates DHX36 localization in response to G-quadruplex stabilization during nascent strand synthesis"

University of Arkansas for Medical Sciences[1]

P77 **Mishel Ocho**

Mishel C. Ocho[1], William E. Fantegrossi[1], Brenda M. Gannon[1]

"Rewarding effects of compounds commonly found in kratom products"

University of Arkansas for Medical Sciences[1]

P79 **Clara Park**

Clara Park[1], Jun Gao[2], Eric J. Enemark[2]

"A conserved aromatic residue that structurally mediates DNA melting is dispensable in vivo"

Rhodes College[1]
University of Arkansas for Medical Sciences[2]

Poster Session II / even number posters

12:15 pm – 1:15 pm

P2 **Cayden Bailey**
Tamara S. Haselkorn[1], Cayden Bailey[1]
"The epidemiology of chlamydiae symbionts in natural populations of social amoebae"
University of Central Arkansas[1]

P4 **Danny Caceres**
Danny Caceres[1], Linda Larson-Prior[2], Darcy Hagood
"The impact of diet and lifestyle on cognitive function in children"
Hendrix College[1]
University of Arkansas for Medical Sciences[2]

P6 **Angelica Concepcion**
Angelica Concepcion[1], Fiona Goggin[1], Jiamei Li[1]
"How Plants Fight Back: The Role of Reactive Oxygen Species in Defense Against Aphids"
University of Arkansas - Fayetteville[1]

P8 **Savea Cunningham**
Savea Cunningham[1], Dr. Tudor Moldoveanu[2], Jackson Lewis[2]
"Investigating MLKL's Membrane Poration in Necroptosis"
University of the Ozarks[1]
University of Arkansas for Medical Sciences[2]

P10 **Paige Davies**
Paige Davies[1], Dr. Sara E. Hubbard[1]
"Analysis of the Leaching of Bisphenol-A from Feminine Hygiene Products into a Synthetic Vaginal Fluid Solvent using Fluorescence Spectrophotometry"
Ouachita Baptist University[1]

P12 **Lawson Duke**
Lawson Duke[1]
"Enhancing Low-Input Protein Recovery in the Chloroform Methanol Extraction Workflow Using SP3 Paramagnetic Beads in Bottom-Up Proteomics"
University of South Carolina[1]

P14 **Aniyah Freeman**
Aniyah Freeman[1], Priya Yadav + Swathi Nedunchezian + Flavelia Stigger + Sankar Devarajan[1], Shengyu Mu[2]
"Antioxidant, Anti-inflammatory, and Anti-diabetic Properties of Rice Bran from Arkansas-Grown Rice Cultivars"
University of Arkansas - Pine Bluff[1]
University of Arkansas for Medical Sciences[2]

P16 **Jansen Garner**
Jansen Garner[1], Dr. Ruth Plymale[1]
"Preliminary Characterization of Pineapple and White Sugar Kombucha Microbiomes"
Ouachita Baptist University[1]

P18 **Yazi Greer**
Yazi Greer[1], Sara E. Hubbard[1]
"Analysis of BPA in Athletic Clothing Using Fluorescence Spectroscopy"
Ouachita Baptist University[1]

P20 **Hannah Harshaw**

Hannah Harshaw[1], Joseph Bradshaw[1]

"Development of [MnIII(TPP-PEG3)Cl] as a Novel Water-Soluble MRI Contrast Agent"

Ouachita Baptist University[1]

P22 **Selah Havens**

Selah Havens[1]

"Spheroid Formation of Invasive Lung Cancer Cell Lines and Their Effects on Tumor Microenvironment"

Ouachita Baptist University[1]

P24 **Audrey Hill**

Audrey Hill[1], Dr. Craig Porter[1]

"Does Severe Burn Injury Result in Skeletal Muscle Creatine Depletion?"

University of Arkansas for Medical Sciences[1]

P26 **Betty Hu**

Mengye Hu[1]

"*The Role of SMARCAL1-Mediated Replication Fork Reversal in TatDN1-dependent Replication Fork Protection"*

University of Arkansas for Medical Sciences[1]

P28 **Sloka Kovi**

Sloka Kovi[1], Somaya Y. Ibrahim[1]

"Characterization of the molecular phenotype of a murine model of diabetic cardiomyopathy"

University of Arkansas for Medical Sciences[1]

P30 **Andrew Lever**

Andrew Lever[1]

"Sustainable Polyaddition of Diamine-Bridged Crotonate Monomers"

University of Arkansas - Fayetteville[1]

P32 **Tamara Lowe**

Tamara Lowe[1], Alex Mcdonald[2], Yuet-Kin Leung, PhD[2]

"The Role of Estrogen- Related Receptor Alpha in Androgen Receptor Signaling"

University of Arkansas - Little Rock[1]
University of Arkansas for Medical Sciences[2]

P34 **Ravyn Mays**

Ravyn Mays[1], Miranda Wallace[2]

"A Saccharomyces boulardii chassis to deliver therapeutic proteins that inhibit diarrheal pathogens in the GI Tract"

University of Arkansas - Little Rock[1]
University of Arkansas for Medical Sciences[2]

P36 **Milo Moore**

Milo Moore[1], Isabelle Rose[1], Andrea A. Duina[1]

"Using the yeast model system to gain insights into a recently described neurodevelopmental condition in humans"

Hendrix College[1]

P38 **Mohammed Naif**

Mohammed Naif[1], Sudip Panday, Allison Abney[2], Amy Sato[2]

"Mice with Duchenne Muscular Dystrophy exhibit increased susceptibility to Glucocorticoid-Induced Bone Fragility"

University of Arkansas - Little Rock[1]
University of Arkansas for Medical Sciences[2]

P40 **Maddie Nelson**

Maddie Nelson[1]

"The Megacancer Project; Selection and Characterization of Highly Invasive H460 Lung Cancer Cell Lines"

Ouachita Baptist University[1]

P42 **Devyn O'Daniel**

Devyn O'Daniel[1]

"The roles of sexual selection and natural selection in a polyphenic nematode"

Arkansas State University[1]

P44 **Samuel Paez**

Samuel Paez[1]

"Alchemically Assessing Boiling Point Elevation Via Water Chemical Potential in Sodium Chloride Solutions with Molecular Dynamics"

John Brown University[1]

P46 **Sonja Ramirez**

Sonja Ramirez[1], Lydia Morales-Castillo[1]

"Optimization of the Purification of the TraI Helicase"

Lyon College[1]

P48 **Simra Rana**

Simra Rana[1]

"Computational Modeling of Interstrand Crosslink Repair in Fanconi Anemia"

Haas Hall Academy Fayetteville[1]

P50 **Jordan Rodgers**

Jordan Rodgers[1]

"Benzimidazole-based N-Heterocyclic Carbene (NHC) Ligand Synthesis"

University of Central Arkansas[1]

P52 **Lídize Sagarnaga Rivera**

Lídize Sagarnaga Rivera[1], Dr. Virginie Rolland[1], Dr. Lori Neuman-Lee[1]

"Skin pattern as a potential monitoring tool for frog populations"

Arkansas State University[1]

P54 **Isabella Sellers**

Bella Sellers[1], Jenna Caswell[1], Mitchell R. McGill[1]

"What is a "therapeutic" dose of acetaminophen in mice? A pharmacokinetics investigation."

University of Arkansas for Medical Sciences[1]

P56 **Himanshi Shorie**

Himanshi Shorie[1], Prabha Thenuwari Dayawansha[2], Dr. Paul Adams[2]

"Bacterial Expression and Purification of Cdc42 and PBD46"

University of Arkansas - Fort Smith[1]
University of Arkansas - Fayetteville[2]

P58 **Joshua Skipworth**

Josh Skipworth[1], Isabel L. Ritter & Marina Avram, Justin Pressley, Jeffery Moran, & William Fantegrossi[2]

"Fentanyl and fentanyl analog quantification via blood micro-fluidic sampling and LC-MS/MS"

University of Central Arkansas[1]
University of Arkansas for Medical Sciences[2]

P60 **Christine Tan**

Christine Tan[1], Qingfang He[1]

"Impact of Environmental Stressors on Biofuel Production in Cyanobacteria"

University of Arkansas - Little Rock[1]

P62 **Ashley Wallace**

Ashley Wallace[1], Kirk L. West[2]

"Production of Phosphorylated Recombinant Peptides"

University of Arkansas - Monticello[1]

University of Arkansas for Medical Sciences[2]

P64 **Ella Watson**

Ella Watson[1], Samrat Choudhury[2], Arundhati Chavan[2]

"Epigenetic Regulation of CDK9 in KMT2A-Rearranged Acute Myeloid Leukemia"

University of Arkansas - Fayetteville[1]

University of Arkansas for Medical Sciences[2]

P66 **Jadyn Wilhite**

Jadyn Wilhite[1], Thomas Williams[2]

"CD28-Mediated Regulation of PCK2 Expression and T Cell Metabolic Adaptation"

Harding University[1]

University of Arkansas for Medical Sciences[2]

P68 **Kalyn You**

Kalyn You[1]

"Effects of a Single Lysergic Acid Diethylamide (LSD) Administration on Negative Affective Behaviors Elicited by Daily Unpredictable Mild Stress in Mice"

University of Arkansas - Little Rock[1]

P70 **Aidan Zhao**

Aidan Zhao[1], Sergio Cortes[1], Yuet-Kin Leung[1]

"GPER1 Agonist G-1 Inhibits Anchorage-Independent Growth on Castration Resistant Prostate Cancer in Vitro"

University of Arkansas for Medical Sciences[1]

P72 **Lillie Ballany**

Lillie Ballany[1], Joseph S. Beard[2], Alicia K. Byrd[2]

"Suppressor of Cancer Cell Invasion inhibits DNA unwinding by DNA Helicase B"

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Dr. Rupak Pathak[1]

"GPVI Deficiency Limits Total Body Irradiation-Induced Platelet–Immune Cell Crosstalk"

University of Arkansas for Medical Sciences[1]

P78 **Rebekah Caffey**

Rebekah Caffey[1], Meghan L. Ruebel[1], Laxmi Yeruva[1]

"EPA and DHA supplementation with HFD during pregnancy leads to changes in circulating and liver lipid metabolism."

Microbiome and Metabolism Research Unit, USDA-ARS, SEA; Arkansas Children's Nutrition Center, Little Rock, AR, USA[1]

Talk Session II / even number talks

1:20 pm – 2:50 pm

T2 **Clara Park**

Clara Park[1], Jun Gao[2], Eric J. Enemark[2]

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"REV1 facilitates DHX36 localization in response to G-quadruplex stabilization during nascent strand synthesis"

University of Arkansas for Medical Sciences[1]

T12 **Spencer Stutts**

Spencer Stutts[1], Dr. Patrick Pysz[1], Dr. Julie Stenken[1]

"Development of Low Weight and Power Microfluidic Pumping Systems for a Portable Microsampling Backpack"

University of Arkansas - Fayetteville[1]

Attendee Index

First Name	Last Name	Institution	Research Program	Role / Session
1. Het	Adhvaryu	University of Arkansas for Medical Sciences (UAMS)		Judging Only
2. Qudes	Al-anbaky	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
3. Ryan	Allen	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
4. Aisha	Al-Rizzo	New York Institute of Technology College of Osteopathic Medicine		Guest
5. Benjamin	Anderson	University of Arkansas - Little Rock	INBRE - IDeA Network of Biomedical Research Excellence	P1 Poster
6. Trinity	Armstrong	Philander Smith University		Guest
7. Haile	Assress	University of Arkansas for Medical Sciences (UAMS)		Judging Only
8. Aanya	Asudani	Sigma XI		Guest
9. Nathan	Avaritt	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
10. Marina	Avram	University of Arkansas for Medical Sciences (UAMS)		Guest
11. Rushita	Bagchi	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
12. Cayden	Bailey	University of Central Arkansas	Other	P2 Poster
13. Mark	Baillie	University of Arkansas - Little Rock		Faculty Mentor
14. Lillie	Ballany	University of Arkansas - Fayetteville	SURF (Biochemistry and Molecular Biology, UAMS)	T3 Oral, P72 Poster
15. Joey	Beard	University of Arkansas for Medical Sciences (UAMS)		Judging Only
16. Karen	Beenken	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
17. Sibes	Bera	University of Arkansas for Medical Sciences (UAMS)		Guest
18. Debasree	Bhadra	University of Arkansas for Medical Sciences (UAMS)		Judging Only
19. Anna	Bolding	University of Arkansas - Fayetteville		Guest
20. Detri	Brech	Ouachita Baptist University		Faculty Mentor
21. Bailee	Brinkley	Arkansas Tech University	INBRE - IDeA Network of Biomedical Research Excellence	T9 Oral
22. Saanvi	Buddha			Guest
23. Fariha Alam	Bushra	Southeast Missouri State Univrsity	SURF (Biochemistry and Molecular Biology, UAMS)	T11 Oral, P74 Poster

First Name	Last Name	Institution	Research Program	Role / Session
24. Owen	Bussell	University of Central Arkansas	American Chemical Society - Petroleum Research Fund	T7 Oral
25. Lily	Butler	Episcopal Collegiate School	Jackson T. Stephens Summer Science Scholars	P3 Poster
26. Alicia	Byrd	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
27. Danny	Caceres	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	P4 Poster
28. Rebekah	Caffey	Hendrix College	USDA-ARS/ACNC Microbiome and Metabolism Research Unit	P78 Poster
29. Mia	Canizares	Mount St. Mary Academy	Physiology and Cell Biology	T5 Oral
30. Noah	Canon	Ouachita Baptist University	OBU Summer Research Program	P5 Poster
31. Kathy	Carlson	University of Arkansas for Medical Sciences (UAMS)		Administrator
32. Jingyi	Chen	University of Arkansas - Fayetteville		Judging Only
33. Chelsea	Cliff	Howard University	SURP - Summer Undergraduate Research Program (UAMS)	T8 Oral, P71 Poster
34. Brooks	Coleman	n/a	SURP - Summer Undergraduate Research Program (UAMS)	Guest
35. Elena	Coleman	Rhodes College		T1 Oral, P76 Poster
36. Angelica	Concepcion	University of Arkansas - Fayetteville	STEAM - Science, Technology, Engineering, Art and Math	P6 Poster
37. Emily	Cox	University of Arkansas for Medical Sciences (UAMS)		Guest
38. Georgie	Crain	Ouachita Baptist University	OBU Summer Research Program	P7 Poster
39. Savea	Cunningham	University of the Ozarks	SURF (Biochemistry and Molecular Biology, UAMS)	P8 Poster
40. Willow	Danielle	University of Arkansas - Little Rock	UALRS (Upholding Active Learning Reform in STEM) Project	P9 Poster
41. Paige	Davies	Ouachita Baptist University	OBU Summer Research Program	P10 Poster
42. Mason	DeLaMater	Princeton University	I am working independently in the Smeltzer Lab	P11 Poster
43. Pete	DelNero	University of Arkansas for Medical Sciences (UAMS)		Judging Only
44. Elizabeth	Diaz Fuentes	Pulaski Academy		Guest
45. Alan	Diekman	University of Arkansas for Medical Sciences (UAMS)		Judging Only

First Name	Last Name	Institution	Research Program	Role / Session
46. Bennett	Dishongh	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	T4 Oral, P73 Poster
47. Eileen	Dishongh	Hendrix College		Guest
48. Katherine	Dishongh	Hendrix College		Guest
49. Matt	Dishongh			Guest
50. Andrea	Duina	Hendrix College		Faculty Mentor
51. Lawson	Duke	University of South Carolina	Proteomics - IDeA National Resource for Quantitative Proteomics Internship Program	P12 Poster
52. Ahmed	Elashwah	University of Arkansas for Medical Sciences (UAMS)		
53. Eric	Enemark	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
54. Robert	Eoff	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
55. Praise	Fabiyi	University of Arkansas - Pine Bluff	PRO - Professional Research Opportunity	P13 Poster
56. Elisabeth	Ferreira	University of Arkansas for Medical Sciences (UAMS)		Judging Only
57. Kaylee	Francis	Hendrix College	SURF (Biochemistry and Molecular Biology, UAMS)	T10 Oral, P75 Poster
58. Aniyah	Freeman	University of Arkansas - Pine Bluff	INBRE - IDeA Network of Biomedical Research Excellence	P14 Poster
59. Jun	Gao	University of Arkansas for Medical Sciences (UAMS)		Judging Only
60. Sarita	Garg	University of Arkansas for Medical Sciences (UAMS)		Judging Only
61. Brooke	Garlington	University of Arkansas - Little Rock	SURP - Summer Undergraduate Research Program (UAMS)	P15 Poster
62. Jansen	Garner	Ouachita Baptist University	OBU Summer Research Program	P16 Poster
63. Luke	Garrett	Hendrix College	SURF (Biochemistry and Molecular Biology, UAMS)	P17 Poster
64. Katherine	Gasaway	Central Baptist College		Judging Only
65. Debasish	Ghosh	Arkansas Children's Hospital		Judging Only
66. Kuppan	Gokulan	National Center for Toxicological Research (NCTR)		Judging Only
67. Erica	Gourley	University of Central Arkansas		Guest
68. Yazi	Greer	Ouachita Baptist University	OBU Summer Research Program	P18 Poster
69. Karen	Grimes-Rucker			Guest

First Name	Last Name	Institution	Research Program	Role / Session
70. Sam	Grose	Ouachita Baptist University	OBU Summer Research Program	P19 Poster
71. Julie	Gunderson	Hendrix College		Faculty Mentor
72. Kelly	Hair	n/a		Guest
73. Tim	Hampton	University of Arkansas - Little Rock		Guest
74. Hannah	Harshaw	Ouachita Baptist University	OBU Summer Research Program	P20 Poster
75. Kaley	Haskins	Ouachita Baptist University	OBU Summer Research Program	P21 Poster
76. Selah	Havens	Ouachita Baptist University	OBU Summer Research Program	P22 Poster
77. Graham	Heberlig	University of Arkansas for Medical Sciences (UAMS)		Judging Only
78. Jeff	Hernandez	Harding University		Guest
79. Mia	Hernandez			Guest
80. Murphy	Hernandez	Harding University	INBRE - IDeA Network of Biomedical Research Excellence	P23 Poster
81. Audrey	Hill	University of Arkansas - Fayetteville	INBRE - IDeA Network of Biomedical Research Excellence	P24 Poster
82. Newton	Hilliard	Arkansas Tech University		Judging Only
83. Madison	Holmes	Ouachita Baptist University	Ouachita Baptist University	P25 Poster
84. Betty	Hu	Vanderbilt University	SURF (Biochemistry and Molecular Biology, UAMS)	P26 Poster
85. Clay	Jackson-Litteken	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
86. Shafina	Jahan	University of Arkansas for Medical Sciences (UAMS)		Judging Only
87. Rishi	Jaiswal	University of Arkansas for Medical Sciences (UAMS)		Judging Only
88. Lisa	Jansen	University of Arkansas for Medical Sciences (UAMS)		Judging Only
89. Ayush	Kanekar			Guest
90. Tamer	Kaoud	University of Arkansas for Medical Sciences (UAMS)		Judging Only
91. Oleg	Karaduta	University of Arkansas for Medical Sciences (UAMS)		Judging Only
92. Sangeeta	Khare	National Center for Toxicological Research (NCTR)		Judging Only
93. Hannah	King	Oral Roberts University		Judging Only
94. Jada-Lynn	King	Lyon College	INBRE - IDeA Network of Biomedical Research Excellence	P27 Poster
95. Sloka	Kovi	Wayzata High School	Physiology and Cell Biology	P28 Poster

