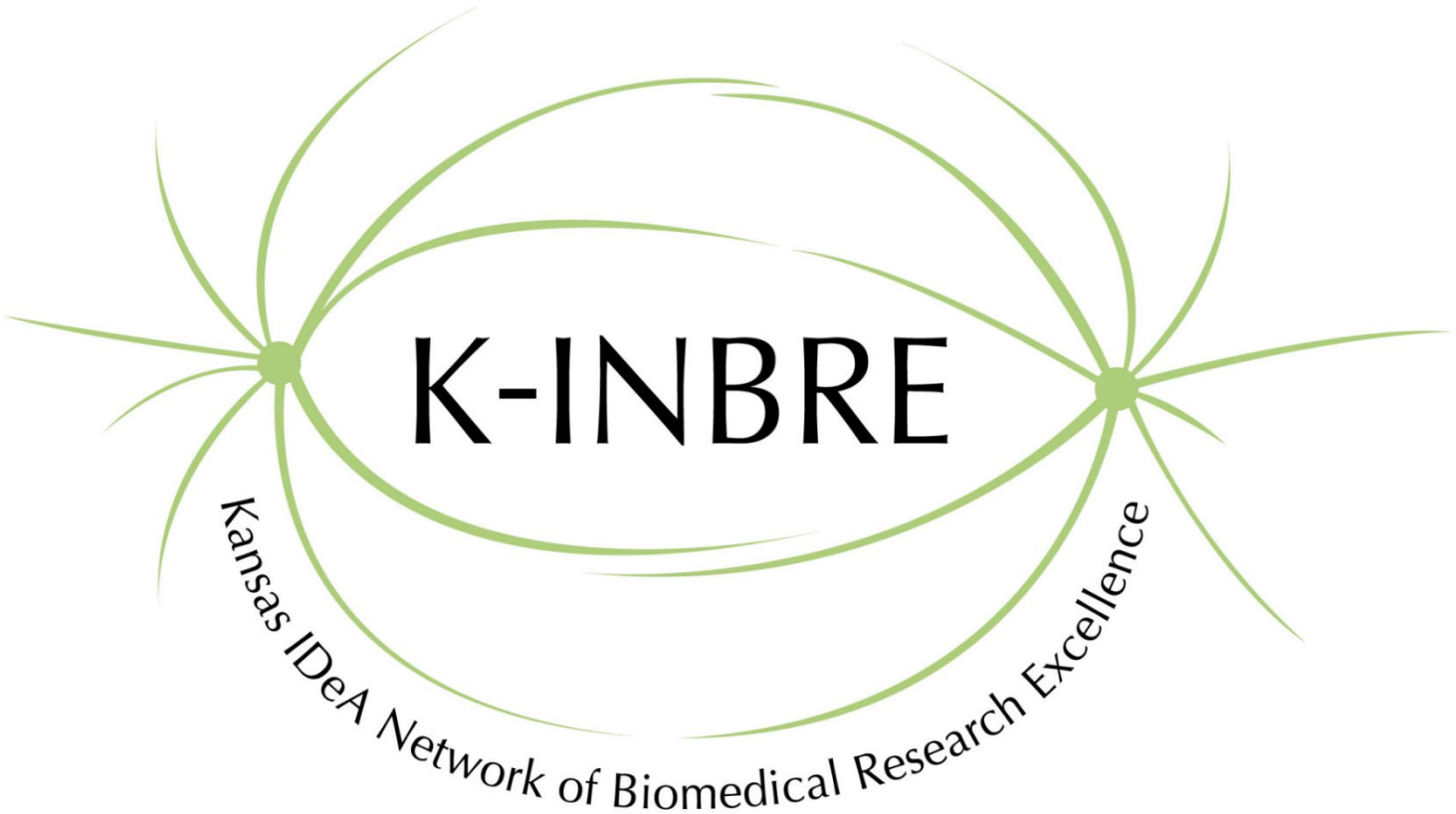
The 23rd Annual Kansas-IDeA Network of Biomedical Research Excellence Symposium



January 17-19, 2025
InterContinental Hotel on the Plaza
Kansas City, MO

This program was made possible by an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences (NIGMS) of the National Institutes of Health (NIH) under grant number P20 GM103418. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH.