First Name	Last Name	Institution	Research Program	Role / Session
96. Sandhya	Krishnan	University of Arkansas - Little Rock		Faculty Mentor
97. Baani	Kumar	Central High School		Guest
98. Gabin	Kwon	Seoul National University		Guest
99. Renny	Lan	University of Arkansas for Medical Sciences (UAMS)		Judging Only
100. Ava	Lang	Arkansas State University	SURP - Summer Undergraduate Research Program (UAMS)	P29 Poster
101. Ricky	Leung	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
102. Andrew	Lever	University of Arkansas - Fayetteville	Fulbright Honors College Undergraduate Research	P30 Poster
103. Jackson	Lewis	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
104. Georgia	Liu	Hendrix College	Hendrix Odyssey Program	P31 Poster
105. Yunmeng	Liu	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
106. Kim	Lowe	University of Arkansas - Little Rock		Guest
107. Tamara	Lowe	University of Arkansas - Little Rock	SURF (Pharm/TOX, UAMS)	P32 Poster
108. Vladimir	Lupashin	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
109. Rosemary	Mallory	Episcopal collegiate school		Guest
110. John	Marecki	University of Arkansas for Medical Sciences (UAMS)		Judging Only
111. Avery	Maxwell	Ouachita Baptist University	OBU Summer Research Program	P33 Poster
112. Ravyn	Mays	University of Arkansas - Little Rock	SURP - Summer Undergraduate Research Program (UAMS)	P34 Poster
113. Alex	McDonald	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
114. Maci	McJunkins	Ouachita Baptist University	OBU Summer Research Program	P35 Poster
115. Diane	McKinstry	University of Arkansas for Medical Sciences (UAMS)		Administrator
116. Grover	Miller	University of Arkansas for Medical Sciences (UAMS)		Administrator
117. Isabelle	Miousse	University of Arkansas for Medical Sciences (UAMS)		Judging Only
118. Christian	Mitchell	University of Arkansas for Medical Sciences (UAMS)		Judging Only
119. Jacqueline	Montero	John Brown University		Guest
120. Michael	Moore	University of Arkansas - Little Rock		Faculty Mentor

First Name	Last Name	Institution	Research Program	Role / Session
121. Milo	Moore	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	P36 Poster
122. Lydia	Morales-Castillo	Lyon College	Lyon College Summer Research	P37 Poster
123. Suneth	Mudalige	Little Rock Central		Guest
124. Sakina	Mushtaq			Guest
125. Mohammed	Naif	University of Arkansas - Little Rock	PRO - Professional Research Opportunity	P38 Poster
126. Akshath	Narravula	National Center for Toxicological Research (NCTR)	NCTR Summer Student Research Program	P39 Poster
127. Avnik	Narravula			Guest
128. Swathi	Nedunchezian	University of Arkansas - Pine Bluff		Judging Only
129. Maddie	Nelson	Ouachita Baptist University	OBU Summer Research Program	P40 Poster
130. Anisha	Neupane	University of Arkansas for Medical Sciences (UAMS)		Judging Only
131. Vy	Nguyen	University of Arkansas for Medical Sciences (UAMS)		Judging Only
132. Sanja	Novak	University of Arkansas - Little Rock		Faculty Mentor
133. Joan	Obi	University of Arkansas - Fayetteville	INBRE - IDeA Network of Biomedical Research Excellence	P41 Poster
134. Mishel	Ocho	Wake Forest University	SURF (Pharm/TOX, UAMS)	T6 Oral, P77 Poster
135. Devyn	O'Daniel	Arkansas State University	INBRE - IDeA Network of Biomedical Research Excellence	P42 Poster
136. Jessica	Offordum	Grambling State University	SURF (Biochemistry and Molecular Biology, UAMS)	P43 Poster
137. Feleica	Omoware	University of Arkansas - Little Rock		Guest
138. Jasamine	Omoware	University of Arkansas - Little Rock		Guest
139. Xavion	Omoware	University of Arkansas - Little Rock		Guest
140. Carlos	Paez	John Brown University		Guest
141. Samuel	Paez	John Brown University	INBRE - IDeA Network of Biomedical Research Excellence	P44 Poster
142. Sarah	Paez	John Brown University		Guest
143. Jyotheeswaran	Panapakam	Arkansas Tech University		Judging Only
144. Clara	Park	Rhodes College	SURF (Biochemistry and Molecular Biology, UAMS)	T2 Oral, P79 Poster

First Name	Last Name	Institution	Research Program	Role / Session
145. Pritam Saha	Podder	University of Arkansas for Medical Sciences (UAMS)		Judging Only
146. Zhychaeus	Powell	Philander Smith University	INBRE - IDeA Network of Biomedical Research Excellence	P45 Poster
147. Justin	Pressley	University of Arkansas for Medical Sciences (UAMS)		Guest
148. Dennis	Province	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
149. Christin	Pruett	Ouachita Baptist University		Faculty Mentor
150. Arnav Emil	Raghavan			Guest
151. Iniya	Rajendran			Guest
152. Sonja	Ramirez	Lyon College	Lyon College Summer Research	P46 Poster
153. Javier	Ramirez Santamaria	University of Central Arkansas	4C EFRC	P47 Poster
154. Simra	Rana	Haas Hall Academy Fayetteville	STEAM - Science, Technology, Engineering, Art and Math	P48 Poster
155. Kevin	Raney	University of Arkansas for Medical Sciences (UAMS)		Judging Only
156. Andrea	Richards	University of Arkansas - Fayetteville	Haas Hall Academy	P49 Poster
157. Kahla	Robinson	University of Arkansas for Medical Sciences (UAMS)		Administrator
158. Jordan	Rodgers	University of Central Arkansas	Other	P50 Poster
159. Isabelle	Rose	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	Non-Presenter
160. Samrat	Roy Choudhury	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
161. Edward	Rucker	University of Arkansas for Medical Sciences (UAMS)		Guest
162. Kiah	Rucker	Philander Smith University	PRO - Professional Research Opportunity	P51 Poster
163. Lauren	Russell	University of Arkansas for Medical Sciences (UAMS)		Judging Only
164. Katie	Ryan	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
165. Lídize	Sagarnaga Rivera	Arkansas State University	Arkansas State University	P52 Poster
166. Ian	Scarth	University of Hawai'i at Hilo	Proteomics - IDeA National Resource for Quantitative Proteomics Internship Program	P53 Poster
167. Isabella	Sellers	Samford University	SURF (Pharm/TOX, UAMS)	P54 Poster
168. Ashish	Sharma	University of Arkansas for Medical Sciences (UAMS)		Guest

First Name	Last Name	Institution	Research Program	Role / Session
169. Dishant	Sharma	University of Southern California	INBRE - IDeA Network of Biomedical Research Excellence	P55 Poster
170. Andrew	Shen	National Center for Toxicological Research (NCTR)		Judging Only
171. Himanshi	Shorie	University of Arkansas - Fort Smith	INBRE - IDeA Network of Biomedical Research Excellence	P56 Poster
172. Akul	Shrivastava	Stanford University		P57 Poster
173. Manish	Shrivastava	Stanford University		Guest
174. Sydnye	Shuttleworth	University of Arkansas for Medical Sciences (UAMS)		Judging Only
175. Joshua	Skipworth	University of Central Arkansas	SURF (Pharm/TOX, UAMS)	P58 Poster
176. Janice	Smith	University of Arkansas - Little Rock		Guest
177. Kathryn	Smith	University of Arkansas for Medical Sciences (UAMS)		Judging Only
178. Nadira	Souley Issaka	University of Central Arkansas		Guest
179. Paige	Spicer	Ouachita Baptist University	OBU Summer Research Program	P59 Poster
180. Julie	Stenken	University of Arkansas - Fayetteville		Faculty Mentor
181. Kim	Stephens	University of Arkansas for Medical Sciences (UAMS)		Judging Only
182. Spencer	Stutts	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	T12 Oral
183. Christine	Tan	University of Arkansas - Little Rock	UALR Signature Experience Grant (SEG)	P60 Poster
184. Matthew	Thompson	Lyon College		Faculty Mentor
185. Anthony	Tran	University of Arkansas for Medical Sciences (UAMS)		Guest
186. Godistruth	Umeakuekwe	University of Arkansas - Pine Bluff	PRO - Professional Research Opportunity	P61 Poster
187. Gerson	Valencia Villegas	Arkansas State University		Guest
188. Dan	Voth	University of Arkansas for Medical Sciences (UAMS)		Administrator
189. Ashley	Wallace	University of Arkansas - Monticello	SURF (Biochemistry and Molecular Biology, UAMS)	P62 Poster
190. Rylan	Ward	University of Central Arkansas	INBRE - IDeA Network of Biomedical Research Excellence	P63 Poster
191. Jerry	Ware	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor

First Name	Last Name	Institution	Research Program	Role / Session
192. Prisha	Warikoo	University of Arkansas for Medical Sciences (UAMS)		Guest
193. Ella	Watson	University of Arkansas - Fayetteville	ACH Summer Science Program	P64 Poster
194. Kirk	West	University of Arkansas for Medical Sciences (UAMS)		Faculty Mentor
195. Jacee	Wheat	Hendrix College	INBRE - IDeA Network of Biomedical Research Excellence	P65 Poster
196. Jadyn	Wilhite	Harding University	INBRE - IDeA Network of Biomedical Research Excellence	P66 Poster
197. Thomas	Williams	University of Arkansas for Medical Sciences (UAMS)		Guest
198. Cory	Willis	University of Arkansas for Medical Sciences (UAMS)		Guest
199. Rhys	Wolter	Episcopal Collegiate School	Jackson T. Stephens Summer Research Fellowship	P67 Poster
200. Deana	Wyatt	University of Arkansas - Little Rock		Guest
201. Aarav	Yadav			Guest
202. Priya	Yadav	University of Arkansas - Pine Bluff		Judging Only
203. Yaswanthi	Yanamadala	NCTR		Judging Only
204. Kalyn	You	University of Arkansas - Little Rock	INBRE - IDeA Network of Biomedical Research Excellence	P68 Poster
205. Bushra	Zaman	University of Arkansas for Medical Sciences (UAMS)		Judging Only
206. Youtong	Zhang	Arkansas School for Mathematics, Sciences, and the Arts	ASMSA student intership	P69 Poster
207. Aidan	Zhao	University of Texas at Dallas		P70 Poster

Abstracts

Anderson, Benjamin, University of Arkansas – Little Rock, “Beyond Flat Cell Culture: 3D-Printed Villus-Crypt Scaffolds for Intestinal Modeling” **P01**

Compared to two-dimensional cell cultures, three-dimensional scaffolds are critical for re-creating human tissues, particularly the human intestine, where villus-crypt geometry dictates cell attachment, proliferation, migration, differentiation, and importantly the effects of therapeutic procedures including cancer radiation treatment. To create these biomimetic scaffolds, a model was developed in CAD software to approximate the villi and crypt geometry found in the small intestine. Prototypes were created with masked SLA vat photopolymerization, tested with photopolymer resins including flexible biomedical-grade resin (Formlabs Biomed Flex 50A), chosen for mucosa compatibility, and to mimic tissue softness. Mechanical testing of a flexible prototype revealed an elastic modulus of 8.26 MPa, a hardness of 1.34 MPa, and adhesion force of 60 μN. A 4-times scale compared to native tissue was achievable, while smaller scales including 2x and 1x began to see erroneous resin retention making the villus-crypt geometry unviable. Optical microscopy revealed the samples preserved a 10:1 villi-to-crypt ratio as intended by the model, as well as a surface roughness of 5.49 μm. While full results are pending, preliminary findings have suggested that while the SLA samples can grow cells, the coverage continuity may not be tissue-like. This limitation, and the lack of internally embedded cells necessitates exploration into hydrogel-based fabrication, such as extrusion-based bioprinting with sodium alginate bioinks. These bioinks can be customized for viscosity, cell incorporation, temperature control and cross-linking strategies such as the freeform reversible embedding of suspended hydrogels (FRESH) method. Additionally, fused filament fabrication is being explored as a secondary method via sacrificial-channel molds for working with hydrogels. Altogether these methods form a workflow from geometric feasibility toward cell-embedded biomimetic models for the human intestine.

Bailey, Cayden, University of Central Arkansas, “The epidemiology of chlamydiae symbionts in natural populations of social amoebae” **P02**

The bacterial phylum Chlamydiae is highly diverse, encompassing over a thousand different families. Among these are "environmental Chlamydiae," discovered through metagenomic sequencing, whose specific hosts are only beginning to be elucidated. These environmental Chlamydiae have been found to live intracellularly in an increasingly wide range of hosts, including amoebas, where they can sometimes act as defensive mutualistic symbionts rather than pathogens. Novel Chlamydiae lineages have been identified in the social amoeba Dictyostelium discoideum, a commonly used model organism to study host-symbiont interactions. The prevalence, diversity, and transmission of Chlamydiae in

other species of social amoebas, however, is unknown To elucidate this potential symbiotic relationship and the broader epidemiology of this symbiont in natural populations, our research addresses three primary questions We are investigating how Chlamydiae prevalence varies by geographic location and environmental conditions, whether genetic diversity and prevalence patterns of Chlamydiae are more host-specific or location-dependent, and how temperature affects the symbiosis

Ballany, Lillie, University of Arkansas – Fayetteville, "Suppressor of Cancer Cell Invasion inhibits DNA unwinding by DNA Helicase B" **T3, P72**

Improper genome maintenance is a hallmark of cancer development HELB (DNA Helicase B) unwinds DNA and, plays roles in the replication stress response and DNA damage response that are important for proper maintenance of the genome HELB also interacts with the tumor suppressor SCAI (Suppressor of Cancer Cell Invasion) Like HELB, SCAI is involved in DNA damage repair and the replication stress response Altered levels of HELB and SCAI affect the survival of patients with different cancers, such as glioma and lung adenocarcinoma Although the reason for this effect is unknown, the specific interactions between HELB and SCAI could provide insight into how the DNA repair and replication stress response limit the development of cancer Another interactor of HELB, RPA (Replication Protein A), facilitates HELB recruitment to sites of DNA damage and replication stress Our work aims to determine the extent that SCAI influences HELB activity as well as to investigate potential HELB-SCAI interaction interfaces in hopes of creating a SCAI interaction-deficient HELB variant Our results demonstrate that SCAI is a HELB inhibitor in both the presence and absence of RPA Additionally, SCAI inhibits HELB activity both when SCAI is pre-incubated with the DNA substrate and when SCAI is pre-incubated with the HELB before adding DNA We have interrogated an Alpha Fold prediction of the HELB-SCAI complex and have identified several potential interaction sites as well as generated several constructs through mutagenesis to test their impact on HELB SCAI interaction This study provides novel insight into the interactions between RPA, HELB, and SCAI, as well as an improvement of the overall understanding of HELB biology and DNA repair

Brinkley, Bailee, Arkansas Tech University, "Investigating New Methods to Detect Gammaherpesviruses in vivo" **T9**

Gammaherpesviruses (GHVs) are an oncogenic subfamily of double-stranded DNA viruses that establish lifelong latency within a cell Human GHVs include Epstein-Barr virus (EBV) and Kaposi sarcoma-associated virus (KSHV) Due to the host-restrictive nature of herpesvirus infection, our lab uses the murine gammaherpesvirus 68 (MHV68) model to study GHV pathogenesis and host response in vivo Several 'reporter viruses exist in the

field and are widely used to identify and monitor MHV68 infection in vivo, however each virus has clear limitations We aim to integrate the strengths of prior reporter virus approaches into an innovative solution We generated two recombinant MHV68 viruses encoding the fluorescent marker, ZsGreen alone (M3-ZsGreen) or fused to histone H2b (M3-H2b-ZsGreen) to prevent diffusion of fluorescence, under the control of the MHV68-specific M3 gene promoter Single-step and multi-step growth curves illustrate that MHV68 M3-ZsGreen viruses are analogous to wild-type MHV68, indicating that insertion of ZsGreen under the control of the M3 promoter does not impact viral growth in vitro Immunofluorescence microscopy and flow cytometry revealed that MHV68 M3-ZsGreen fluorescence is 10X brighter than the current standard MHV68 fluorescent virus, MHV68 H2b-YFP We establish that MHV68 M3-ZsGreen viruses disseminate and infect lymphocytes in the draining lymph nodes, and ZsGreen-positive cells are readily detected by flow cytometry Further in vivo studies are necessary to determine if MHV68 M3-ZsGreen and M3-H2b-ZsGreen disseminate to distal sites of infection and establish latency, and if fluorescence is maintained during chronic infection Together, these data demonstrate the development of a fluorescent reporter virus that replicates similarly to WT MHV68, but by expressing the ZsGreen protein, is 10X brighter than current detection methods, and specifically labels infected cells within draining lymph nodes in vivo

Bushra, Fariha Alam, Southeast Missouri State University, “Sense Oligonucleotide Pharmacology: A Potential Therapeutic Strategy to Correct Oncogenic Splicing” **T11, P74**

Cancer is still incurable, highlighting the unmet medical need to develop more effective therapies Advances in RNA-sequencing unexpectedly identified RNA splicing defects as a major driver of cancer Mutations in splicing factors (most frequently in SRSF2, SF3B1, and U2AF1) are the root of defective splicing The Rahman lab previously deciphered that mutations in SRSF2 change its RNA-binding affinity and promote genome-wide splicing alterations This dysregulation compromises the function of several critical genes (such as EZH2 and INTS3) linked to hematopoiesis, leading to blood cancer Despite identifying aberrant splicing targets and mechanisms, therapeutic development remains ineffective Several small-molecule drugs failed in clinical trials due to off-target toxicity Antisense oligonucleotide pharmacology is an emerging strategy, but it is limited to a single target To address these key gaps, here a novel strategy is introduced called sense oligonucleotide (SO) pharmacology The hypothesis is that SOs can be exploited to correct splicing errors and rescue tumorigenesis SOs are short stretches of single-stranded nucleic acid sequences The idea is that SOs comprising high-affinity binding motifs of a mutant splicing factor will sequester the mutant protein, preventing its binding to downstream targets This will circumvent genome-wide splicing alterations and rescue defective hematopoiesis As a proof of concept, we develop SOs targeting SRSF2-mutant

cancers Preliminary analyses in a model cancer cell line using RT-PCR and western blotting reveal that SOs can efficiently correct splicing errors Cell biology assays demonstrate that SOs can rescue defective hematopoiesis and tumorigenic hallmarks Future research will test this in preclinical patient-derived xenograft and animal models and hopefully progress to clinical development SO pharmacology shows a novel and promising therapeutic avenue for treating cancers associated with splicing dysregulation

Bussell, Owen, University of Central Arkansas, "Recycling carbon dioxide into sustainable fuels with Mn-NHC electrocatalyst", **T7**

The need for sources of sustainable energy has become increasingly apparent as increasing levels of atmospheric carbon dioxide (CO2) threaten Earth s climate and biosphere Through electrocatalytic reduction, atmospheric CO2 can be recycled to create a renewable source of hydrocarbon fuels N-heterocyclic carbene (NHC) ligands play a critical role in the reduction of CO2 due to their strong σ-donor activation of transition metal complexes Additionally, alterations to the structure of the NHC ligands can decrease the free energy of metal-substrate intermediates, increasing the product selectivity of CO2 reduction The NHC ligand, 3-methyl-1-picolylbenzimidazol-2-ylidene (Mebim-pic), was proposed by our lab as an effective mediator for CO2 reduction in transition metal complexes Using Schlenk line techniques, Mebim-pic was synthesized and chelated to Mn(CO)5Br complex to form Mn(MeBim-pic)(CO)3Br, followed by structural confirmation via IR spectroscopy and NMR spectroscopy To determine the electrocatalytic properties of the complex, samples of the catalyst will undergo cyclic voltammetry and bulk electrolysis The complex will then be used for the electrocatalytic reduction of CO2, and samples of the reduction product will undergo gas phase mass spectroscopy and IR spectroscopy for structural determination The data collected from MeBim-pic can then be compared to ligands with slight structural differences, and conclusions can be drawn about how MeBim-pic s structural components affect the efficiency and selectivity of the catalyst

Butler, Lily, Episcopal Collegiate School, "Pharmacologically activating ATF6 to enhance Immunotherapy response in Melanoma" **P03**