Intercontinental Hotel on the Plaza
Floor Plan

LOCATION OF EVENTS:

- **Registration:** **Ballroom Foyer**
- **Friday Night Dinner:** **Rooftop Ballroom**
- **Breakfast:** **Rooftop Ballroom**
- **General Session:** **Salon 1A/1B**
- **Breaks:** **Ballroom Foyer**
- **Lunch:** **Rooftop Ballroom**
- **Poster Session/Reception:** **Salon 2/3**
- **Saturday Night Dinner:** **Salon 1A/1B**
- **Boxed Lunches:** **Ballroom Foyer**

K-INBRE 2025 Symposium
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Poster Presentations

Saturday, January 18th, 2025

(3:30-4:30 PM) Poster Session A

(4:30-5:30 PM) Poster Session B

(5:30-6:30 PM) Poster Session C

SUNDAY (10:10-11:10 AM) Poster Session D

See Poster Presentation Schedule for details

IMPORTANT:

Please ensure that all publications resulting from INBRE funds are in compliance with the NIH Public Access Policy. Future awards from NIH will be delayed until evidence of compliance has been demonstrated. For more information on the Public Access policy, please visit this link: http://publicaccess.nih.gov/policy.htm

When K-INBRE funds have supported your research, please remember to acknowledge this support by including the grant number P20 GM103418, regardless of the time period between receipt of funding and the publication or presentation.

K-INBRE 2025 Symposium
Program Schedule
Intercontinental Hotel on the Plaza
Kansas City, MO

Friday, January 17, 2025

3:00 PM	**Early Registration**	**Ballroom Foyer**
	Open Poster practice (until 6pm)	**Salon 2/3**
4:30 PM	**Early Registration Closes**	
6:00 PM	**Friday Night Dinner**	**Rooftop Ballroom**
8:00 PM	**Dinner Ends**	

Saturday, January 18, 2025

7:30 AM	**Breakfast Buffet**	**Rooftop Ballroom**
	Registration	**Ballroom Foyer**
8:30 AM	**General Session**	**Salon 1A/1B**
	Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center Opening Remarks	
9:00 AM	*Dr. Salathe, Ph.D., Vice Chancellor for Research, University of Kansas Medical Center* Welcome from University of Kansas Medical Center	
9:10 AM	*Christie Befort, Ph.D., Professor and COBRE PI, University of Kansas Medical Center* Keynote Speaker	
	Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center Moderator: First Trainee Presentation	
9:45 AM	*Lauren Apprill, Kansas State University, Manhattan KS* Title: A transcriptional repression complex that controls glucosinolate pathway gene expression in *Arabidopsis thaliana*	
10:00 AM	*Erin Blocker, Department, Emporia State University* Title: Exercise From Afar: Progressing At-Risk Rural Adults to Effective Independent Exercise for Dementia Risk Reduction	
10:25 AM	**Break**	**Ballroom Foyer**
	University Photos	**Rooftop Ballroom**
10:30 AM	University of Kansas Medical Center Photo	
10:35 AM	Emporia State University Photo	
10:40 AM	Langston University Photo	
10:45 AM	Washburn University Photo	
10:50 AM	Haskell Indian Nations University Photo	
10:55 AM	**General Session**	**Salon 1A/1B**
	Anna Zinovyeva, Ph.D., Associate Professor, Kansas State University Moderator: Trainee Presentations	
11:00 AM	*Nate Schemmel, University of Kansas, Lawrence KS* Title: Testing the ability of novel peptidomimetic-based inhibitors targeting the coronavirus main protease to inhibit coronavirus replication in cell culture	
11:15 AM	*Emma Simmons, Wichita State University, Wichita KS* Title: Developing A Wearable Fetal Heart Monitor: A Practical Evaluation of Fetal Electrocardiogram Extraction Algorithms	
11:30 AM	*Ayushee Dasgupta, Pittsburg State University, Pittsburg KS* Title: Exploring the gut microbiota of gray bats in Kansas following culturable and metagenomic approaches	
11:45 AM	**Lunch**	**Rooftop Ballroom**
1:00 PM	**Breakout Sessions**	
	Data Science Core	**Salon 1A**
	Title: "The Data Science Core presents: Make your figures beautiful and effective"	
	Curriculum-Based Undergraduate Research Experience (CURE) Panel	**Pavilion 1**
	Panelists: Dr. Steve Fields, Dr. Anu Ghosh, Dr. Eileen Hotze, Dr. Moriah Beck, and Dr. Tim Burnett (moderator)	