Melanoma is one of the most aggressive forms of cancer with a 5-20% 5-year survivability rate when metastasized However, due to immune checkpoint blockade (ICB) therapies, the survival rate has increased to 50% ICB is the use of immune checkpoint inhibitors (ICI) to increase antitumor immunity by promoting T-Cell activation; however, half of patients have an intrinsic resistance to ICI treatments Previous studies have shown that an increase in the activating transcription factor 6 (ATF6) – which is part of the unfolded protein response (UPR) - improved ICB responsiveness The mechanism by which ATF6

improves melanoma response to ICB is yet unknown To understand how ATF6 enhances response to immunotherapies, we investigated the effects of pharmacologically activating ATF6 in a murine melanoma cell line (YUMM1 7) using proteomics We used the experimental small molecule drug AA147 to activate ATF6 and interrogated the resulting enriched pathways We show the expected activation of the UPR, ATF6, and ER stress pathways, which drive genomic reprograming and promote production of GRP78 (BiP), Calreticulin (CALR), and GRP94 BiP is the primary molecular chaperone within the ER and ensures proteins fold correctly, identifies misfolded proteins for destruction, and reduces ER stress CALR sends out signals to instigate an immune response and the tumor cells to be destroyed However, unexpectedly we see NRF2 and Pentose Phosphate pathways (PPP), in addition to the upregulation of NFE2L2 pentose pathway phosphate genes as a possible result of the activation of ATF6 This suggests that melanoma cells exploit the natural production of NADPH in these pathways to increase cellular stability and avoid immune detection This hints at ATF6 as potentially related to how cells handle oxidative stress and suggests a role in improving immunotherapy reactivity and increased patient survival Our findings offer a step towards understanding and enhancing the melanoma patient response

Caceres, Danny, Hendrix College, “The impact of diet and lifestyle on cognitive function in children” **P04**

Preadolescence is a critical period for a child s brain development, especially in the organization of neural network function Current literature indicates that overweight individuals show a decline in cognitive functioning compared to normal-weight individuals, in tasks involving executive functioning, such as attention and cognitive inhibition Despite this disparity, there is a lack of data investigating the nutritive-neurocognitive relationship in preadolescents of different body mass indexes (BMI) Some key factors associated with BMI include meal timing and food compositions Weight has been shown to increase with high-protein diets as well as low-fat diets Particularly, the macronutrient characteristics of breakfast meals are noted to affect neurocognitive function These benefits stem from the interactions between carbohydrate-related factors, metabolism rates, and nutrients It follows that an early intervention strategy targeting nutritive factors that influence brain development is needed This project bridges a gap in the literature by examining the relationship between executive functioning following a high-carb or high-protein breakfast meal in overweight and normal weight preadolescents We hypothesized that a high protein/low carbohydrate meal would be associated with better executive functioning compared to a low protein/high carbohydrate meal To test this, high density electroencephalography (EEG) was collected following a cognitive inhibition task conducted on overweight and normal weight children Data was analyzed using event

related potentials (ERPs) to examine the brain's response to task performance. [Results and conclusion statements to be added here in final rendition]

Caffey, Rebekah, Hendrix College, "EPA and DHA supplementation with HFD during pregnancy leads to changes in circulating and liver lipid metabolism." **P78**

Objective: Maternal obesity during pregnancy has altered systemic metabolism, pregnancy complications, and adverse fetal outcomes. Polyunsaturated fatty acids (PUFA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) improve lipid and liver function in non-pregnant state, but their effects on maternal liver metabolism and fetal growth during pregnancy remain unknown. Methods: Pregnant mice (n = 10-13) were fed control (CON) or HFDs with and without EPA or DHA supplementation prior to breeding and throughout pregnancy. Body composition was collected via EchoMRI. Maternal body and liver weights, serum markers, and fetal and placental weights were measured at late pregnancy. Liver gene expression was evaluated by RT qPCR. Results: HFD in the absence of EPA or DHA significantly increased maternal BW during pregnancy and changes in body composition prior to breeding compared to CON diets. Liver to BW ratio were increased in the HFD compared to CON ($p<0.05$), while EPA and DHA supplementation within HFD groups significantly reduced liver to BW ratios. HFD increased serum total cholesterol and HDL relative to control ($p<0.05$). EPA and DHA supplementation within HFD groups showed a protective effect for both total cholesterol and HDL with significantly reduced levels compared to HFD and similar to CON groups. EPA and DHA partially normalized the expression of several metabolic genes compared to HFD alone. There was no effect of HFD with or without DHA or EPA on placental or fetal weights. Conclusion: These findings suggest that while EPA and DHA supplementation do not prevent HFD-induced weight gain during pregnancy or fetal growth, they did beneficially modulate maternal liver metabolic gene expression and circulating lipid profiles in mice fed an HFD. This work supports further investigation into targeted PUFA supplementation as a potential dietary intervention to reduce metabolic complications, specifically in the liver of pregnant women with obesity.

Canizares, Mia, Mount St. Mary Academy, "A novel target of chemotherapy-induced cardiotoxicity in valvular interstitial cells" **T5**

Chemotherapeutics such as doxorubicin (DOX), despite known cardiotoxic effects, remain the first line of treatment for many cancers. This cardiotoxicity eventually causes fibrosis and heart failure. While this has been studied in detail in cardiomyocytes in the heart, we are yet to understand the underlying mechanisms in non-myocyte cells, especially the non-cardiac muscle associated cells. The aortic valves are the structural gatekeepers that control flow of blood in and out of the heart. While DOX exposure is

known to alter inflammation and fibrosis in this over time, there is no information available about their cellular pathophysiology Histone deacetylase 11 (HDAC11) has recently emerged as a regulator of cardiac fibroblast activation and inflammatory signaling, implicating it in the development of DOX-induced cardiac remodeling Valvular interstitial cells (VICs) are remarkably similar in form and function to fibroblasts Here, we hypothesize that HDAC11 is a key driver of DOX-induced maladaptive remodeling in VICs, and its inhibition would provide a protective effect against the cardiotoxicity elicited by DOX HDAC11 is strongly induced in human VICs in response to acute non-cytotoxic DOX treatment Reactive oxygen species assay demonstrated that DOX increased production of hydrogen peroxide This is mitigated with co-treatment with an HDAC11-selective inhibitor However, this protective effect was not seen when HDAC11 enzyme activity was inhibited prior to or post-DOX exposure in VICs, thus timing of the treatment is critical for cellular protection Intriguingly, VICs exposed to DOX for just 24 hours showed induction of pro-fibrotic and inflammatory genes, which was strongly suppressed by HDAC11 inhibition This indicates a potential early trigger for maladaptive remodeling in the disease process than is known Thus, our observations collectively present HDAC11 as a potential pharmacological target for mitigating cardiotoxic effects in cardiac valves

Canon, Noah, Ouachita Baptist University, "[MnIII(TPP-HEP)Cl]: A Novel Water-Soluble MRI Contrast Agent for Brain Imaging" **P05**

Gadolinium (Gd)-based contrast agents (GBCAs) have been widely used in clinical magnetic resonance imaging (MRI) for cancer detection, diagnosis, and staging However, increasing evidence has shown that GBCAs can accumulate in the body, raising concerns about adverse effects such as nephrogenic systemic fibrosis (NSF) and gadolinium deposition in the brain and bone, even among patients with normal renal function These safety concerns have prompted regulatory warnings and intensified efforts to develop safer, gadolinium-free MRI contrast agents In this study, we report the development of a novel manganese-based MRI contrast agent, [MnIII(TPP-HEP)Cl], a water-soluble manganese porphyrin designed to enhance biocompatibility while maintaining effective imaging performance Water solubility was achieved through incorporation of the alcohol amine 1-(2-hydroxyethyl)piperazine (HEP), which is structurally related to a moiety found in WAY-100635, a highly selective antagonist of the serotonin 5-HT1A receptor The complex was synthesized via the reaction of [MnIII(TPPC)Cl] with HEP, followed by purification and comprehensive characterization using NMR, IR, and UV–visible spectroscopy High-performance liquid chromatography (HPLC) confirmed the purity of the final product Preliminary in vitro T1 relaxation studies demonstrated that [MnIII(TPP-HEP)Cl] possesses promising contrast-enhancing properties, highlighting its potential as an effective MRI contrast agent for cancers such as glioblastoma As manganese-based systems are

generally associated with more efficient biological clearance than gadolinium-based agents, they may offer a safer alternative for clinical imaging applications Collectively, these findings contribute to the growing development of next-generation MRI contrast agents with improved safety profiles, enhanced biocompatibility, and greater potential for clinical translation

Cliff, Chelsea, Howard University, "In Vitro Characterization of Recombinant VSV Expressing MMR Glycoproteins for Pancreatic Cancer Therapy" **T8, P71**

Pancreatic ductal adenocarcinoma (PDAC) is the third leading cause of cancer deaths in the US Approximately 29-34% of patients present with unresectable, locally advanced disease and have limited treatment options These tumors are poorly immunogenic and do not respond adequately to checkpoint blockade immunotherapy, highlighting the critical need for innovative, less toxic therapeutic approaches Vesicular Stomatitis Virus (VSV) demonstrated promise as an oncolytic agent, selectively killing cancer cells by exploiting their defective interferon signaling However, maximizing therapeutic efficacy requires leveraging multiple anti-tumor mechanisms simultaneously We hypothesized that engineering VSV to express transgenes encoding Measles, Mumps, and Rubella (MMR) glycoproteins will create dual-mechanism viruses that combine direct oncolytic killing with vaccine-driven immune activation We developed three recombinant VSV variants (VMG-MMR) designed to exploit dual anti-tumor mechanisms We infected six pancreatic cancer cell lines, three human (Miapaca, PANC1, HPAFII) and three mouse (603PDA, KPCLUC, PAN02LUC), and evaluated viral efficacy through: viral kinetics analysis via growth curves, crystal violet assay to quantify cancer cell death, western blotting to confirm transgene protein expression, and viral titer measurements to quantify infectious particle production and demonstrated successful viral replication and comparative efficacy across recombinant variants Results demonstrated that the recombinant VSV replicated in pancreatic cancer cells and induced robust cancer cell death across all cell lines This dual efficacy, efficient viral replication coupled with cytopathic effects, confirms that genetically engineered VSVs maintain comparable oncolytic activity to the parental VMG This in vitro characterization provides foundational data to advance in vivo studies of recombinant dual-mechanism VSV variants and clinical translation for PDAC patients

Coleman, Elena, Rhodes College, "GPVI Deficiency Limits Total Body Irradiation-Induced Platelet–Immune Cell Crosstalk" **T1, P76**

Cellular activation and apoptosis promote the generation and release of submicron membrane-derived bioactive vesicles known as microparticles (MPs) Platelets are the predominant source of circulating MPs, and studies have shown that deficiency of platelet glycoprotein VI (GPVI) suppresses platelet-derived MP generation and attenuates

inflammation. Because inflammation enhances platelet–leukocyte interactions, we hypothesize that platelet GPVI deficiency limits radiation-induced platelet–immune cell interactions and subsequent inflammatory responses. We exposed wild-type male C57BL/6J and GPVI-deficient mice to 2 Gy total-body irradiation (TBI) and harvested spleen 8 days after exposure. Single cell suspension was prepared, and cells were subjected to multicolor flow cytometry for platelet-immune cell interactions. TBI significantly increased platelet interactions with lymphoid lineage cells, including NKT, NK, CD4+ T, CD8+ T, B, and regulatory T (Treg) cells in wild-type C57BL/6J, not in GPVI-deficient mice. Similarly, TBI significantly increased platelet interactions with myeloid lineage cells, including macrophages, neutrophils, and conventional dendritic cell type-1 in C57BL/6J, not in GPVI-deficient mice. Interestingly, TBI significantly increased platelet interactions with myeloid lineage cells, including monocytes, conventional dendritic cell type 2, and plasmacytoid dendritic cells, in both C57BL/6J and GPVI-deficient mice. These findings indicate that GPVI deficiency significantly attenuates TBI-induced platelet interactions with the majority of immune cells across lymphoid and myeloid lineages, which may contribute to reduced radiation-induced inflammation. Future studies are warranted to elucidate the signaling pathways governing GPVI-dependent platelet–immune cell interactions and to evaluate the therapeutic potential of GPVI inhibition as a countermeasure against radiation-induced tissue injury and immune dysregulation.

Concepcion, Angelica, University of Arkansas – Fayetteville, “How Plants Fight Back: The Role of Reactive Oxygen Species in Defense Against Aphids” **P06**

Aphids are a large family of insects that attack many crops, vector numerous plant viruses, and reduce plant growth and agricultural yields. One of the most effective ways to combat aphids is to select crop varieties with enhanced host-plant defenses. However, the development of aphid-resistant cultivars is limited by a lack of understanding of how plants defend themselves against aphids. To address this gap, this project investigates a signaling response that may contribute to induced defenses against aphids: the generation of reactive oxygen species (ROS). ROS are oxidant molecules produced in response to stress and can act as early danger signals that activate other defense responses. Although ROS are well known to function in plant immune-like defenses against pathogens, their role in responses to aphids and other insects remains poorly understood. This project uses Arabidopsis thaliana expressing a redox-sensitive fluorescent protein (roGFP2) to detect ROS accumulation in chloroplasts, the organelle responsible for photosynthesis. Using confocal microscopy and fluorescence-based assays, I measured ROS accumulation in response to two aphid species: the green peach aphid (Myzus persicae), a generalist that attacks hundreds of plant species, and the cabbage aphid (Brevicoryne brassicae), a specialist of a single plant family. Our results suggest that both species induce strong and

persistent ROS accumulation in chloroplasts, indicating that this response may be broadly relevant across aphid species The next phase of this project will compare aphid-induced ROS accumulation in light and dark conditions to determine whether light-dependent processes, such as Photosystem II activity, are required Identifying the mechanisms underlying ROS production during aphid attack will advance understanding of plant defense responses and support the development of sustainable strategies to protect crops from multiple aphid pests

Crain, Georgie, Ouachita Baptist University, "Nutrition Assessment of Older Adults" **P07**

The objective of the research study is to assess malnutrition and determine its severity in older adults using BMI and body fat percentages, 24-hour diet recalls, hand grip strength test, and NFPE Participants of this study (n=25) were attendants of the Arkadelphia Senior Center Participants were examined using 24-hour diet recall BMI and body fat percentages were assessed using a Bioelectrical Impedance Machine Hand grip strength was assessed using a hand dynamometer Anthropometric data was also obtained Data was assessed through Excel®, U S Department of Agriculture (USDA) FoodData Central®, and the Statistical Package for the Social Sciences® Eighty percent (n=20) were female, and twenty percent (n=5) were male, out of 25 participants in total Of the 25, 52% (n=13) were deemed underweight or overweight Out of 5 men, 20% (n=1) were overweight Out of 20 women, 20% (n=4) were underweight and 40% (n=8) were overweight Male and female hand grip strengths were standard average for age Males and females had below the Dietary Reference Intake (DRI) levels of kcals, calcium, vitamin C and vitamin D, while protein, sodium, and saturated fats were above average for age Eighty-eight percent (n=22) of fat intake was above the DRI level while the other twelve percent (n=3) was below the DRI level for males and females Of the 25 older participants, 52% (n=13) had a healthy BMI, leaving the other 48% either under average (n=4) or above average (n=8) All five assessments indicated signs of malnutrition The NFPE results indicated mild-moderate fat loss in the orbital and upper arm region, mild-moderate muscle loss in the temple region, Bitot's spots in the eyes, edema, and skin dehydration in some participants Data confirmed most participants to be at risk for malnutrition

Cunningham, Savea, University of the Ozarks, "Investigating MLKL's Membrane Poration in Necroptosis" **P08**

During viral and bacterial infections or cancer development, organisms have several options for programmed cell death including apoptosis, necroptosis, and pyroptosis Necroptosis is a highly inflammatory form of cell death which undergoes crude lysis of the cell and is executed upon activating signaling by kinase RIPK3 and pseudokinase MLKL

MLKL is a pore-forming protein that targets several cellular membranes including the mitochondrial and plasma membranes. The Moldoveanu lab is actively pursuing structures of the MLKL necroptotic pores. My project focuses on producing several constructs of MLKL, including some that are stabilized in circular configurations of different diameter, and interrogating them in biochemical and biophysical assays prior to cryo-electron microscopy structure determination. I expressed MLKL constructs in E. coli and purified proteins using nickel affinity chromatography followed by size exclusion chromatography. The purified proteins are analyzed through protease digestion to test construct integrity, liposome poration, live cell poration, and negative staining transmission electron microscopy. These studies aim to understand the molecular mechanisms of necroptosis poration.

Danielle, Willow, University of Arkansas - Little Rock, "Social network analysis of STEM faculty communities in pedagogical learning experiences" **T09**

Pedagogical learning experiences (PLEs) train faculty in how to use evidence-based practices (EBPs) in undergraduate STEM education, which positively influence student outcomes (Freeman et al., 2014). PLEs not only improve faculty knowledge and implementation of EBPs (Manduca et al., 2017), they create faculty communities that support improvement (Kleinschmit, 2023). Our project offered STEM instructors multiple opportunities to engage in ongoing PLEs at UALR, an urban four-year public R2 university. STEM faculty (1) participate in a week-long workshop on EBPs, (2) can opt into semester-long interdisciplinary teaching communities of practice to improve their curricula (CoP), and (3) can further opt into a community of transformation to impact university-wide teaching policy changes (CoT). To answer our research of "How do faculty interactions about teaching change over time and with participation in the PLEs?", we analyzed longitudinal change using social network analysis (SNA). SNA allows us to quantify and visualize changes in instructor communities globally, individually, and by teaching interaction type. Using the Louvain algorithm (Blondel et al., 2008), we detected the community structure of our project participants and tested the change in network density. We found that the overall density change of participants was significant ($p=0.0123$). While participation in the week-long workshop on EBPs alone was not significant, instructors who also participated in CoPs showed a significant shift toward discussions about implementing EBPs ($p=0.0412$), as did those participating in CoTs ($p=0.0317$). This evidence is consistent with prior work on sustaining faculty engagement in teaching-focused communities (McCourt et al., 2017). These findings about the implementation of PLEs can be translated to other undergraduate or medical education institutions, such as UAMS.

Davies, Paige, Ouachita Baptist University, “Analysis of the Leaching of Bisphenol-A from Feminine Hygiene Products into a Synthetic Vaginal Fluid Solvent using Fluorescence Spectrophotometry” **P10**

Bisphenol-A, also known as BPA, is a synthetic chemical found in many everyday products, including some feminine hygiene products BPA is a concern because its structure is similar to estrogen, which allows it to mimic estrogen in the body and bind to estrogen receptors This could interfere with normal hormone signaling and may be connected to reproductive and hormonal health concerns Since feminine hygiene products are used in close contact with sensitive areas of the body for prolonged periods of time, this research examined whether BPA leached out of these products during use This research focused on detecting BPA leaching from feminine hygiene products using fluorescence spectroscopy and the standard addition method Initial testing was performed on several different brands of pantyliners The prepared samples were placed in a 50% methanol/water solution for 10 different set time points ranging from 0 to 360 minutes After each time point, the liquid was collected and used to prepare standard addition solutions The solutions were analyzed using an FS5 Spectrofluorometer from Edinburgh Instruments (λEx= 278 nm, λEm= 304 nm) to determine the concentration of the BPA present This first step was used to confirm the testing method before moving into a more biologically representative solvent The methodology above was then repeated using artificial vaginal fluid to better represent the environment these products may experience during normal use The data obtained from these two different methods were then compared to see the effect of changing the solvent Future plans for this research project include testing of other period products, such as period underwear and menstrual cups The goal of this project is to provide a better understanding of possible BPA exposure from commonly used feminine hygiene products and support future studies on product safety, chemical leaching, and consumer awareness

DeLaMater, Mason, Princeton University, “Differences in the Staphylococcus aureus sarA/sigB/agr regulatory axis define alternative pathways in the pathogenesis of osteomyelitis” **P11**

Anti-virulence strategies are increasingly explored as therapeutic alternatives in the current era of increasing antibiotic resistance In Staphylococcus aureus, the two most extensively investigated regulatory loci are the accessory gene regulator (agr) and the staphylococcal accessory regulator (sarA) Our previous work on osteomyelitis led us to conclude that sarA is a better therapeutic target than agr, because mutation of sarA limits virulence to a greater extent than mutation of agr, and does so irrespective of the functional status of agr We also proposed that the regulatory balance between agr and

sarA differs among clinical isolates to a degree that defines two alternative pathways, with sigB playing an important role We examined the impact of sigB on agr- and sarA-associated phenotypes in methicillin-resistant CC8/USA300 LAC and methicillin-sensitive CC30/USA200 UAMS-1 AgrA, RNAIII, and SarA were higher in LAC, and mutation of sigB moderated these differences Protease activity increased comparably in both sigB mutants, sufficient to limit biofilm formation Cytotoxicity was greater in LAC and was decreased in sarA mutants, but increased in sigB mutants, correlating with cytotoxin production and protease-mediated degradation Most importantly, mutation of sigB limited bacterial burdens in the femur of mice infected with UAMS-1 but not LAC, while mutation of sarU had no significant impact on LAC phenotypes These results support the hypothesis that S aureus uses alternative but overlapping regulatory pathways in the pathogenesis of osteomyelitis, with one (UAMS-1) defined by limited agr expression to favor biofilm formation and chronic infection, and the other (LAC) defined by increased expression of agr to favor cytotoxicity and acute infection They confirm that mutating sigB potentially precludes it as a therapeutic alternative, and demonstrate that sarA can be exploited to therapeutic advantage despite such strain-dependent differences

Dishongh, Bennett, Hendrix College, "CST4 as a Novel Regulator of Triple Negative Breast Cancer" **T4, P73**

Breast cancer is the most common cancer in women Triple negative breast cancer (TNBC) accounts for 15-20% of breast cancer cases and has the lowest five-year survival rate with 46% of TNBC patients developing metastases Metastasis is a multistep process, and cell invasion is one of the earliest steps of the process There are currently no targeted treatments for TNBC and therefore there is a need to identify novel therapeutic targets or prognostic markers To do this, highly invasive "i8" MB231 cells were generated by eight rounds of invasive selection and were confirmed to have a more invasive phenotype with a transwell invasion assay Through mass spectrometry, cystatin 4 (CST4) was identified as significantly downregulated in i8 cells compared to parental cells CST1, CST2, and CST3 were also slightly downregulated Cystatins are cysteine protease inhibitors, and CST3 is known to affect invasion by inhibiting cathepsins and therefore preventing the degradation of the extracellular matrix, however there is no reported role for CST4 in TNBC Here we investigate whether the downregulation of CST 1-4 is responsible for the increased invasive phenotype of the i8 MB231 cells and if cathepsins are involved I used qPCR to examine the expression of CST proteins and their effector cathepsins at the mRNA level and found that CST 1-4 are downregulated in i8 cells, while cathepsins B and L remain unaffected This matches mass spectrometry findings from preliminary studies To determine whether downregulation of CST4 contributes to the overly invasive phenotype of i8 MB231 cells, I performed a knockdown of CST4 followed by a transwell assay and observed a more

invasive phenotype CST4 is the primary target, but I also performed siRNA knockdown on CST1, CST2, and CST3 to examine their role in invasion I validated all knockdowns with western blotting My results will define a role for cystatin proteins as potential therapeutic targets or novel biomarkers of TNBC metastasis

Duke, Lawson, University of South Carolina, "Enhancing Low-Input Protein Recovery in the Chloroform Methanol Extraction Workflow Using SP3 Paramagnetic Beads in Bottom-Up Proteomics" **P12**

In proteomics, a critical determinant of mass spectrometry performance is the quality of sample preparation, as contaminants introduced during cell lysis and protein solubilization can significantly compromise downstream detection The Chloroform Methanol Extraction (CME) protocol is a widely used sample preparation method that isolates and digests proteins through sequential reduction, alkylation, organic solvent-based precipitation, and tryptic digestion before mass spectrometry analysis However, a key limitation of standard CME is the difficulty in visualizing and recovering protein pellets at low input quantities, which reduces workflow reliability and downstream identification rates This study evaluates whether the incorporation of SP3 paramagnetic beads into the CME workflow can improve protein pellet visualization and recovery, particularly at lower protein input amounts SP3 beads are carboxylate-coated hydrophilic beads that capture proteins through an ethanol-driven solvation mechanism, allowing contaminants to be rinsed away while proteins remain bound to the bead surface for direct on-bead tryptic digestion HeLa cells, a well-characterized immortalized human cell line, were used as the experimental model Samples were processed at both 50µg and 10µg protein inputs, comparing standard CME to CME with SP3 beads introduced before the extraction step Protein identification and yield were assessed by liquid chromatography-mass spectrometry (LC-MS) analysis and compared across conditions

Fabiyi, Praise, University of Arkansas - Pine Bluff, "Rnd3 regulates metastatic hallmarks in lung adenocarcinoma through integrin Alpha 5 modulation" **P13**

Lung cancer is the leading cause of cancer-related deaths worldwide due to its high metastatic rate Expression of Rnd3, a small Rho-GTPase, has been linked to tumor progression and lung adenocarcinoma patients with lower Rnd3 expressions exhibit significantly improved survival outcomes Our preliminary data revealed that knockdown of Rnd3 in lung adenocarcinoma cells resulted in upregulation of alpha 5 integrin and reduced cellular migration and invasion, two key hallmarks of metastasis Integrin alpha 5 is a transmembrane receptor thatfacilitates cell adhesion and migration Knockdown of alpha 5 in Rnd3-depleted cells rescued cell migration and invasion to wild-type levels, suggesting a connection between Rnd3, alpha 5, and the metastatic cascade Here, we

investigate how knocking down Rnd3 contributes to alpha 5 upregulation by using qPCRs to determine whether Rnd3 affects alpha 5 transcriptionally, or if Rnd3 affects alpha 5 stability via cycloheximide-chase assay We also investigate the phenotypical outcome of the Rnd3-alpha 5 regulation in lung cancer by performing adhesion assays to determine whether adhesion is altered in Rnd3-depleted cells compared to control qPCR results present no significant increase in the mRNA levels of alpha 5, as well as its heterodimeric partner, beta 1, in Rnd3-depleted cells However, following cycloheximide treatment, alpha 5 protein in Rnd3-knockdown cells is more stable compared to control These data indicate that when Rnd3 is depleted in lung cancer cells, alpha 5 becomes more stable, leading to increased alpha 5 protein levels Additionally, adhesion assays revealed that Rnd3-knockdown cells adhere more strongly to the extracellular matrix compared to control We hypothesize that this increase in adhesion decreases cell migration and invasion Further investigation into the specific pathway by which Rnd3 contributes to alpha 5 stability will be important in developing novel therapies and possibly preventing metastasis

Francis, Kaylee, Hendrix College, "REV1 facilitates DHX36 localization in response to G-quadruplex stabilization during nascent strand synthesis" **T10, P75**

G-quadruplexes (G4s) are four-stranded nucleic acid structures formed through non-canonical base pairing within guanine-rich strands G4 DNA presents a barrier that the replisome cannot overcome without assistance from specialized proteins REV1, a translesion synthesis (TLS) polymerase with an unconventional mechanism of action, helps to mediate G4 replication through a multifaceted mechanism that involves G4-selective binding properties and protein-protein interactions Our lab discovered that REV1 helps recruit the DHX36 helicase to chromatin and facilitates its accumulation on DNA following treatment with the G4 stabilizer pyridostatin (PDS) The interaction between REV1 and DHX36 was crucial for preventing ssDNA gap formation, but we do not know if a similar response is required for other G4 stabilizers or other DNA damaging agents The broad aim of our project is to investigate the role of the REV1-DHX36 axis as a general stress response mechanism to different barriers that impede DNA replication We used isolation of proteins on nascent DNA (iPOND) coupled with immunoblotting to determine how much DHX36 accumulated near nascent DNA in both wild-type and $REV1^{KO}$ 293 FT cells We first tested the effect of PDS at 50 μM, which was a 5x higher concentration than our previous iPOND experiments DHX36 signal increased in a time-dependent manner upon exposure to 50 μM PDS in the wild-type cells, but this trend was not seen when REV1 was absent The results at the higher dose of PDS were almost identical to what we observed in cells treated with 10 μM PDS To test the generality of REV1-dependent DHX36 recruitment to sites of G4 replication stress, we will repeat these studies with N-methylmesoporphyrin IX

(NMM), another G4 stabilizer with a strong preference for parallel-folding quadruplexes. With these studies, we will obtain a better understanding of how broadly the REV1-DHX36 axis participates in the replication stress response.

Freeman, Aniyah, University of Arkansas - Pine Bluff, "Antioxidant, Anti-inflammatory, and Anti-diabetic Properties of Rice Bran from Arkansas-Grown Rice Cultivars" **P14**

Rice bran, a byproduct of the rice milling process, contains several bioactive compounds in its matrix, including gamma-oryzanol, tocotrienols, tocopherols, and phytosterols. These bioactives are natural antioxidants that help reduce oxidative stress and systemic inflammation, which are key causative factors of chronic diseases. This study aims to investigate the potential of the ethanolic extract of rice bran cultivars (Dellmati, Lynx, and 1099) for their antioxidant, anti-inflammatory, and anti-diabetic properties. The crude rice bran extracts of these varieties were prepared by conventional maceration and subsequently lyophilized. All three varieties exhibited a dose-dependent increase in phenolic content. Also, the DPPH radical-scavenging assay showed a dose-dependent total antioxidant capacity, with a maximum at a higher concentration (2 mg/mL), at which Dellmati, Lynx, and 1099 varieties demonstrated 85%, 93%, and 92% inhibition, respectively. Similarly, the anti-inflammatory action was strongly dose-dependent, with the highest concentrations producing the most pronounced effects. Ongoing research is evaluating these extracts for their potential to modulate α-amylase and α-glucosidase activities to determine their anti-diabetic potential. The findings of our study reveal that rice bran extract has the potential to prevent oxidative stress, downregulate pro-inflammatory mediators, and reduce the activity of carbohydrate-metabolizing enzymes.

Garlington, Brooke, University of Arkansas - Little Rock, "Discovery of Novel Antiviral Drug Candidates for Hantavirus Infection" **P15**

Hantaviruses are enveloped, negative-sense, single-stranded RNA viruses maintained primarily in rodent reservoirs and transmitted to humans through contact with infected urine, feces, and saliva. These viruses cause significant human disease worldwide, including hemorrhagic fever with renal syndrome and hantavirus cardiopulmonary syndrome, with approximately 200,000 cases occurring annually. Currently, no specific approved antiviral therapies are available for the treatment of hantavirus infections. Herein, we identified novel triptan-derived antiviral compounds that inhibit hantavirus infection, addressing the critical need for antiviral therapeutics. Triptan-derived compounds 44 and 45 were evaluated using human lung organoids and an Andes orthohantavirus (ANDV) glycoprotein pseudovirus assay with a luciferase reporter. Live-

virus plaque reduction assays were then performed to evaluate antiviral activity Additionally, pharmacokinetic (PK) and toxicology studies using both intravenous and oral dosing were conducted in hamsters Compounds 44 and 45 both resulted in undetectable pseudovirus entry at approximately 1 5 μM The live-virus plaque reduction assays demonstrated antiviral activity for both compounds at nanomolar concentrations (ANDV IC_{50} values of 10 nM and 2 nM for compounds 44 and 45, respectively) PK studies indicated that compound 45 was detected in hamster plasma 1 h after oral administration Acute toxicity studies were conducted at doses up to 100 mg/kg with no observable adverse effects Chronic toxicity evaluation, including CBC and clinical chemistry analyses, showed tolerability during repeated dosing for compound 45 at 12 5 mg/kg Pharmacodynamic studies evaluating the antiviral activity of compounds 44 and 45 in infected hamsters are currently ongoing Overall, these findings suggest that compounds 44 and 45 inhibit viral entry and reduce live-virus infection, making them promising candidates for antiviral therapies against hantavirus

Garner, Jansen, Ouachita Baptist University, "Preliminary Characterization of Pineapple and White Sugar Kombucha Microbiomes" **P16**

Kombucha is a fermented tea that is produced using a symbiotic culture of bacteria and yeast At Ouachita Baptist University, all STEM majors participate in a Course-Based Undergraduate Research Experience (CURE) centered around kombucha fermentation, choosing different ingredients for their experimental kombucha and comparing that to a control batch fed white sugar This study used metagenomic sequencing to identify and compare the bacteria and yeast genera present in experimental pineapple kombucha compared to traditional white sugar kombucha Samples of kombucha soup were analyzed, with the 16S (V4-V5 region) and 18S (V9 region) rRNA genes amplified and sequenced on an Illumina NovaSeq 6000 platform to generate paired-end reads The sequence data were then processed and analyzed in QIIME 2 to classify microbial taxa and calculate percent abundance of specific genera Results show high levels of Komagataeibacter, a bacterium which produces both cellulose and organic acids and is vital in kombucha fermentation By identifying the microbes associated with fermentation, this work provides a better understanding of the core kombucha microbiome and how carbohydrate source impacts kombucha microbe diversity and abundance

Garrett, Luke, Hendrix College, "Examining the impact of Rev1 interaction on DHX36 helicase activity" **P17**

Earlier work in Dr Eoff's lab has determined that the translesion polymerase Rev1 destabilizes G-quadruplexes (G4) Further, they have evidence to suggest that DHX36 interacts with Rev1 and may facilitate resolution of G4 structures during replication The

purpose of this project is to test and determine the mechanism of how DHX36 works with Rev1 to unwind the G4s and to get a better understanding of how the catalytic activities of the two enzymes influence each other. To do this, we are working on optimizing expression and purification of human DHX36 from bacterial cells. Previously, results from the BL21 (E3) cells were mixed, after running it through a Histrap column and a purity of less than 20% was achieved. We now switched to using E. coli (LOBSTR) cells which are expected to reduce the expression of contaminant proteins that bind to Ni-NTA affinity column and increase purity of expressed His-tagged DHX36. Our first attempt at expression with the LOBSTR strain showed a greater yield and improved purity of DHX36 (60%) than our previous attempt with BL21 cells. We then ran a heparin column with a concentrated Histrap DHX36 protein and achieved a purity of 78%. Then we ran the concentrated Heparin DHX36 through a GSTrap column to cleave the affinity tag. Once we have finalized the purification strategy, we will purify a version of DHX36 with key residues in the RIR-motif are mutated (F912A/F913A). These purified DHX36 proteins will be used along with purified Rev1-core and full-length proteins to study kinetic parameters of helicase action, especially focusing on how REV1 impacts the processivity of DHX36.

Greer, Yazi, Ouachita Baptist University, "Analysis of BPA in Athletic Clothing Using Fluorescence Spectroscopy" **P18**

Bis-phenol A (BPA) is a chemical compound used in manufacturing items like plastics, polyester clothing, and feminine hygiene products for its strength and elasticity factors. However, it's been linked to various health risks such as breast cancer, alterations in fetal development, and decreased fertility as it leaches out of the item and is absorbed into the body. BPA acts as an endocrine disruptor, and its similar structure to the hormone estradiol allows it to bind to endocrine receptors. BPA is a fluorescent compound, so fluorescence spectroscopy with the FS-5 spectrofluorometer from Edinburgh Instruments was used. Previous research analyzed the amount of BPA that leached out of athletic clothing made up of various percents of polyester. The first part of the experiment used 50% methanol/water as the solvent to assist the leaching of BPA, and the second part used artificial sweat to simulate a more accurate biological response. The results for both parts were compared to see how the artificial sweat affected BPA leaching over time and the fluorescent intensity measured by the spectrofluorometer. First, a calibration curve was made for each solvent to ensure that increasing concentrations of BPA has a direct relationship with fluorescent intensity. Fluorescence intensities were measured at an excitation wavelength of 278nm and an emission wavelength of 304nm. The linear range, limit of detection, and limit of quantitation were determined for each calibration curve. The standard addition method was used to monitor leaching due to the small concentrations of BPA and complex matrix of the sample. Samples were prepared to determine the unknown

concentration of BPA that leached out for time stamps ranging from 0min to 360min This was done for leggings made up of 95% polyester/5% spandex, 83% polyester/17% spandex, and a negative control of 95% cotton/5% spandex

Grose, Sam, Ouachita Baptist University, "Piloting a Biocube CURE Project" **P19**

Course-based Undergraduate Research Experiences (CUREs) provide students with the opportunity to do real scientific research as a part of their coursework, which promotes critical thinking and collaboration This project focused on developing and piloting a new CURE project for the Biology II course at Ouachita Baptist University Students will select and investigate the biodiversity of a one-square-foot area on Ouachita's campus The lab will emphasize observation and data analysis while the students gather biodiversity data from their square by collecting, identifying, and documenting organisms found within their square foot, including plants, invertebrates, and soil microbes This project compared diversity results across several locations, using several laboratory techniques, including plant pressing, Berlese funnels, Baermann funnels, microscopy, serial dilutions, and spread plating A noteworthy result from this project was the impact of tree cover on the number of nematodes and invertebrates Biocubes with more trees overhead had more nematodes and a more diverse invertebrate population due to the presence of leaf litter, which provides a cool, moist environment for these organisms Remaining results from this pilot will be reported and used to improve Biology II lab procedures and directions for future classes

Harshaw, Hannah, Ouachita Baptist University, "Development of [MnIII(TPP-PEG3)Cl] as a Novel Water-Soluble MRI Contrast Agent" **P20**

Gadolinium-based contrast agents (GBCAs) are widely used in magnetic resonance imaging (MRI) for cancer detection and staging; however, concerns over nephrogenic systemic fibrosis (NSF) and gadolinium retention in tissues have prompted the search for safer alternatives This study presents the development of [MnIII(TPP-PEG3)Cl], a novel gadolinium-free, water-soluble manganese porphyrin MRI contrast agent Manganese is an attractive alternative because it is an essential biological metal with more favorable clearance characteristics than gadolinium [MnIII(TPP-PEG3)Cl] was synthesized by reacting [MnIII(TPPC)Cl] with PEG-3 amine, a hydrophilic polyethylene glycol derivative designed to improve water solubility and biocompatibility The product was purified and characterized using IR, NMR, and UV–Vis spectroscopy, with purity confirmed by HPLC analysis Preliminary in vitro T1 relaxation studies demonstrate promising contrast-enhancing properties, supporting the potential of [MnIII(TPP-PEG3)Cl] as an effective MRI contrast agent These results contribute to the development of safer, gadolinium-free MRI contrast agents for cancer imaging and other diagnostic applications

Haskins, Kaley, Ouachita Baptist University, “Comparison of the Body Mass Indices of Children Participating in a Summer Nutrition Education Program to Children Not Participating in a Summer Nutrition Education Program” **P21**

Approximately 14.7 million U.S. children and adolescents have obesity, which is defined as a child having a body mass index (BMI) at or above the 95th percentile for their age and sex. Childhood obesity increases the risk of obesity continuing into adulthood and serious health consequences. An important factor in the prevention of childhood obesity is nutrition and physical activity education. There will be a statistical difference, showing a decrease from pre-assessment to post-assessment BMI-for-age percentile of the children who received nutrition and physical activity focused education and there will be no statistical difference between pre-assessment and post-assessment BMI-for-age percentile of the children who did not receive nutrition and physical activity focused education. Two summer childcare programs participated as research sites for the study. Nutrition and physical activity lessons were taught to the treatment group for eight weeks. Anthropometric measurements of both groups were obtained at the beginning and the end of the seven weeks.