	Undergraduate Career Panel Panelists: Dr. Christine Brodsky (moderator), Dr. Brian Ackley, Aidyn Medina-López, Kim Wilson	**Salon 1B**
	Steering Committee Meeting By invitation only	**Pavilion 2**
2:35 PM	**Breakout sessions conclude/Break**	**Ballroom Foyer**
	University Photos	**Rooftop Ballroom**
2:45 PM	Fort Hays State University Photo	
2:50 PM	Wichita State Nations University Photo	
2:55 PM	Pittsburg State University Photo	
3:00 PM	University of Kansas Lawrence Photo	
3:05 PM	Kansas State University Photo	
3:20 PM	**Poster Judge Meeting**	**Pavilion 2**
3:30 PM	**Reception/Poster Session A**	**Salon 2/3**
4:30 PM	**Reception/Poster Session B**	**Salon 2/3**
5:30 PM	**Reception/Poster Session C**	**Salon 2/3**
6:30 PM	**Poster Sessions End**	
	Dinner	**Salon 1A/1B**
7:00 PM	**Award Presentations** *John Stanford, Ph.D., K-INBRE Associate Director, University of Kansas Medical Center* *Virginia Rider, Ph.D., K-INBRE Icon, Retired from Pittsburg State University*	**Salon 1A/1B**
7:30 PM	*Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center* Closing Remarks	

Sunday, January 19, 2025

7:30 AM	**Breakfast Buffet**	**Rooftop Ballroom**
8:30 AM	**General Session** *John Stanford, Ph.D., Associate Director, University of Kansas Medical Center* Opening Remarks *James Balthazor, Ph.D., Professor, Fort Hays State University* Moderator: Trainee Presentations	**Salon 1A/1B**
8:40 AM	*Faith Porter, Langston University, Langston OK* Title: SLE-associated risk alleles, TASL and SLC15A4, show strong genetic synergism among lupus male subjects	
8:55 AM	*Alexa Magstadt, University of Kansas, Lawrence KS* Title: Understanding the Role of Mutant KRAS in Colorectal Cancer Metastasis	
9:10 AM	*Sofia Steigner, Emporia State University, Emporia KS* Title: Quality Control of Cannabinoid Products: A Comprehensive Examination of CBD Oils	
9:25 AM	*Caleb Gillmore, University of Kansas Medical Center, Kansas City KS* Title: How APOE Genotypes Affect Mitochondrial Phenotypes In Alzheimer's Disease	
9:45 AM	**Break**	**Ballroom Foyer**
10:10 AM	**Poster Session D**	**Salon 2/3**
11:10 AM	**Poster Session D ends**	
11:15 AM	**General Session** *John Stanford, Ph.D., Associate Director, University of Kansas Medical Center* Oral Presentation Awards	**Salon 1A/1B**
11:45 AM	*John Stanford, Ph.D., Associate Director, University of Kansas Medical Center* Closing Remarks	
11:45 AM	**Boxed lunches available for pickup**	**Ballroom Foyer**

Saturday, January 18, 2025

A transcriptional repression complex that controls glucosinolate pathway gene expression in *Arabidopsis thaliana*
Lauren E. Apprill[1], Bilal Ahmad[2], and Kathrin Schrick[1,2]
[1]Department of Biochemistry and Molecular Biophysics and [2]Division of Biology, Kansas State University

Glucosinolates are sulfur-containing secondary metabolites that serve as defense compounds in *Arabidopsis* and other cruciferous plants. Relevant to human health, glucosinolate derivatives have anti-inflammatory and antioxidant properties as potential anticancer compounds. *Arabidopsis thaliana* MERISTEM LAYER1 (ATML1) is a homeodomain leucine-zipper (HD-Zip) transcription factor that drives cell differentiation in the epidermis. We found that GLABRA2 INTERACTING REPRESSOR1 (GIR1), an adapter protein first identified by interacting with GL2, physically interacts with both the ATML1 transcription factor and TOPLESS RELATED (TPR) corepressor proteins that are involved in chromatin remodeling. Mutant analysis in conjunction with yeast two-hybrid and co-immunoprecipitation assays revealed that GIR1 interacts with ATML1 through the START Adjacent Domain (STAD) of ATML1 and a putative zinc finger of GIR1. Conversely, GIR1 interacts with TPR proteins through its EAR (ethylene-responsive element-binding factor-associated amphiphilic repression) motif. The giant cell phenotype of *gir1* mutant sepals suggests its function as a negative regulator of ATML1. We hypothesize that GIR1 acts as an adaptor protein to aid in the transcriptional repression of ATML1 target genes. RNA sequencing identified several glucosinolate pathway genes including *MYB29*, a transcription factor gene required for glucosinolate production, that are upregulated in *gir1* mutants but downregulated in *atml1* mutants compared to wild type. Chemical analysis of glucosinolate levels in *gir1*, *atml1* and wild-type leaves is in progress. Discovery of a novel regulatory complex that controls glucosinolate production in plants may aid in advancing strategies for cancer prevention.

This project is supported by the Kansas INBRE (P20 GM103418) and USDA-NIFA (KS00-0009-NC1203).

Testing the ability of novel peptidomimetic-based inhibitors targeting the coronavirus main protease to inhibit coronavirus replication in cell culture
Nathaniel F. Schemmel,[1] Nathan C. Quinton,[1] Joseph O'Connor,[1] Teruna Siahaan,[2] Anthony Fehr [1]
[1]Department of Molecular Biosciences, University of Kansas, Lawrence, Kansas, USA
[2]Department of Pharmaceutical Chemistry, University of Kansas, Lawrence, Kansas, USA

Coronaviruses (CoVs) can infect humans and cause severe disease pathology. The ongoing COVID-19 pandemic demonstrates the urgent need for antiviral therapies targeting essential viral components of CoVs. One conserved and critical component of all CoVs is the main protease (Mpro), which is necessary for viral replication and polyprotein processing. Therefore, Mpro is a common target for antivirals, with some on the market, including Paxlovid. Here, we used a high-throughput viral screening assay using the model β-CoV MHV, which is in the same genus as SARS-CoV-2. Using this assay, we identified three SARS-CoV-2 Mpro inhibitors that repress MHV replication in cell culture, named 0571, 558, and 3011. Furthermore, we demonstrated using a viral titer assay that 0571 and 3011 repressed SARS-CoV-2 replication. We then defined the EC_{50} for these compounds using a viral titer assay using MHV. The EC_{50}'s for the compounds are approximately: 0571: ~11 μm, 558: ~16 μm, 3011: ~55 μm. Additionally, since Mpro is conserved across all CoVs, we assessed these inhibitors for their ability to inhibit 229E, an α-CoV that causes the common cold in humans. We found that 0571 and 558 also inhibited 229E with similar EC_{50} values as they did for MHV. We believe these are promising candidates for further drug development, with potential as a therapeutic agent for SARS-CoV-2 and related CoVs as a broad-spectrum protease inhibitor.

Developing A Wearable Fetal Heart Monitor: A Practical Evaluation of Fetal Electrocardiogram Extraction Algorithms
Emma Simmons, Yongkuk Lee
Department of Biomedical Engineering, Wichita State University
Supported by Kansas INBRE, P20GM103418