Havens, Selah, Ouachita Baptist University, “Spheroid Formation of Invasive Lung Cancer Cell Lines and Their Effects on Tumor Microenvironment” **P22**

Lung cancers are composed of a heterogeneous mix of cell types. Invasive tumor cells contribute to lung cancer progression and metastasis not only through their aggressive behavior but also by influencing their microenvironment. To better understand these interactions, we examined three-dimensional spheroid growth and the effects of cell-secreted factors. Transwell-selected invasive H460 cell lines were selected through eight rounds (i8) and characterized alongside the parental H460 cell line using a three-dimensional GILA spheroid assay in a 96-well plate format. This model was used to evaluate phenotypic differences in spheroid formation and growth within a three-dimensional tumor-like environment. Initial experiments compared baseline differences in spheroid formation and growth among parental H460, and both i8 lines under standard culture conditions, with six replicate wells analyzed per cell line. Spheroids were monitored at 24, 48, 72, and 96 hours. Spheroid area was quantified at each time point to compare three-dimensional growth among the three cell lines. Conditioned-media experiments were then used to investigate whether cell-secreted factors influence spheroid growth. Each cell population was exposed to conditioned media taken from an alternate H460 cell population. Spheroid area was measured at 24, 48, and 72 hours to assess changes in growth following conditioned-media exposure. [Results will be presented at poster presentation.] This study aims to characterize phenotypic differences between parental

and Transwell-selected invasive H460 cells and determine whether secreted factors from invasive cell populations influence spheroid growth behavior These findings may provide insight into cell-cell communication and microenvironmental factors associated with aggressive phenotypes in non-small cell lung cancer and may support future investigation of molecular mechanisms and potential biomarkers associated with invasive disease

Hernandez, Murphy, Harding University, "Role of Acinetobacter baumannii Regulators in Pneumonia Pathogenesis" **P23**

Drug resistance in microbes is a growing international epidemic As these organisms continue to develop mechanisms with which to fight current treatments, a deeper understanding of individual virulent species is critical to developing increasingly targeted and potent antibiotics Our research has characterized various genes and translational products to ascertain their impact on the ability of A baumannii to persist upon infection Using both ventilator-associated pneumonia (VAP) and catheter-associated urinary tract infection (CAUTI) models, our lab has uncovered several aspects of A baumannii pathogenesis that could mitigate the effects of infection and continues to research a variety of loci in the A baumannii genome to further this goal This project investigated the role of two A baumannii regulators in the ability of A baumannii to persist in the lungs: MetR and SsuR Preliminary assay data suggests a decrease in virulence mechanisms in each of the mutants along with attenuated bacterial burden with one mutant based on a murine acute pneumonia model These data will provide valuable information regarding which genes are required for A baumannii to exhibit full virulence, potentially providing new antibiotic targets to ease bacterial burden in human patients

Hill, Audrey, University of Arkansas – Fayetteville, "Does Severe Burn Injury Result in Skeletal Muscle Creatine Depletion?" **P24**

Hypermetabolism is a hallmark of the physiological response to severe burn injury and contributes to skeletal muscle wasting, cachexia, and organ dysfunction Creatine, meaning kreas or meat in Greek, plays a critical role in skeletal muscle energy metabolism and function The vast majority (>95%) of the bodies total creatine stores reside in skeletal muscle The impact of burn-induced hypermetabolism and cachexia on skeletal muscle creatine content remains unknown We have recently developed a novel mouse model of severe burn trauma that demonstrates profound hypermetabolic cachexia Building on these findings, this study aims to determine whether severe burn injury results in skeletal muscle creatine depletion Quadricep muscle samples from mice subjected to scald burn injury and subsequent wound debridement and sham controls underwent biochemical analysis to quantify total creatine concentrations using enzymatic spectrophotometric assays Defining changes in skeletal muscle energy metabolites following burn injury may

improve our understanding of the metabolic mechanisms underlying postburn cachexia and identify creatine metabolism as a potential therapeutic target to improve recovery in burn survivors

Holmes, Madison, Ouachita Baptist University, "Optimization of Cross-Linked Pyranine-Loaded Electrospun Nanofiber Mats for Visual Wound pH Detection" **P25**

Chronic and infected wounds experience changes in pH, with bacterial infections becoming more basic than healthy or healing wound environments Developing a wound dressing that provides a visual indication of pH changes could provide a simple method for monitoring infection progression This project focuses on the optimization of electrospun polymer nanofiber mats containing pyranine, a pH-sensitive dye, for wound-sensing applications Polyvinyl alcohol (PVA) and alginate were used as polymer components due to their electrospinnability, biocompatibility, and relevance in wound dressing materials Pyranine, a low-molecular-weight dye, with diverse applications, was incorporated into the mats to provide a visual pH response, with basic conditions (~9 00-~11 00) causing the dye response to become vibrant yellow Initial studies compared two methods of pyranine incorporation: dissolving pyranine in a prepared PVA solution versus solubilizing pyranine and PVA simultaneously Results indicated that incorporating pyranine after the PVA dissolution produced a more consistent solution and was the preferred method Multiple polymer mat formulations have been electrospun and pH testing has begun Results suggest that mat stability remains a major challenge, as the fibers are not maintaining structural integrity across pH environments Several pathways have been explored to improve fiber mat stability To address this challenge, covalent cross-linking strategies are being investigated Chitosan-containing mats are being explored because genipin crosslinks effectively with amine-containing polymers Ongoing studies will expand the pH test range of the pyranine-loaded fibers, evaluate pyranine release/leaching rates with and without cross-linkers, and simulate wound infection by gradually increasing pH The goal is to develop an optimized mat that remains stable across diverse environments while providing a rapid, visible pH response for wound infection monitoring

Hu, Betty, Vanderbilt University, "The Role of SMARCAL1-Mediated Replication Fork Reversal in TatDN1-dependent Replication Fork Protection" **P26**

TatDN1, like other members of the TatD protein family, is an exonuclease that preferentially cleaves single-stranded DNA and RNA, contributing to DNA repair through excision of deaminated nucleotides and, in certain species, mediating chromosomal DNA degradation during apoptosis Genome stability is contingent upon accurate DNA replication However, replication forks frequently encounter lesions, genotoxic agents, and transcription-replication conflicts, collectively termed replication stress To ensure acurate

genome duplication, cells employ protective mechanisms that stabilize and remodel stalled forks, permitting replication to resume while minimizing errors Unresolved replication stress can result in asymmetric fork progression, under-replicated genomic regions, and fork collapse Replication fork reversal constitutes a key protective mechanism, converting the three-way junction of a stalled fork into a four-way, Holliday junction-like structure via annealing of the nascent strands and reannealing of the parental strands This process is mediated by ATP-dependent DNA translocases, including SMARCAL1, ZRANB3, HLTF, and FBH1, and requires the RAD51 recombinase; while certain factors function independently, others act cooperatively to regulate fork remodeling and stability Our preliminary data indicate that TatDN1 is required for fork protection under conditions of replication stress, leading us to hypothesize that fork reversal constitutes a prerequisite for TatDN1-mediated protection To test this hypothesis, we will determine whether inhibition of fork reversal, achieved through depletion of key fork remodelers such as SMARCAL1, alters the fork degradation phenotype observed following TatDN1 depletion These studies will elucidate the mechanism by which TatDN1 preserves fork stability during replication stress by examining its interaction with SMARCAL1 and the reciprocal effects of altered expression of each protein

King, Jada-Lynn, Lyon College, "Direct Activation of BCL-2 Protein, BAK, to Initiate Apoptosis in Cancer Cell Lines" **P27**

Cancer is a global risk to public health, with >2 million newly recorded cases and >600,000 deaths in the US during 2026 alone The Moldoveanu lab discovers and develops small molecules to activate cell death pathways Apoptotic cell death is activated through poration of the outer mitochondrial membrane (MOMP) by the pore-forming BCL-2 family protein, BAK Several dozen aminobenzothiazole small molecules have been synthesized based on a previously published BAK activator discovered in the lab My summer project involves profiling these compounds in established binding assays to better define their mechanism of action Some have already been shown to promote BAK-dependent liposome permeabilization and bind the activation groove of BAK to trigger apoptosis One aim of my research is focused on testing the ability of these compounds to induce a shift in thermal melting of BAK, indicating binding and possible activation or inhibition Another aim is to use Time-Resolved Fluorescence Resonance Energy Transfer (TR-FRET) assays measuring displacement of a fluorescent probe from BAK by these compounds to demonstrate direct interaction and establish binding parameters These assays will enable rank ordering of BAK activators based on direct binding potency, and some compounds are better than the published BAK activator Yet another aim is to help assemble and profile a focused compound library to activate and inhibit apoptotic, necroptotic, ferroptotic, and pyroptotic cell death to allow efficient pharmacologic determination of the forms of cell

death accessible in any given cell line. Initially, we are testing HeLa and HCT116 tumor cells. Preliminary cell death results convey increased apoptosis when HeLa cells are treated with apoptosis activators, and this is inhibited with a caspase inhibitor, as expected. Next, this library will be used to test drug combinations with the top BAK activators in various cell lines and eventually in preclinical tumor mouse models.

Kovi, Sloka, Wayzata High School, "Characterization of the molecular phenotype of a murine model of diabetic cardiomyopathy" **P28**

Diabetic cardiomyopathy (DiabCM), the primary cause of heart failure and mortality among patients with type II diabetes (T2D), is characterized by impaired contractility of and structural changes of the heart and compromised metabolic function. Despite the rising awareness of this condition, the underlying mechanisms remain yet to be fully understood. Due to its complex nature, it is difficult to recapitulate the multiple stages of this debilitating disease in rodent models. Here, we demonstrate the molecular signature of a mouse model of early onset of DiabCM and compare those with human heart failure samples. This allows us to investigate how T2D alters signaling pathways in the heart early, and their ramifications in late stage of the disease. Using histological analysis, we observed prominent changes in cardiomyocyte size and fibrosis in mice that were subjected to DiabCM. Lipid droplet accumulation was also evident in these tissues. These features are consistent with observations in human heart tissues. Metabolic dysregulation was assayed using western blotting, gene expression and ATP assays in both mice and human cardiac tissue samples. An imbalance in the electron transport chain complexes was evident in heart samples from both species. Hyperglycemic stress in mice led to dysregulation of inflammation and mitochondrial metabolism-associated markers, majority of which paralleled those observed in whole transcriptome analysis of human failing hearts. This suggests that the diagnosis of acute onset of DiabCM is a critical need in the clinic to improve patient outcomes. These findings warrant further investigation using a more translational approach such as 3D bioprinting of cardiac organoids generated using patient-derived induced pluripotent stem cells, an effort that is currently underway in the lab. This will allow us to identify molecular targets for future therapeutic strategies that can help slow down or ideally prevent the progression of DiabCM.

Lang, Ava, Arkansas State University, "In Vitro Characterization of Recombinant VSV (VSV-Maraba-G)" **P29**

Pancreatic adenocarcinoma (PDAC) is frequently diagnosed at advanced stages, offering limited treatment options. PDAC is uniquely resistant to treatment due to the dense and fibrous microenvironment of the cancer, hindering both immune responses and therapeutic penetration. Oncolytic virotherapy (OVT) is a promising strategy for treating

widespread cancers, including PDAC, but antibody-mediated neutralization of circulating oncolytic virus particles remains a significant challenge Vesicular Stomatitis Virus (VSV) is a promising virus in clinical stages of OVT research Maraba virus, a close yet serologically distinct member of the Rhabdoviridae family, has a G protein that shares approximately 80% amino acid sequence identity with the VSV G protein The Maraba G protein shows greater resistance to neutralization by both immune and non-immune human serum Our team has engineered a pseudotyped VSV incorporating the Maraba virus glycoprotein to decrease VSV s vulnerability to serum neutralization Here, we present the in-vitro characterization of this recombinant VSV-Maraba G virus Our results show the infectivity of VSV-MARABA-G at multiple time points post-infection (HPI) and multiplicities of infection (MOI) across three human PDAC cell lines (HPAFII, MIA-PaCa-2, and PANC1) and three murine pancreatic cancer cell lines (603PDA, PAN02-LUC, and KPC-LUC)

Lever, Andrew, University of Arkansas – Fayetteville, "Sustainable Polyaddition of Diamine-Bridged Crotonate Monomers" **P30**

Many conventional plastic synthesis rely on carbon-carbon backbones that exhibit high resistance to environmental degradation Incorporation of hydrolytically breakable functional groups such as amides and esters offers a promising strategy for polymer degradability This work explores the synthesis and base catalyzed polymerization of three diamine-bridged monomers (DAC-0, DAC-1, and DAC-2) Each monomer consists of a diamine coupled through an acylation reaction with two crotonyl chloride molecules DAC-0, DAC-1, and DAC-2 incorporate piperazine, N,N-dimethylethylenediamine, and N1,N6-dimethylhexane-1,6-diamine bridging units, respectively Following synthesis, the monomers were purified by recrystallization from acetone at −20 °C The purified monomers were subsequently polymerized through a base-catalyzed step-growth polyaddition Polymerizations were conducted under an inert atmosphere in a nitrogen-filled glovebox using potassium tert-butoxide as the catalyst in tetrahydrofuran Tertbunaol was added to the reaction as an chain-transfer/end-capping strategy to prevent gelation of the polymer Reaction products were characterized using H1 NMR Spectroscopy, MALDI TOF spectrometry, and gel permeation Chromatography Successful synthesis of each monomer was confirmed by 1H NMR analysis 1H NMR analysis confirmed greater than 99% monomer conversion after 1 h for DAC-0 using 5 mol% potassium tert-butoxide and 5 mol% tert-butanol, and for DAC-1 using 0 5 mol% potassium tert-butoxide and 5 mol% tert-butanol Reaction conditions for the DAC-2 reaction are currently being optimized to achieve comparable monomer conversions These findings demonstrate the potential of utilizing diamine-bridged crotonate monomers sustainable polyaddition building blocks Continuing optimization of reaction conditions may provide versatile information geared towards environmentally responsible polymers with tunable properties

Liu, Georgia, Hendrix College, “Role of the CYBA gene in diabetes-induced bone disease” **P31**

Diabetes (DM) is a high prevalent disease that affects several tissues DM-induced bone disease increases the bone fracture risk leading to morbidity and mortality However, underlying mechanisms are uncertain and understudied Transcriptome analysis of diabetic bones by the Bellido lab demonstrated higher expression of the CYBA gene, encoding the p22 protein essential for production of reactive oxygen species (ROS) Thus, we formulated the hypothesis that CYBA/p22 is responsible for inducing DM-induced bone disease and that deletion of the CYBA gene will protect bone from the effects of DM We tested this hypothesis using genetically modified mice expressing normal levels of CYBA (+/+), one copy of the CYBA gene (+/-), or no copies of the CYBA gene (knock out -/-) DM is induced by high-fat diet and streptozotocin injections, and control non-diabetic mice are fed a low-fat diet Changes in blood glucose, body weight and bone mineral density (BMD) are being quantified throughout the study at 4 time points (initial: T0, 1 month: T1, 2 months T2, and 3 months: T3 BMD is quantified by Piximus DEXA Results of the first cohort of mice demonstrated that DM mice of all genotypes exhibited increased blood glucose at T2 and T3 and no changes in body weight, demonstrating that the CYBA gene is not needed for DM induction Diabetic mice of all genotypes exhibited lower BMD in the total body, spine and femur compared to control mice However, due to the low mouse number per group, statistical significance could not be established Ongoing studies are being performed with additional cohorts of mice to validate these preliminary studies During this research, I learned how to genotype mice, analyze BMD and graph results I met with the lab manager, the technician and with Dr Bellido regularly and participated in the weekly lab meetings

Lowe, Tamara, University of Arkansas - Little Rock, “The Role of Estrogen- Related Receptor Alpha in Androgen Receptor Signaling” **P32**

Current androgen deprivation therapies initially work; however, most cases eventually develop castration resistant prostate cancer (CRPC) Identifying new therapeutic targets is critical for improving treatment outcomes The orphan nuclear receptor Estrogen-Related Receptor Alpha (ESRRA) has been shown to support the growth, progression and stemness of CRPC While CRPC is androgen independent, Androgen Receptor (AR) signaling is still active Our lab is looking at how ESRRA interacts with AR signaling We hypothesize that ESRRA modulates AR regulated gene expression programs through direct protein-protein interactions We first started with a LNCaP-R1881 model, representing an androgen dependent model, and C4-2 cells representing CRPC We then treated these cells with the ESRRA inverse agonist XCT-790, to inhibit ESRRA We will examine the drug effect on AR regulated gene expression, as well as proliferation in the presence or absence of R1881 Finally, we validated the interaction of the two proteins

using co-immunoprecipitation. Utilizing domain deletion mutants we will map the interaction site. We are currently analyzing gene expression using PCR, validating the domain deletion plasmids, and optimizing antibody conditions for western blot. The results of this study can be utilized to design novel therapeutics targeting the interaction between AR and ESRRA.

Maxwell, Avery, Ouachita Baptist University, "Developing eDNA tools to identify the geographical distribution of endangered madtoms in Arkansas" **P33**

Caddo (Noturus taylori) and Ouachita (N. lachneri) madtoms are small catfish endemic to central and southwestern Arkansas. The IUCN have listed both species as endangered and they are considered to be species of conservation concern in Arkansas due to habitat loss and limited geographical distribution. Traditional seining methods for detection are time consuming and only marginally successful, therefore we developed environmental DNA tools as they are non-invasive and efficient in detecting the presence of species in aquatic habitats. We used cytochrome oxidase 1 sequences from GenBank to develop species-specific primers and Taqman probes and used qPCR methods to refine to test for the presence of madtoms from streams where they are likely to be present and from areas where they do not occur. Extracted DNA from tissue samples of Ouachita, Caddo, and Mountain (N. eleutherus) madtoms were used as positive and negative controls to optimize PCR conditions and ensure that non-target species were not identified. Four liters of water were collected per site, water was filtered, and DNA extracted from filters. eDNA extracts were then tested to determine if target species DNA were present. We will discuss our work on developing a qPCR protocol using species-specific primers and probes as well as our findings on the current geographical distribution of Caddo and Ouachita madtoms.