Congenital heart defects (CHDs) are the leading cause of infant mortality. Early detection of CHDs enables healthcare providers to intervene timely, reducing fetal and infant death rates. The current gold standards for fetal observation include Doppler sound and invasive fetal electrocardiogram (I-fECG). However, these techniques are either too imprecise for effective CHD detection or pose significant risks to the mother and fetus. The non-invasive fetal electrocardiogram (NI-fECG) offers a safer alternative for detecting the fetal QRS (fQRS). This technique utilizes a configuration of electrodes on the maternal abdomen to capture fetal signals. However, it also captures maternal signals and noise, making the fQRS difficult to analyze. Therefore, developing an accurate extraction algorithm is essential to enable reliable, non-invasive, and ambulatory fetal heart monitoring. In this study, we analyzed simulated and practical abdominal ECG (aECG) data. For the simulated data, an open-source database (FECGSYN) was utilized to synthesize 1 minute-long, 160 samples of aECG recordings containing fetal signals, maternal signals, and noise. For the practical data, aECG signals were collected from 20 participants for comparison. Each sample was analyzed with three main extraction algorithms (BSS, TS, and AFM) along with various electrode configurations. Average F1 values were used for statistical analysis. Our findings indicate that BSS provides the most consistent results, but low F1 values. In contrast, AFM produced higher F1 values but more variable outcomes. TS compromised with higher F1 values than BSS and more consistent results than AFM. These trends were consistent across both simulated and practical data sets.

Exploring the gut microbiota of gray bats in Kansas following culturable and metagenomic approaches
Ayushee Dasgupta, Bobbi Monroe, Andrew George, and Anuradha Ghosh
Biology Department, Pittsburg State University, Pittsburg, KS

Bats play essential roles in ecosystems, controlling insect populations but also acting as disease reservoirs, as highlighted by recent pandemics. This study examines bacterial diversity in the Gray Bat (*Myotis grisescens*) in Southeast Kansas. From guano samples, 32 bacterial isolates were obtained, majority was Gram-positive (65%). Sugar fermentation profiles showed 78% of isolates fermented all tested sugars, and a smaller proportion showed urea hydrolysis (21%) as well as indole production (3%). Sequencing with an Illumina miniSequencer yielded 2.9 million reads, with *Serratia*, *Achromobacter*, *Lysinibacillus*, and *Bacillus* as the most abundant genera. Ongoing research aims to characterize the gut microbiota of male and female bats. Beta diversity analysis showed 68% of variance, indicating greater intra-variability in females. Alpha diversity (Chao1 and Shannon indices) indicated comparable species richness, with slightly higher diversity in females. Identifying bacteria associated with bats supports disease prevention and bat conservation efforts.

Sunday, January 14, 2024

SLE-associated risk alleles, TASL and SLC15A4, show strong genetic synergism among lupus male subjects.
Faith Porter and R. Hal Scofield, MD.
Oklahoma Medical Research Foundation

SLE is a systemic autoimmune disease that predominately affects women, with a female-to-male ratio of approximately 10:1. SLC15A4, TLR7, and TASL are known SLE risk genes that play a role in TLR7-signaling IFN production which is related to common pathogen-mediated immune responses. We predict the presence of these associated risk variants could adversely affect IFN and autoantibody production in male SLE subjects. Our goal, through genotyping, is to assess the presence of SNPs SLC15A4, TLR7, and *CXorf*21 in SLE-affected and unaffected male subjects, and measure genetic synergism. We genotyped the DNA samples of 95 male SLE subjects and 132 unaffected male subjects from the OMRF Lupus Family Registry and Repository. For each subject, We ran Taqman probes *TLR7(RS3853839)*, *SLC15A4(RS1059312) (RS1385374)(RS10847697)*, and *CXorf21(RS887369)* using the real-time polymerase chain reaction (PCR) TaqMan-based allelic discrimination assay. To assess genetic synergism between the associated SLE risk variants we will initially calculate the Synergy factor for each binary combination of the three genes in question – namely, SLC15a4xCXorf21, SCL15a4xTLR7, and CXorf21Xtlr7. We found that SLE-affected AA male subjects were at least 3 times more likely to have Cxorf21 and SLC15A4 risk alleles than healthy male controls (p-value: OR, CI). Additionally, genetic synergy calculations suggest positive gene-gene interaction in all groups of SLE subjects. These data suggest that the presence of RS887369 and RS1053912 risk variants among SLE-affect men may be important in predicting disease susceptibility.