Mays, Ravyn, University of Arkansas - Little Rock, "A Saccharomyces boulardii chassis to deliver therapeutic proteins that inhibit diarrheal pathogens in the GI Tract" **P34**

Diarrheal bacteria such as Clostridioides difficile and Shiga Toxin-producing E. coli can cause severe colon ulcerations, intestinal inflammation, and fulminant diarrhea by releasing destructive toxins and disrupting the gut's natural microbiome. The annual burden of Clostridioides difficile alone in the U.S. is estimated at 453,000 cases, encompassing both health care-associated and community-associated incidence. There are currently commercial antibiotics used to suppress diarrheal bacteria in the gut microbiome. However, in the process of minimizing harmful bacteria, these antibiotics also target healthy bacteria, thereby leaving the gut susceptible to reinfection. To address this, we are interested in genetically engineering the probiotic Saccharomyces boulardii (Sb) to secrete recombinant therapeutic proteins directly at the site of infection to selectively

inhibit diarrheal bacteria Sb is an over-the-counter probiotic yeast used to manage diarrhea and restore the gut microbiome; it is also capable of secreting heterologous proteins, making it a reliable chassis We have genetically engineered Sb strains using microbiology techniques, plasmid purification and digestion, Gibson Assembly, electroporation, Polymerase Chain Reactions, and Li/AcSS Carrier DNA/PEG method transformation We successfully cloned secretion systems into Sb encoding 15 therapeutic payloads Our next steps are to perform an ELISA to detect protein secretion, and immediately afterward, we will assess whether the secreted proteins are functional using assays to determine toxin binding and direct antimicrobial activity against the bacterial pathogens

McJunkins, Maci, Ouachita Baptist University, "A Toxic Tale: An XRF Analysis of Arsenic in Victorian Bookbindings" **P35**

What do Paul Cezanne, Claude Monet, Vincent Van Gogh, and Napoleon Bonaparte have in common? Arsenic has been identified as a potential contributing factor to each of their health declines, and potentially their deaths In conjunction, they all enjoyed the use of a vibrant green, Emerald green, from the artists paints to Napoleon's wallpaper This phenomenon is caused by the arsenic and heavy metals used to make such vibrant colors These pigments were found to have been widely used in Victorian bookbindings The Poison Book Project, developed by the University of Delaware, in collaboration with the Winterthur Museum, Garden & Library, investigates these suspect bookbindings The chemistry department and the Riley-Hickingbotham Library of Ouachita Baptist University have partnered in their own Poison Book Project, where the library's over 600 Victorian titles are being catalogued and those that appear to potentially harbor emerald green pigments will be analyzed utilizing X-ray fluorescence spectroscopy These tests will show any indication of arsenical compounds and toxic heavy metals, ensuring that these titles can be safely handled by students and staff The decomposition of the books as they age can pose safety concerns from the flaking and transfer of particles from the books to other surfaces including human skin Research at Ouachita Baptist University has catalogued over 200 titles thus far, and narrowed them down to over 60 Victorian titles showing visual signs of potential arsenic-based green pigments Of the books tested by the University of Delaware, roughly 11 percent of the Victorian titles test positive for arsenic, and a similar amount is estimated to be found in the Riley-Hickingbotham Library To ensure the safety of students and staff, titles found to be hazardous will be placed under handling protocols, such as gloves, restricted access, and warning labels

Moore, Milo, Hendrix College, "Using the yeast model system to gain insights into a recently described neurodevelopmental condition in humans" **P36**

In cells, genetic information is stored as DNA. To perform necessary functions, cells require large amounts of DNA, which must be compacted to fit within the cell. During compaction, DNA is wrapped around protein complexes, forming nucleosomes, which further condense into chromosomes. While nucleosomes are essential, they act as roadblocks for RNA polymerase during the process of gene transcription. To overcome these roadblocks – as well as to re-establish them following transcription – RNA polymerase uses several factors, including one called FACT. In previous work in yeast, we showed that specific alterations in a region of the nucleosome, which we refer to as the ISGI region, impair interactions between DNA and yeast FACT (yFACT). Interestingly, one of the ISGI mutants we have studied has recently been implicated in a human neurodevelopmental condition called Bryant-Li-Bhoj syndrome (BLBS). The connection between an ISGI mutant and BLBS has motivated us to use yeast as a model for gaining insights into this human condition. In this poster, we present our strategy for assessing the molecular defects conferred by BLBS histone mutants in yeast and also describe some recent progress we have made so far this summer towards this goal. Because yeast and human cells share a high degree of functional similarities, our work can provide insights into the molecular mechanisms responsible for BLBS.

Morales-Castillo, Lydia, Lyon College, "Optimization of the Purification of the TraI Helicase" **P37**

Antibiotic resistance in bacteria is rapidly increasing due to ubiquitous usage of antibiotics in medicine and agriculture. The spread of antibiotic resistance starts from a donor bacterial cell that possesses a specific plasmid that carries an antibiotic resistance gene. During the process of conjugation, the donor cell will insert a strand of the plasmid's DNA into another bacterial cell, allowing the recipient bacterial cell to now possess a copy of the plasmid's genetic code. The specific helicase TraI is the enzyme that unwinds the plasmid during conjugation of F-family plasmids. Our overall focus is to identify the biochemical mechanisms behind the highly-efficient DNA unwinding abilities of TraI. The goal of our current project is to purify TraI through affinity and anion-exchange chromatography to allow for use in in vitro DNA unwinding assays. We passed our bacterial lysate through a nickel column that binds histidine residues at the N-terminus of TraI, which allows untagged proteins to pass through the column, isolating TraI along with other contaminants that also sticks to the column. Subsequently our solution was passed through an anion exchange column to further isolate the TraI by removing other contaminant proteins. In our first attempt we observed successful binding of TraI to the chromatography columns, but we were not fully successful in purifying TraI away from other contaminants. Our next goal will be to refine the wash and elution conditions and try additional affinity purification methods to successfully isolate TraI.

Nai, Mohammed, University of Arkansas - Little Rock, “Mice with Duchenne Muscular Dystrophy exhibit increased susceptibility to Glucocorticoid-Induced Bone Fragility” **P38**

Duchenne muscular dystrophy (DMD) is a genetic neuromuscular disorder that progressively causes severe impairment in muscle and bone, significantly impacting daily life. Lifelong glucocorticoid (GC) therapy is the standard of care for DMD because it suppresses inflammation and slows muscle function loss. Patients with DMD are at high risk of fractures, with approximately 75% exhibiting a fracture by the age of fifteen. Despite initially stalling the muscle disease, prolonged GC use is associated with adverse skeletal effects, such as increased bone loss and fracture risk. Currently, there is a need for novel therapeutic strategies to address the GC-induced bone fragility and musculoskeletal atrophy, representing an unmet clinical challenge for patients with DMD. The objective of this study was to investigate the impact of GC on bone in DMD musculoskeletal disease. To do so, we examined the effects of the commonly prescribed GC, prednisolone, on the bone structure of mdx mice. Three-month-old mdx mice and wild-type (WT) littermates (n = 12-15) received prednisolone or placebo treatment and were evaluated after 2wks of treatment. Femoral heads were harvested and analyzed using micro-computed tomography (μCT) to assess trabecular bone microarchitecture. μCT analysis showed that mdx mice exhibited greater trabecular separation and thinner trabeculae compared to WT controls. Prednisolone treatment significantly reduced bone volume fraction (BV/TV) in both genotypes; however, GC-treated mdx mice demonstrated greater deterioration of trabecular microarchitecture than GC-treated WT mice, including reduced trabecular number (Tb.N; 10.9 vs. 10.3, 1/mm, p=0.01) and increased trabecular separation. These findings indicate that bone in mdx mice is more susceptible to GC-induced bone fragility than bone in WT mice.

Narravula, Akshath, National Center for Toxicological Research (NCTR), “Dose and Time Dependent Disruption of Intestinal Barrier Genes by a Prohibited Antifungal” **P39**

Background: Due to consumer safety concerns, the impact of antimicrobial agents in food animals is closely monitored by regulatory agencies. Despite this, exposure to residual compounds persists, particularly through imported seafood. “Compound X”, a prohibited antifungal previously utilized in aquaculture, continues to be detected in seafood. This study investigates its impact on intestinal epithelial barrier functions, a critical component of host defense. Methods: RNA was extracted from T-84 cells exposed to varying concentrations (3μm, 30μm, 50μm, 200μm) of “Compound X” for 24 or 72 hrs. DNase-treated and reverse transcribed into cDNA using an Invitrogen SuperScript VILO kit. Gene expression was analyzed using the Qiagen RT^2 Profiler Human Cell Junction

Pathway Assay, with GAPDH as the normalization control. Relative expressions were calculated using Qiagen's Gene Globe software. Results: "Compound X" induced dose and time-dependent changes in gene expression and signaling pathways. Key epithelial barrier genes, including CLDN10, NOTCH2, and ITGB2, were significantly altered. Increased concentrations upregulated CLDN10, suggesting disruption of tight junction integrity, while NOTCH2 downregulation indicates impaired epithelial regeneration. Additionally, activation of leukocyte extravasation, granulocyte adhesion, and IL-8 pathways suggests increased inflammatory responses. Alterations in the actin cytoskeleton and integrin further indicate structural remodeling of epithelial cells. The effects were most pronounced at higher concentrations and longer exposure durations. Conclusion: This study demonstrates that "Compound X" disrupts intestinal epithelial barrier integrity through effects on junctional, cytoskeletal, and immune signal pathways. These findings provide mechanistic insight into how residual exposure may increase intestinal permeability and susceptibility to infection, highlighting potential public health risks.

Nelson, Maddie, Ouachita Baptist University, "The Megacancer Project; Selection and Characterization of Highly Invasive H460 Lung Cancer Cell Lines" **P40**

To identify novel biomarkers associated with increased invasive cancer phenotype, highly invasive H460 (non-small cell lung cancer) cell lines were developed using repeated Transwell invasion assays. The most invasive cells were collected and used for the next assay. This was repeated eight times, resulting in the formation of the H460-I8 cell lines. These aggressive cell lines exhibited a greater invasive phenotype than the parental H460 cells. To further characterize the migratory capacity of the parental and I8 cell lines, wound-healing assays were performed in 6 well plates with 6 replicates of each cell line, and scratch width was measured at 0, 24, 48, and 72 hours. To investigate the role these cells play in the tumor microenvironment conditioned media was collected from each cell line to determine whether cell-secreted factors influence migration. Parental H460 cells were cultured in the conditioned media from the parental and I8 cell lines and evaluated using a wound healing assay to assess changes in wound closure after being exposed to conditioned media. [Results will be presented at the poster presentation.] This research aims to characterize the migratory behavior of the I8 H460 cell lines, compared to the parental, and determine whether factors secreted by invasive cell lines alter the migratory behavior of parental H460 cells.

Obi, Joan, University of Arkansas – Fayetteville, "Investigating the Role of Notch3 in Dormant Multiple Myeloma Cells." **P41**

Multiple myeloma (MM) occurs due to uncontrolled growth of plasma cells in bone marrow, leading to a vicious cycle of tumor growth and bone destruction. Despite

advances in therapeutics, MM is incurable in most patients due to high rates of disease relapse driven by the development of drug resistance and outgrowth of residual dormant MM cells. Dormancy is a temporary state of cell cycle arrest that is induced in a small population of MM cells upon interactions with the tumor microenvironment. However, the specific molecular interactions involved in initiating dormancy are not well defined. Previous work has shown that Notch3 signaling, which is initiated through physical contact between two cells, promotes tumor growth and drug resistance in MM. The Delgado-Calle lab found that systemic Notch3 inhibition in vivo with a neutralizing monoclonal antibody (N3-ab) decreased dormant cells without causing apoptosis, suggesting that Notch3 signaling promotes dormancy. However, it is not clear if Notch3 signaling in tumor cells or the bone marrow is responsible for inducing dormancy in MM cells. We hypothesize that inhibition of Notch3 in MM cells will reduce their entry into dormancy. To test this hypothesis, we used an ex vivo model where 100,000 5TGM1 GFP + cells pre-treated with either vehicle or N3-Ab were injected into explanted murine tibiae and femurs that were pre-treated with vehicle or N3-ab. After 4 days, we will harvest cells from the bone and perform flow cytometry to quantify dormant cells using GFP (tumor cell marker) and AXL (dormancy marker). While this experiment is still ongoing, we expect that both groups receiving cells pre-treated with N3-ab will have fewer dormant cells than groups with vehicle pre-treatment, as we expect that Notch3 in MM cells, and not in bone marrow, is required for dormancy.

Ocho, Mishel, Wake Forest University, “Rewarding effects of compounds commonly found in kratom products” **T6, P77**

Kratom products containing mitragynine (MG, the primary psychoactive compound in kratom) and/or 7-hydroxymitragynine (7-OH, a human metabolite and semi-synthetic analog of MG) have become available in shops across the United States. MG has been shown to be a partial agonist at mu opioid receptors and to produce mild stimulant-like effects, while 7-OH exhibits greater binding affinity for and efficacy at mu opioid receptors, but their abuse-related effects are poorly understood. The goal of this study was to examine the rewarding effects of MG and 7-OH in adult male C57BL/6J mice (n=6 per group) using the conditioned place preference assay. After four daily pairings, 100 mg/kg MG and 10 mg/kg 7-OH elicited place preferences, but conditioning with smaller doses of either drug had no systematic effects. The mu opioid receptor antagonist naltrexone (1 mg/kg) blocked the development of place preference in mice treated with 10 mg/kg 7-OH, implicating opioid receptors in the mediation of these rewarding effects. In daily extinction trials, the preference elicited by 100 mg/kg MG extinguished faster than that conditioned by 10 mg/kg 7-OH. Following extinction, “priming” injections of 10 mg/kg MG or 1 mg/kg 7-OH – but not injection of the drug vehicle – reinstated preference in mice initially conditioned

with 7-OH. Similarly, in mice conditioned with MG, preference was reinstated by 1 mg/kg 7-OH but not by vehicle. The results of this study suggest that both MG and 7-OH produce rewarding effects in mice, most likely via agonist binding at opioid receptors. Following extinction, these effects can be reinstated by injection of 10-fold smaller doses than those that were required to produce the initial preference. Overall, these results suggest that MG and 7-OH would likely elicit abuse-related effects in humans, including acute rewarding effects and persistent craving and/or drug seeking when exposed to drug-associated cues.

O'Daniel, Devyn, Arkansas State University, "The roles of sexual selection and natural selection in a polyphenic nematode" **P42**

Sexual selection can favor traits that increase mating behaviors and the number of offspring produced, while natural selection can favor traits that increase fitness of the species as a whole. Although these selection processes can exist parallel to each other, they can also contradict depending on their environment. This contradiction can be observed by studying the polyphenic nematode Pristionchus pacificus. P. pacificus exhibits two distinct morphs in its oral cavity depending on resource availability during development, which results in two different feeding strategies. One is the microbivorous stenostomatous (St) morph, which is favored under high bacterial availability, while the other is the predatory eurystomatous (Eu) morph, which is favored under low resource availability. Males tend to be biased to the St morph, suggesting that male sexual selection favors the faster maturation trait associated with the St morph. However, in a B12 environment, the Eu morph tends to be favored more via natural selection. In this experiment, we facilitate a direct competition between one St male and one Eu male for mating with one hermaphrodite to test the differences in male fitness between the two morphs as well as how natural selection in developmental and competitive environments affects the different phenotypes. These experiments aim to test whether natural selection or sexual selection have a greater effect on male fitness, if a B12 environment changes competitive dynamics for males, and if competitive dynamics are affected by the mouth phenotype itself or a specific gene.

Offordum, Jessica, Grambling State University, "Investigating Histone Modification Changes At FAM60A regulated gene promoters" **P43**

FAM60A is a component of the Sin3/HDAC complex, which helps regulate gene expression by modifying chromatin structure. Because the Sin3/HDAC complex is generally involved in turning genes off, loss of FAM60A is expected to increase the expression of its target genes. However, previous RNA-sequencing studies showed that loss of FAM60A caused some genes to increase in expression while others decreased, suggesting that FAM60A may have a more complex role in gene regulation. The goal of this study is to

determine whether changes in chromatin structure contribute to these differences in gene expression To investigate this, chromatin immunoprecipitation followed by quantitative PCR (ChIP-qPCR) will be performed in control and FAM60A knockout cells Antibodies against the active histone marks H3K4me3 and H3K27ac will be used to isolate chromatin associated with actively expressed genes, and qPCR will be used to measure enrichment at selected gene promoters Six genes that showed large expression changes following FAM60A loss were selected for analysis These include three downregulated genes (WWC3, SOX11, and TNFRSF10A) and three upregulated genes (BCHE, MEOX2, and TACR3) We will examine the levels of H3K4me3 and H3K27ac at the promoter regions of these genes to determine whether changes in chromatin activation are associated with their altered expression This study will improve our understanding of how FAM60A regulates gene expression and chromatin structure It may also help explain why loss of FAM60A can lead to both increased and decreased expression of different genes

Paez, Samuel, John Brown University, "Alchemically Assessing Boiling Point Elevation Via Water Chemical Potential in Sodium Chloride Solutions with Molecular Dynamics"
P44

With the advent of computational chemistry in the 1950s, researchers now have a whole new world of opportunities for discovering the physical properties of matter, even thermal properties notorious for being extremely difficult to measure experimentally Particularly, molecular dynamics allows for statistical predictions which could otherwise be unsafe, costly, or inaccurate in a traditional laboratory setting Yet, a large gap remains to be bridged between fundamental atom-atom interactions and macromolecular phenomena in biochemistry, such as protein-solvent salting effects The present study aims to determine whether the four-site BLYPSP-4F water model predicts boiling point elevation, which is a well-documented colligative property, in sodium chloride solutions by alchemically measuring the free energy of vaporization with Bennett's Acceptance Ratio (BAR) As a result, the impact of electrolytes on the chemical potential of liquid water will be elucidated, hopefully revealing potential applications of the Hofmeister series to medical and industrial purposes Current results reveal good equilibration, but nonlinear overestimation of boiling points within 0-6M NaCl persists despite excellent coefficients of determination across 370-430K In addition, the enthalpy of vaporization oscillates about 45 91kJ/mol, in agreement with the assumptions of the free energy method described

Park, Clara, Rhodes College, "A conserved aromatic residue that structurally mediates DNA melting is dispensable in vivo" **T2, P 73**

DNA replication is the fundamental life process of duplicating genetic material in preparation for cell division In eukaryotes, the minichromosome maintenance (MCM)

complex forms a hexameric ring that separates the constituent strands of the DNA double-helix so that each can serve as a template in the synthesis of DNA For this replication process to begin, a small region of DNA base-pairs must be broken in a process termed "melting" Our previous cryo-EM study showed that the MCM ring directly mediates DNA melting with fully conserved aromatic residues of adjacent subunits that stack on DNA sugars and bases, suggesting an essential function To determine the features of this residue that are essential in vivo, we generated all 19 mutants of Saccharomyces cerevisiae Mcm6 Y621 in test plasmids carrying a TRP1-selectable marker Each plasmid was transformed into a yeast strain with an MCM6 gene deletion and a wild-type MCM6 gene on a plasmid with a URA3-selectable marker Single colonies carrying both plasmids were selected on agar plates with medium lacking uracil and tryptophan Cultures were grown from the selected colonies and then spotted onto plates with or without 5-fluoroorotic acid (5-FOA) 5-FOA selects against URA3-containing plasmids, such that yeast strains are only viable if the MCM gene on the TRP1-containing test plasmid supports all essential in vivo functions of Mcm6 All of the Mcm6 Y621 mutants grew on the 5-FOA condition, indicating that the fully conserved aromatic residue at this position does not provide an essential function in vivo The single Mcm6 Y621 mutants may retain viability due to the presence of aromatic residues on neighboring subunits Future studies will determine the essential roles of these adjacent aromatic residues and whether yeast strains with mutation of multiple aromatic residues are also viable

Powell, Zhychaeus, Philander Smith University, "TUNEL-Positivity in Acute Kidney Injury:"
P45

Cells with DNA Damage Are Alive: Injury Decreases Natural Recovery Process of Cells Background: TUNEL (terminal deoxynucleotidyl transferase dUTP nick end labeling) positivity has been interpreted as cell death for decades But TUNEL detects DNA damage, and DNA damage can be repaired Cells can recover from the brink of death via anastasis, an active recovery process marked by SNAIL, a zinc-finger transcription factor required for recovery Whether this happens in the injured kidney is unknown Methods: We examined three mouse models of acute kidney injury (AKI): cisplatin nephrotoxicity, glycerol-induced rhabdomyolysis (muscle breakdown that causes kidney damage), and renal ischemia-reperfusion (IR; interruption and restoration of blood flow) We stained 4 different kidney samples from each mouse model for TUNEL and SNAIL using multi-label immunofluorescence We manually counted TUNEL+ cells, SNAIL+ cells, and colocalization in 3 images from each sample Renal function was assessed by BUN (blood urea nitrogen) and creatinine, two markers of kidney filtration capacity Results: All three injury models significantly elevated BUN and creatinine, confirming injury In controls, 21 4% of TUNEL+ cells co-expressed SNAIL, suggesting active repair or recovery However,

injury reduced this co-localization: 7□1% in cisplatin, 3□6% in rhabdomyolysis, and 1□6% in IR□TUNEL positivity increased significantly only in the IR group; cisplatin and rhabdomyolysis showed elevated TUNEL that didn't reach significance□Conclusion: TUNEL-positive cells express SNAIL in healthy kidneys, but injury suppresses this expression□Our data suggest that anastasis is a feature of normal tissue homeostasis, not a response to injury□Injury overwhelms the recovery program, pushing cells toward death□ The gradient of repair marker loss (21□4% → 1□6%) represents a signal for recovery potential and identifies the suppression mechanism as a therapeutic target for preserving repair in AKI

Ramirez, Sonja, Lyon College, "Optimization of the Purification of the TraI Helicase" **P46**

Antibiotic resistance in bacteria is rapidly increasing due to ubiquitous usage of antibiotics in medicine and agriculture□The spread of antibiotic resistance starts from a donor bacterial cell that possesses a specific plasmid that carries an antibiotic resistance gene□During the process of conjugation, the donor cell will insert a strand of the plasmid's DNA into another bacterial cell, allowing the recipient bacterial cell to now possess a copy of the plasmid□s genetic code□The specific helicase TraI is the enzyme that unwinds the plasmid during conjugation of F-family plasmids□Our overall focus is to identify the biochemical mechanisms behind the highly-efficient DNA unwinding abilities of TraI□The goal of our current project is to purify TraI through affinity and anion-exchange chromatography to allow for use in in vitro DNA unwinding assays□We passed our bacterial lysate through a nickel column that binds histidine residues at the N-terminus of TraI, which allows untagged proteins to pass through the column, isolating TraI along with other contaminants that also sticks to the column□Subsequently our solution was passed through an anion exchange column to further isolate the TraI by removing other contaminant proteins□In our first attempt we observed successful binding of TraI to the chromatography columns, but we were not fully successful in purifying TraI away from other contaminants□Our next goal will be to refine the wash and elution conditions and try additional affinity purification methods to successfully isolate TraI□

Ramirez Santamaria, Javier, University of Central Arkansas, "Comparative Study of Bipyridine and Mebim-pic Ligands in Homogeneous Rhenium Catalysts for Enhanced Captured CO_2 Reduction" **P47**

Over the years, the reliance on fossil fuels for energy has grown, leading to a significant rise in atmospheric carbon dioxide levels□This research focuses on developing a rhenium-based electrocatalyst capable of converting captured carbon dioxide into carbon-based commodities□Two ligand systems were investigated to prepare and evaluate potential captured CO_2 reduction catalysts□First, the N-heterocyclic carbene ligand bim-

pic was synthesized from benzimidazole and 2-bromomethylpyridine hydrobromide using potassium hydroxide in THF. The successful synthesis of bim-pic was confirmed through thin-layer chromatography and 1H NMR analysis, with yields reaching up to 73%. The ligand was subsequently alkylated to form [Mebim-pic]I, which was coordinated with $Re(CO)_5Br$ to synthesize the rhenium complex $Re(Mebim\text{-}pic)(CO)_3Br$. In parallel, the bipyridine (bpy) ligand system was explored as a benchmark for comparison. The precursor $Re(CO)_5Br$ was synthesized from $Re_2(CO)_{10}$ and bromine with a 67–88% yield and subsequently reacted with 2,2'-bipyridine in toluene to produce $Re(bpy)(CO)_3Br$ in 94–65% yield. The synthesized complexes were characterized using NMR and IR spectroscopy to confirm their structures. These complementary ligand systems provide a foundation for evaluating the influence of ligand design on the catalytic performance of rhenium complexes for the electrochemical reduction of captured carbon dioxide

Rana, Simra, Haas Hall Academy Fayetteville, “Computational Modeling of Interstrand Crosslink Repair in Fanconi Anemia” **P48**

Fanconi anemia (FA) is a rare inherited disorder caused by mutations in genes responsible for repairing DNA damage, leading to bone marrow failure and a greatly increased risk of cancer. A defining feature of FA is defective repair of DNA interstrand crosslinks (ICLs), lesions that physically link the two strands of DNA and block essential processes such as replication and transcription. Because ICLs stall replication forks and can generate double-strand breaks if not resolved correctly, cells must coordinate multiple DNA repair systems to restore genome stability. Successful ICL repair requires the FA core pathway to recognize damage and activate the FANCI–FANCD2 complex through ubiquitination, followed by downstream repair through homologous recombination (HR) and nucleotide excision repair (NER) to complete the repair process. Many studies describe these pathways individually, but fewer approaches capture how they work together dynamically over time or how breakdowns in coordination contribute to the FA disease phenotype. The goal of this project is to build a computational model that integrates the FA core pathway, HR, and NER into one quantitative framework. Using rule-based modeling in PySB and fitting parameters to published experimental data, this model will simulate how DNA damage is detected, processed, and repaired, as well as how key repair complexes change over time. Understanding these interactions in a quantitative, systems-level way may help explain why FA cells accumulate unrepaired DNA damage and could highlight steps in the repair process that may be promising targets for future therapeutic strategies.

Richards, Andrea, University of Arkansas – Fayetteville, "Investigating the Impact of Microhomology-Mediated End Joining Repair in Fanconi Anemia Using Computational Modeling" **P49**

Fanconi anemia (FA) is a hereditary disorder characterized by defects in DNA interstrand crosslink repair and double-strand break (DSB) resolution, resulting in genomic instability and increased cancer susceptibility. DSBs are repaired through multiple pathways, including high-fidelity homologous recombination (HR) and error-prone microhomology-mediated end joining (MMEJ), with pathway choice influenced by protein recruitment dynamics and DNA end resection. Understanding the determinants of repair pathway selection is essential for elucidating FA pathophysiology. A computational model was developed to simulate DNA damage recognition and repair in FA cells, incorporating key mediators such as PARP1, MRE11, CtIP, POLQ, and LIG3. Ordinary differential equations (ODEs) were used to describe the kinetics of protein-DNA interactions, end resection, and competition among HR, MMEJ, and nucleotide excision repair (NER) pathways. Simulation results indicate that MMEJ can compensate for HR deficiencies under specific cellular conditions, providing insight into the factors that drive preferential use of error-prone repair mechanisms. This modeling framework enables integration of experimental data, prediction of repair pathway utilization, and mechanistic exploration of how modulation of specific repair factors affects genomic stability. These findings contribute to a deeper understanding of DNA repair regulation in FA and may inform potential therapeutic strategies.

Rodgers, Jordan, University of Central Arkansas, "Benzimidazole-based N-Heterocyclic Carbene (NHC) Ligand Synthesis" **P50**

The heavy reliance on burning fossil fuels in the modern world has caused the widespread release of CO_2 as a waste product, a well known greenhouse gas. To prevent releasing more carbon dioxide into the atmosphere from burning fossil fuels, research towards converting CO_2 into natural gas has been explored. To convert CO_2 into CO, an ingredient used in natural gas production, electrochemical reduction is required. Currently, the energy cost of this process is too high for widespread commercial use. We theorize that a manganese catalyst with NHC (N-heterocyclic carbene) ligand attachments would make the reduction more effective. The specific ligands proposed for the catalyst are 3-methyl-1-picolylbenzimidazol-2-ylidene (MeBim-pic) and 3-methyl-1-(6-methylpicolyl)benzimidazol-2-ylidene (MBM-pic). 1-methyl-benzimidazol-2-ylidene-3-(2-pyridine) (MeBim-py) is used as a comparison. It is theorized that as the ligands crowd the manganese center, its effectiveness increases and properties change, thereby lowering the energy cost to reduce CO_2. This research focuses on synthesizing these ligands effectively. Proton NMR and thin

layer chromatography (TLC) are used to verify structure and purity This research can give insight into how different ligand structures affect catalytic properties

Rucker, Kiah, Philander Smith University, "Assessment of Urinary Heavy Metals Concentrations and Geospatial Exposure in the Arkansas Rural Community Health Study" **P51**

Background Environmental exposure to heavy metals has been identified as a potential risk to breast cancer, yet the relationship between environmental contamination and human exposure remains unclear Arkansas Rural Community Health (ARCH) Study, a population-based cohort, examined breast cancer risk in rural Arkansas, providing a unique opportunity to study environmental exposures and breast cancer The current follow-up of ARCH, the ARCH-HER study, builds upon the existing cohort by closely examining environmental exposures that may contribute to breast cancer risk Although the original study identified increased breast cancer risk with certain airborne exposures, changes in exposure over time, and residential mobility were not evaluated We hypothesize that individuals in areas with higher arsenic and chromium exposure will have higher urinary concentrations of these metals, reflecting greater exposure and breast cancer risk Objectives To determine whether higher exposure concentrations of arsenic and chromium compounds are reflected in urine samples and whether they indicate an increased risk of breast cancer Methods Urine samples from the ARCH-HER study were analyzed for heavy metals by inductively coupled plasma mass spectrometry (ICP-MS), with analyses focused on arsenic and chromium compounds County-level AirTox and NATA data from available years were compiled and geospatially mapped to characterize exposure patterns across Arkansas counties Cumulative exposure concentrations were estimated using LexisNexis-derived residential histories and assessed for associations with urinary arsenic and chromium concentrations Conclusion The findings will provide insights into whether environmental contamination concentrations are reflected in individual exposures and associated with increased breast cancer risk This approach combines geospatial and chemical analyses to support future investigations into environmental contributors to breast cancer

Sagarnaga Rivera, Lídize, Arkansas State University, "Skin pattern as a potential monitoring tool for frog populations" **P52**

Population monitoring, which requires identifying individuals, is crucial for species conservation Common techniques involve marking, which can harm animals and alter their behaviors Natural markings are an alternative not yet explored for amphibians Yet, the traditional method with elastomers (fluorescent implants) is invasive and not completely reliable—the tag may become lost or hard to see The American green tree frog

(Dryophytes cinereus) possesses naturally occurring and distinct golden dots on their dorsal side. Therefore, we aim to assess whether patterns of golden dots on the skin of green tree frogs are unique and can serve as a noninvasive individual identification method or if they change over time. To do this, we attempted to capture, tag, and photograph wild green tree frogs weekly in April-May 2026 at the Crowley's Ridge Nature Center. Additionally, we captured 20 frogs that we kept in the lab and also photographed weekly. From images processed in GIMP and ImageJ, we will report on the presence and/or changes in the frogs' skin patterns over time. With skin patterns being widely present in amphibians, this noninvasive and cost-effective method could be applied to other species and improve current conservation strategies by allowing for a more permanent population monitoring.

Scarth, Ian, University of Hawai'i at Hilo, "Evaluating a DDM-Assisted Sample Preparation Method for Quantitative Proteomics of Melanoma Cells" **P53**

Melanoma is the deadliest form of skin cancer, and immune checkpoint blockade (ICB), its most effective immunotherapy, benefits only about half of patients. Recent work has shown that upregulating the transcription factor ATF6 enhances anti-tumor immune response and ICB efficacy, making the ATF6 pathway a target of interest for improving treatment. Assessing this pathway depends on quantitative proteomics, whose depth and reliability are determined largely by sample preparation. Protein is lost throughout preparation to surface adsorption on tubes, tips, and plates, a loss that becomes limiting at low input and disproportionately affects low-abundance proteins. This project evaluates whether introducing the mass spectrometry compatible surfactant n-dodecyl-β-D-maltoside (DDM), which keeps protein in solution during handling, alters proteome recovery relative to the standard chloroform-methanol extraction (CME) workflow. Using HeLa cells for optimization, a DDM-assisted digest (0.02% DDM at resuspension) was benchmarked against the standard CME digest by data-independent acquisition on an Orbitrap Astral. Overall protein identifications were nearly identical (5,969 of roughly 5,990 shared, over 99%), but the two methods recovered partly distinct peptide populations: of 61,808 distinct peptides, DDM uniquely recovered 1,072 and CME 683. The DDM-unique peptides were significantly more hydrophobic and lower in abundance than the CME-unique set, indicating that the two preparations sample the proteome through different peptides rather than differing in overall protein depth. Because low-abundance detection is where a preparation difference can be most consequential, this distinction is directly relevant to detecting treatment-driven changes. Work is ongoing to extend the comparison to B16F10 and YUMM1.7 melanoma cells under bortezomib treatment.

Sellers, Isabella, Samford University, “What is a “therapeutic” dose of acetaminophen in mice? A pharmacokinetics investigation?” **P54**

Acetaminophen (APAP) is a widely used analgesic and antipyretic, yet its therapeutic mechanism remains uncertain. Proposed mechanisms include cyclooxygenase inhibition, AM404 formation, and serotonergic signaling, but evidence for these effects is inconsistent. Concern has also grown that therapeutic APAP exposure, particularly during pregnancy, may adversely affect neurodevelopment or endocrine function. However, these associations remain controversial. One factor contributing to the confusion in these areas is the frequent use of APAP doses ≥ 150mg/kg in mice and rats compared to the 14-15 mg/kg doses taken by humans. We propose that rodent doses should by selected by matching serum or plasma APAP concentrations achieved in humans after therapeutic dosing. Eight-week-old male C57BL/6J mice received 30 mg/kg APAP by intrapersonal or oral administration. Serum APAP concentrations were measured at 15, 30 ,60, and 180 minutes after dosing by high-performance liquid chromatography with electrochemical detection. Pharmacokinetic parameters (Cmax, Tmax, and t1/2) were estimated directly and by noncompartmental analysis. Additional mice received 15, 30 50, or 100 mg/kg APAP to assess dose-dependent exposure at the Tmax. The 30 mg/kg doses produced a Cmax of 104 ± 12 μM, closely matching the approximately 100 μM mean Cmax reported in humans after a therapeutic dose. Other pharmacokinetic parameters were also similar to human doses, and no significant differences were detected between administration routes. Higher doses resulted in significantly greater concentrations that do not mimic human exposure. These findings support 30 mg/kg as a human-relevant mouse dose.

Sharma, Dishant, University of Southern California, “Empagliflozin Reprograms Mitochondrial Metabolism and Redox Pathways in vivo” **P55**

Colorectal cancer (CRC) is a leading cause of cancer-related deaths. It is associated with dysregulated mitochondrial metabolism which promotes tumor growth and metastasis. Individuals with diabetes are at increased risk for CRC, with worse prognosis. Empagliflozin (empa) is a SGLT2 inhibitor that reduces glucose reabsorption and lowers blood glucose. Preliminary data shows that empa reduced tumor progression in mouse models despite them having negligible expression of sodium-glucose cotransporter-2 (SGLT2), suggesting empa acts through metabolic pathways. Since colorectal tumors rely upon energy metabolism and redox regulation, we hypothesized that empa acts specifically upon the mitochondria. To investigate this, we examined empa’s role within the mitochondria and how it reprograms oxidative stress, metabolic signaling and tumor proliferation within males and females in vivo.

We measured protein expression for oxidative stress, proliferation, and apoptosis markers. However, upon analysis, we found variability among proliferation and apoptosis markers and unexpected sex differences in oxidative stress. Empa increased SOD2 (an enzyme that reacts to ROS) and decreased 4-HNE (byproduct of lipid oxidation) within males, while females showed an increase in 4-HNE, illustrating empa's sex specific effects on oxidative damage. We also found that apoptosis markers remained unchanged. Furthermore, in males, empa decreased markers for growth while cellular energy markers remained similar. This suggests that empa works through regulating growth and nutrient sensing rather than inducing cytotoxicity. At the gene expression level, ATP5F1 (Complex V) was significantly decreased in both males and females (p=0.0003), showcasing empa's effect on mitochondrial ATP production. Ongoing qPCR along with Seahorse Mito Stress test and RNA sequencing will help determine which pathways may be involved. Overall, the results suggest empa reprograms tumor mitochondrial metabolism.

Shorie, Himanshi, University of Arkansas - Fort Smith, "Bacterial Expression and Purification of Cdc42 and PBD46" **P56**

Cancer arises when the signals that normally control cell growth and movement become dysregulated. Cdc42 is a protein that relays these signals, acting as a molecular switch that alternates between an active GTP-bound state and an inactive GDP-bound state. In its active form, Cdc42 engages partner proteins such as PAK1 to regulate cell division, migration, and polarity. When regulation is lost, Cdc42 remains persistently active, and this sustained signaling contributes to the uncontrolled growth and invasion characteristic of breast, colon, and rectal cancers. Although Cdc42 is a compelling therapeutic target, no approved drugs act on it directly. The small molecule ZCL278 binds Cdc42 and interferes with its ability to engage PAK1, but its poor solubility in water limits its use as a drug candidate. Studying this interaction requires a dependable supply of both proteins in pure, correctly folded form. The goal of this project was to produce the proteins needed for these studies. Histidine-tagged Cdc42 was expressed in Escherichia coli and purified by nickel affinity chromatography using an FPLC system. Two small-scale test expressions confirmed soluble expression and optimized induction conditions, identifying five hours of IPTG induction as optimal. Five large-scale expressions were then completed, with purified protein confirmed by gel electrophoresis at approximately 23 kDa. PBD46, a peptide derived from PAK1 corresponding to the region that binds Cdc42, was also expressed and purified, allowing the interaction to be examined directly. Intrinsic tryptophan fluorescence titration is currently underway to estimate how tightly ZCL278 binds purified Cdc42. Expression of the Cdc42 T35A variant, which carries a change in the region where ZCL278 binds, has also been initiated to examine how alterations in this

region influence inhibitor binding. Together, this work provides the purified proteins needed for continued characterization of the Cdc42 and PAK1 interaction.

Shrivastava, Akul, Stanford University, "Using EEG-derived ERPs to Determine the Effect of Maternal Weight on Pediatric Neurodevelopment" **P57**

Background: Maternal overweightness during pregnancy has become increasingly common, and infants of gestationally overweight (GOW) mothers show poorer cognitive development than those of gestationally normal-weight (GNW) mothers. Whether this disparity persists into childhood or diminishes as the brain matures remains unknown. This study examined response inhibition, a core facet of executive function, using event-related potentials (ERPs) in five-year-olds born to GNW and GOW mothers, with the hypothesis that GNW children would show greater response inhibition than GOW children. Methods: Mothers of 45 five-year-old subjects were classified as GNW (n = 15, BMI 18.5–24.9) or GOW (n = 30, BMI 25–35). Children completed the Zoo Task, an age-appropriate Go/No-Go assessment, while EEG was recorded with a 128-sensor array. Data were preprocessed with a standard pipeline (0.5–45 Hz band-pass filter, REST referencing, Morlet wavelet artifact correction; all retained subjects kept >70% of channels) and analyzed in Brainstorm. Trials were coded as correct or incorrect to determine the correct-response negativity (CRN), error-related negativity (ERN), and error positivity (Pe) components. ANCOVA models compared these components, along with response accuracy, across groups. Results and conclusions will be shared upon completion of analysis. It is expected that compared to the children of GNW mothers, the children of GOW mothers will demonstrate lower accuracy rates for the Zoo Task as well as delayed and diminished CRN, ERN, and Pe signals.

Skipworth, Joshua, University of Central Arkansas, "Fentanyl and fentanyl analog quantification via blood micro-fluidic sampling and LC-MS/MS" **P58**

The opioid crisis poses legal, medical, and public health challenges because many synthetic opioids evade detection in drug surveillance programs; thus, complex testing is required to track drug trends. This complicates efforts that support frontline workers in emergency care, addiction, and public safety. This study validates a liquid chromatography-tandem mass spectrometry (LC-MS/MS) method for clinical testing of fentanyl and synthetic fentanyl analogs by coupling point-of-care micro-blood sampling devices with ToxBox Volumetric Immersive Blood Extraction (VIBE) filters. Sample collection and analysis were streamlined by premanufacturing devices with deuterium-labeled internal standards, which are required for trace level quantitation. Once samples are loaded, devices are easily transported through courier services or the USPS because these micro blood samples are exempt from DOT biohazard regulation. Upon receipt,

laboratories remove VIBE filters by separating a perforated edge on the shipment container. Samples are analyzed by reconditioning filters for 5 min with 20µl of ToxBox lysing solution. Fentanyl and fentanyl analogs are then extracted by equilibrating conditioned filters for 30 min with 1 ml of isopropanol. An 800 µl aliquot of the supernatant is transferred and dried under nitrogen for approximately 30 min at 37°C. Remaining drug residues are reconstituted in 200 µl ToxBox reconstitution solution and analyzed with a 2.5 min LC-MS/MS method using a Bruker EVOQ DART-TQ+. Gravimetric studies show the micro-fluidic devices accurately deliver 50 µl of whole blood. Accuracy/precision studies conducted with fortified blood containing fentanyl, 3-flurofentanyl, para-flurofentanyl, and acetyl fentanyl show that this method meets strict accreditation standards established for clinical and forensic laboratories. As proof concept, in vivo studies in mice were conducted to further relay product validity and its promising potential in drug surveillance efforts.

Spicer, Paige, Ouachita Baptist University, "Optimization of Amoxicillin-Loaded Electrospun Nanofiber Mats for Controlled Drug Delivery to Prevent Wound Infection"

P59

As advances have been made in the field of wound healing, a shift has occurred to focus on biomimetic materials due to their ability to mimic the structure and function of the extracellular matrix. Polysaccharides are one example of natural polymers that have been investigated as an appropriate biocompatible material to replicate the tissue fibers that the body naturally regenerates to assist in wound healing. The goal of this study was to develop and compare biocompatible nanofiber mats derived from chitosan and alginate that are equipped with amoxicillin to prevent wound infection. Alginate and chitosan are natural polymers that have been used in various biomedical applications including both wound healing and drug delivery. The negative charge carried by alginate and the positive charge carried by chitosan are what create slight differences in the ways these two compounds are utilized in biomedical applications ranging from gel-based wound dressings to drug delivery systems. Polymer solutions were created using polyvinyl alcohol as a structural component for both the alginate and chitosan-based fibers. Upon electrospinning the nanofiber mats, both light microscope and SEM imaging were performed to analyze the morphology of the fibers. Degradation assays were also performed to determine the structural stability of the nanofiber mats. Preliminary data suggests that the chitosan mats provide more stability and the most degradation resistance. Ongoing studies will likely determine that these natural polymer electrospun materials have the capability to assist in infection prevention and encourage cell growth when used as a wound healing treatment especially when equipped with an antibiotic such as amoxicillin.

Stutts, Spencer, Hendrix College, “Development of Low Weight and Power Microfluidic Pumping Systems for a Portable Microsampling Backpack” **T12**

The goal of this work is to develop low-weight, low-power microfluidic pumping systems for a portable microsampling backpack for rat studies Behavioral measurements in rodents often rely on constrained systems with swivels for electrical or fluidic connections Although tether-free platforms are of interest in neuroscience, most provide onboard sensing but lack advanced fluid handling or sample collection This work addresses that gap through an integrated backpack and microsampling system for portable fluid handling The current backpack is limited by the size, mass, and power demand of a stepper-motor-driven peristaltic pump, making pump miniaturization and power reduction central to extending sampling duration Three tasks support this goal First, an automated flow-imaging apparatus was tested and validated for real-time measurement of pump flow rates By imaging a droplet over time, it provides a faster alternative to manual gravimetric measurements, enables sub-second measurement intervals, and offers a platform for comparing pump designs Second, polydimethylsiloxane (PDMS)-based microfluidic chips were designed and fabricated with microchannels serving as custom peristaltic tubing This may reduce fluidic dead volume and the compression force required for peristaltic flow, enabling lower-weight, lower-power motors PDMS molds were refined through 11 design iterations to improve fabrication success and mechanical support of the microchannel Third, custom printed circuit board (PCB) designs are under development to provide battery power management, support piezoelectric pumping, enable wireless power and communication, and integrate spectrophotometric sensors Two board pathways based on Raspberry Pi RP2040 and Nordic Semiconductor nRF54 integrated circuits are nearing completion Remaining work includes improving PDMS chip fabrication reproducibility, developing reliable assembly of the complete pump, and fabricating and testing custom PCB designs

Tan, Christine, University of Arkansas - Little Rock, “Impact of Environmental Stressors on Biofuel Production in Cyanobacteria” **P60**

The effects of salt (0–6%) stress on cyanobacteria were studied to understand growth and biofuel production A 3% salt treatment for 3 days was found to be optimal for lipid accumulation, suggesting that lipid accumulation enhanced by salt stress outweighs its inhibitory effect on growth

Umeakuekwe, Godistruth, University of Arkansas - Pine Bluff, “Determining the Influence of Human Lipoproteins and Serum on Abscess Formation and Dispersal in GFP-Expressing Staphylococcus aureus U1-sarA” **P61**

During bacterial infection, human plasma contains soluble proteins that play a vital role in defending the host against invading bacteria Also contained are antibodies that bind to the bacterial cell wall, creating a complex that causes phagocytosis and allows for immune cells to engulf and destroy the invading bacteria Staphylococcus aureus, a Gram-positive pathobiont commonly found on skin and mucosae organizes into cluster communities, which are stable (SACs), and this presents challenges for immune cell clearance These structures are formed through interactions between S aureus virulence factors that cause the release of an enzyme known as coagulase and plasma-derived host proteins, initiating fibrin deposition around the bacterial community While this structure can effectively limit pathogen dissemination, S aureus can also use abscesses to evade host immunity and limit exposure to antibiotics, all while proliferating within the abscess and priming the release of virulence factors Hence, host factors that alter abscess formation are likely to be important determinants of the initial containment of infection, whereas host factors that promote fibrin capsule stability may influence immune cell accessibility and sensitivity to antibiotic therapy The overall goal of this project is to determine whether host lipoproteins and plasma augment S aureus abscess formation and stability using a three-dimensional collagen gel matrix in vitro Based on anti-thrombosis properties of high- density lipoprotein, we predict that native human HDL will promote fibrin degradation and aid pathogen dissemination, ultimately increasing the sensitivity of S aureus to antibiotics for clearance Conversely, we anticipate that human lipoprotein-deficient serum will form more stable abscesses that permit limited dispersal and maintain antibiotic resistance

Wallace, Ashley, University of Arkansas – Monticello, “Production of Phosphorylated Recombinant Peptides” **P62**

Cells constantly respond to stimuli such as stress This is achieved by signaling pathways made up of many proteins that should interact only during specific events These interactions may be controlled through post-translational mechanisms such as protein phosphorylation Biochemically, we can study the effect of phosphorylation of a specific protein by mutating the modified amino acid to an acidic amino acid such as aspartic or glutamic acid, a phosphomimetic mutation However, a phosphorylated amino acid such as phosphoserine has a charge of negative two compared to a charge of negative one for the acidic amino acids This combined with the longer side chain length of the acidic amino acids can lead to incomplete or incorrect phenotypes in the mutants This summer, we have established an E coli expression system in the lab to insert phosphoserine residues directly into a peptide of interest We first expressed control superfolder GFP with and without a phosphoserine incorporation to get used to the expression system We are currently optimizing expression of a 27 amino acid peptide that includes the PCNA

Interacting Protein (PIP) box sequence from Tousled-like Kinase 1 (TLK1) surrounded by 5 phosphoserines. This peptide was selected because one of the major goals in the lab is to understand the biochemical regulation of TLK1-PCNA interaction. This interaction has been shown to be important for TLK1 recruitment to sites of DNA damage. Control interaction assays showed that while phosphorylated TLK1 in cells does not interact with PCNA, a phosphomimetic peptide still binds to PCNA in vitro. This suggests that the addition of phosphoserine may better represent the interactions found in cells. This technique will be used to generate phosphorylated TLK1 substrates to measure how the phosphorylations effect downstream protein interactions and better understand TLK1's role in DNA damage response.

Ward, Rylan, University of Central Arkansas, "Investigating the Impact of Peroxisomal Gene Knockout on Murine gammaherpesvirus-68 (MHV68) Replication" **P63**

Gammaherpesviruses are double-stranded DNA viruses that establish lifelong infections through alternating periods of latency and lytic reactivation. The two human gammaherpesviruses, Epstein-Barr virus and Kaposi's sarcoma-associated herpesvirus, are collectively responsible for 1–3% of global malignancies. To investigate gammaherpesvirus biology, we use murine gammaherpesvirus-68 (MHV68), a natural rodent pathogen that serves as a model for human gammaherpesvirus infection. The Manzano laboratory performed a genome-wide CRISPR/Cas9 screen to identify major cellular factors required for productive MHV68 infection, the results of which indicated the peroxisome pathway as the most essential group of genes. Peroxisomes are multifunctional organelles that are involved in processes such as lipid and plasmalogen synthesis, reactive oxygen species (ROS) metabolism, and immune signaling. We hypothesized that MHV68 exploits peroxisomal functions to promote lytic replication. To test this hypothesis, Western blot analysis (WB) was used to confirm CRISPR-mediated knockout (KO) of the peroxisomal genes Pex16 and Agps in mouse cells. Reduced protein expression confirmed successful gene KOs compared with control cells. WB of MHV68-infected cell lysates, however, showed no decrease in viral protein expression in KO cells, suggesting that loss of these peroxisomal genes does not impair progression of the lytic phase. Although the CRISPR screen indicated that peroxisomal genes impact cell survival during MHV68 challenge, WB and flow cytometry data suggest that this effect is not due to a requirement for peroxisomes in effective lytic replication. We therefore hypothesize that peroxisomes contribute to self-inflicted host cell death, perhaps as a response to virus-driven oxidative stress. We are currently testing this hypothesis by assessing the viability of KO cells during viral challenge.

Watson, Ella, University of Arkansas – Fayetteville, "Epigenetic Regulation of CDK9 in KMT2A-Rearranged Acute Myeloid Leukemia" **P64**

KMT2A-rearranged acute myeloid leukemia (KMT2A-r AML) is an aggressive leukemia subtype driven by aberrant transcriptional elongation programs. Because the Super Elongation Complex (SEC) is central to KMT2A fusion-mediated transcription, SEC-associated kinases may represent therapeutic vulnerabilities. CDK9 is a core transcriptional kinase within this regulatory axis and may be especially relevant in KMT2A-r AML. To evaluate CDK9 as a candidate target, we analyzed publicly available AML transcriptomic datasets, SEC-associated gene-expression patterns, ChIP-seq datasets, and pharmacologic inhibitor responses. CDK9 was highly expressed in KMT2A-r AML and showed positive correlation with multiple transcription-associated genes, supporting its integration within the leukemic transcriptional network. ChIP-seq analysis further demonstrated CDK9 occupancy at regulatory regions of key transcriptional genes, including MEN1, suggesting a direct regulatory connection between CDK9 and KMT2A-r AML-associated transcriptional circuitry. We next tested the functional relevance of CDK9 inhibition using the small-molecule inhibitor ISH-150. KMT2A-r AML cells showed strong sensitivity to CDK9 inhibition, with IC50 values as low as approximately 5 nM. Treatment with sub-IC50 concentrations of ISH-150 also reduced CDK9 protein abundance, consistent with effective target modulation. Collectively, these findings support CDK9 as a mechanistically relevant and pharmacologically tractable therapeutic vulnerability in KMT2A-r AML. This work provides a rationale for further investigation of CDK9-directed therapeutic strategies, including future studies in treatment-resistant KMT2A-r AML contexts.

Wheat, Jacee, Hendrix College, "Inhibition of DNA polymerase kappa to improve antiproliferative effects of temozolomide on glioblastoma cells" **P65**

Glioblastoma multiforme (GBM) is the most common and most lethal type of malignant brain tumor. Temozolomide (TMZ) is the most frequently used chemotherapy drug used in the treatment plans of GBM patients. However, GBM cells have developed resistance to TMZ due in-part to the presence of DNA polymerase kappa (pol κ). Pol κ is a translesion synthesis (TLS) enzyme that is upregulated in patients with GBM and has been proven to worsen prognosis and median survival times. It is also evident that pol κ activity is tied to TMZ resistance. We have shown that a small-molecule inhibitor of pol κ called IAG-10 sensitizes GBM-derived T98G cells to TMZ in a target-dependent manner. The major goal of my project is to determine if IAG-10 exerts similar effects on other GBM-derived cell lines. We will conduct clonogenic survival assays to measure the EC50 value for GBM cell lines treated with TMZ (+/- IAG-10). The results of these experiments will determine if an

adjunct treatment of TMZ and IAG-10 will induce cytotoxicity in GBM cells, successfully resensitizing the cells to TMZ

Wilhite, Jadyn, Harding University, "CD28-Mediated Regulation of PCK2 Expression and T Cell Metabolic Adaptation" **P66**

Clinical application of T cell-based adoptive cell therapy has shown success in hematologic cancers such as Diffuse Large B-Cell Lymphoma Chimeric antigen receptor (CAR) T cells have limited efficacy in the solid tumor microenvironment (TME) due to stressful conditions such as hypoxia, acidosis, and competition for metabolic resources Such conditions push T cells towards a state of exhaustion characterized by the loss of cytotoxic function and upregulation of inhibitory receptors, decreasing T cell function in the TME and allowing tumor growth We aim to understand the metabolic flexibility surrounding the enzyme Phosphoenolpyruvate Carboxykinase 2 (PCK2) and its implications in T-cell biology This insight may aid in the development of novel therapeutic strategies designed to improve T-cell function despite restrictive solid TME conditions We hypothesize that CD28-driven PCK2 upregulation facilitates metabolic flexibility of T cells and supports an effective immune response against solid tumors To investigate this relationship, primary human CD8+ T cells were isolated and activated with CD3 or CD3/CD28 co-stimulation Western blotting and qPCR were utilized to show that PCK2 protein expression increased despite no significant change in PCK2 mRNA transcript levels, suggesting post-transcriptional regulation Further studies focused on the post-transcriptional regulation of PCK2 by the CD28 YMNM motif using CRISPR-Cas9-based mutagenesis PCK2 expression was decreased following pharmacologic inhibition of the PI3K/AKT/mTOR axis through CAL-101, MK-2206, and Rapamycin treatments Investigation of LARP1-mediated regulation of PCK2 translation suggests that LARP1 contributes to mTOR-dependent regulation of PCK2 protein abundance These findings identify a novel pathway of post-transcriptional regulation in T cells via the CD28-mTOR-LARP1 axis, providing insight into mechanisms that promote T-cell adaptation amidst the nutrient-restricted solid tumor microenvironment

Wolter, Rhys, Episcopal Collegiate School,"Investigation of the cellular mechanism of action for a small-molecule inhibitor of DNA polymerase kappa" **P67**

The DNA replication mechanisms in cancer cells adapt in ways that allow them to overcome DNA damage, making them a valuable target to effectively treat cancer Translesion synthesis (TLS) is a DNA damage tolerance mechanism cells use to overcome DNA replication stalling from therapeutic damages and other replication roadblocks TLS is performed by specialized DNA replication enzymes, relying on the catalytic properties of Y-family polymerases (pols) including pol kappa (Pol κ) Pol κ expression is a prognostic

indicator of shorter survival for patients diagnosed with glioblastoma and predictive of their response to temozolomide An inhibitor called IAG-10 was developed against Pol κ as a way to try and improve therapies for glioblastoma patients A key feature of Pol κ inhibition by IAG-10 was the ability of the small-molecule to prevent binding of the enzyme to DNA This summer, I set out to determine if IAG-10 functioned in cells the same way as it did with purified enzymes To evaluate this idea, we treated glioblastoma-derived T98G cells with IAG-10 (1μM, 24hr and 10μM, 1hr, or DMSO control) We fractionated cellular contents into cytosolic, nuclear soluble, and chromatin-bound proteins We then used immunoblotting to determine if IAG-10 could reduce the amount of Pol κ bound to chromatin As expected, there was no expression of Pol κ in any of the cytosolic fractions Treatment with IAG-10 increased the amount of Pol κ in the nuclear fraction over the DMSO control, and there was a reduction in the amount of Pol κ bound to chromatin, especially at the higher dose of inhibitor We plan to repeat these experiments to ensure reproducibility, also testing some longer treatment times In conclusion, our preliminary findings support the idea that IAG-10 can displace Pol κ from chromatin in glioblastoma cells

You, Kalyn, University of Arkansas - Little Rock, "Effects of a Single Lysergic Acid Diethylamide (LSD) Administration on Negative Affective Behaviors Elicited by Daily Unpredictable Mild Stress in Mice" **P68**

Cells constantly respond to stimuli such as stress This is achieved by signaling pathways made up of many proteins that should interact only during specific events These interactions may be controlled through post-translational mechanisms such as protein phosphorylation Biochemically, we can study the effect of phosphorylation of a specific protein by mutating the modified amino acid to an acidic amino acid such as aspartic or glutamic acid, a phosphomimetic mutation However, a phosphorylated amino acid such as phosphoserine has a charge of negative two compared to a charge of negative one for the acidic amino acids This combined with the longer side chain length of the acidic amino acids can lead to incomplete or incorrect phenotypes in the mutants This summer, we have established an E coli expression system in the lab to insert phosphoserine residues directly into a peptide of interest We first expressed control superfolder GFP with and without a phosphoserine incorporation to get used to the expression system We are currently optimizing expression of a 27 amino acid peptide that includes the PCNA Interacting Protein (PIP) box sequence from Tousled-like Kinase 1 (TLK1) surrounded by 5 phosphoserines This peptide was selected because one of the major goals in the lab is to understand the biochemical regulation of TLK1-PCNA interaction This interaction has been shown to be important for TLK1 recruitment to sites of DNA damage Control interaction assays showed that while phosphorylated TLK1 in cells does not interact with PCNA, a phosphomimetic peptide still binds to PCNA in vitro This suggests that the

addition of phosphoserine may better represent the interactions found in cells This technique will be used to generate phosphorylated TLK1 substrates to measure how the phosphorylations effect downstream protein interactions and better understand TLK1 s role in DNA damage response

Zhang, Youtong, Arkansas School for Mathematics, Sciences, and the Arts, "Identification of an inhibitor of HELB helicase" **P69**

Gliomas are the most common primary tumors of the brain and spinal cord Survival rates vary based on the subtype, but high expression levels of DNA helicase B (HELB) dramatically reduce overall survival in glioma patients and in mouse models of glioma Consistent with this, knockdown of HELB in glioma stem cells reduces proliferation and induces apoptosis while overexpression of HELB in neural stem cells increases cellular proliferation Thus, HELB could be a valuable therapeutic target for glioma; yet, noHELB inhibitors have been discovered Here, we test potential HELB inhibitors identified through fluorescence-based screening using gel-based DNA unwinding assays HELB activity in the presence of most compounds was similar to that of the DMSO control However,5 μM compound #24 inhibited HELB unwinding activity Additional testing is planned to determine the optimal concentration of compound #24 for inhibition of HELB activity Follow-up experiments will also determine the mechanism of inhibition This inhibitor of HELB may be further developed and tested for therapeutic efficacy in glioma

Zhao, Aidan, University of Texas at Dallas, "GPER1 Agonist G-1 Inhibits Anchorage-Independent Growth on Castration Resistant Prostate Cancer in Vitro" **P70**

Prostate cancer (PCa) is the most common cancer among American males, with an estimated 333,830 new cases in 2026 Androgen deprivation therapy (ADT) effectively reduces cancer growth initially but may progress to castration-resistant prostate cancer (CRPC) with a median overall survival of 13 2 months from onset, in which treatment options remain limited A subset of CRPC advances through epithelial-mesenchymal transition, associated with metastatic potential including anchorage independence Cells with this feature are proposed to drive metastasis and treatment resistance, contributing to lethal cancer progression In our lab, we found that the G-protein coupled estrogen receptor (GPER1) is expressed in CRPC cells and mediates suppression of cancer growth in both in vivo and in vitro models In this project, we hypothesize that G-1, a GPER1-specific agonist, could reduce malignant transformation of CRPC in both ADT-treated and untreated models We used androgen-dependent and -independent cell models (C4-2, VCaP, and enzalutamide-resistant VCaP cells) to compare the outcomes of G-1 treatment with or without ADT drugs We plated cells in soft agar supplemented with phenol-red-free, hormone-depleted medium to simulate the castrate environment Following a 21-day, biweekly treatment with ADT (abiraterone for C4-2 and enzalutamide for VCaP) and G-1, colonies were quantified for number and size using one-way ANOVA with post-hoc

comparisons We found that in C4-2 cells, G-1 alone or combined with abiraterone significantly inhibited colony number and size relative to vehicle (p<0 05) Both VCaP cell models showed no significant responses to G-1 or enzalutamide under identical conditions The selective inhibition of colony formation by G-1 in C4-2 cells, but not VCaP cells, may reflect differences in GPER1 expression across cell lines, where overexpressed GPER could cause hypersensitivity to G-1 Future work will quantify the expression of GPER1 and the androgen receptor

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