Understanding the Role of Mutant KRAS in Colorectal Cancer Metastasis
Alexa Magstadt[1], Andrew E. Evans[1], Dan Dixon[1,2]
[1]Department of Molecular Biosciences, University of Kansas, Lawrence, KS, [2]University of Kansas Cancer Center, Kansas City, KS

In the United States, colorectal cancer (CRC) is the third-most diagnosed and second-most lethal cancer for men and women combined. When CRC is localized, patients have a predicted five-year survival rate of 90%. However, when CRC metastasizes, this predicted survival drops to 14%. The epithelial-to-mesenchymal transition (EMT) is a cellular transformation that enhances cell motility and enables metastasis. This project aims to understand the role of a common CRC mutation, Kirsten Rat Sarcoma (KRAS), in EMT reprogramming. To accomplish this, we generated a stable, KRAS G12D-transformed cell line using rat intestinal epithelial (RIE-1) cells. In our KRAS-mutants, we observed an elongated, layered phenotype suggesting EMT reprogramming had occurred. Through western blotting and functional assays, we demonstrated that KRAS-mutants differentially expressed EMT-related proteins E-cadherin and vimentin and had increased invasion and migration capabilities. We then treated KRAS-mutants with a selective KRAS-G12D inhibitor. After treatment, KRAS-mutants demonstrated WT phenotype recovery, decreased migration and invasion capabilities, and 95% and 86% recoveries of WT E-cadherin and vimentin expression, respectively. This data supports the hypothesis that KRAS mutations are implicated in EMT reprogramming and, ultimately, CRC metastasis. The next step of this project is to identify the cell signaling pathways affected by mutant KRAS that precipitate the EMT. To do so, RNA expression will be explored with next-generation RNA Sequencing and RT-qPCR. Data from this project will be used to further understanding of the cellular processes mediated by mutant KRAS that promote EMT reprogramming. This will help identify how specific KRAS inhibitors can be employed to prevent metastasis.

Quality Control of Cannabinoid Products: A Comprehensive Examination of CBD Oils
Sofia Steigner and Qiyang Zhang, Emporia State University

Cannabinoids, a diverse group of psychoactive substances, consist of over a hundred recognized compounds. Their increasing legalization for medical and recreational use leads to concerns about the accuracy of cannabinoid product labeling, especially with unregulated CBD oils in online and retail markets. Notably, the FDA has declared that CBD oils available in stores and online have not undergone full evaluation or approval, raising concerns about labeling accuracy.
Preliminary investigations have revealed discrepancies between advertised claims and actual product contents, with many CBD oil products listing only THC on their labels while actually containing undisclosed cannabinoid compounds.
This study examines commonly available CBD oils to determine if products contain undisclosed cannabinoid compounds and whether consumers are exposed to potential risks. We analyzed the concentration of both THC-H and THC-A using RP-HPLC-UV/VIS (reverse phase high-performance liquid chromatography coupled with ultraviolet/visible light detector). Commercial standards were used to determine the concentrations of THC-A and THC-H in our samples.
Although all samples claimed to contain neither THC-A nor THC-H, 80% had detectable THC-A, and 70% had detectable THC-H. While ingestion of the samples tested should not cause impairments at the recommended dosages, the presence of THC compounds, even at trace levels, raises concerns about the accuracy of product labeling and the potential for cumulative effects with regular use.

This research was supported by K-INBRE funding during the summer of 2023, the 2023-2024 academic year, and the summer of 2024.

How *APOE* Genotypes Affect Mitochondrial Phenotypes In Alzheimer's Disease
Caleb Gilmore, Colton Lysaker, Heather Wilkins PhD
Department of Alzheimer's Disease KUMC

Alzheimer's disease (AD) is the most common form of dementia, causing memory loss and behavioral changes. Genetic variation in the lipid transporter protein, apolipoprotein E (APOE), can increase (APOE E4) or decrease (APOE E2) the risk of AD, however, the mechanism is not yet fully understood. Past research has proposed the mitochondria to be a likely candidate for triggering AD progression and some studies suggest APOE E4 can affect mitochondrial function. Therefore, we investigated the effect of APOE genetic variation on mitochondria. We hypothesized that APOE E4 would lead to decreased mitochondrial health. We found that astrocytes and neurons derived from APOE E4 iPSCs had increased hydrogen peroxide production, increased mitochondrial membrane potential, and increased mitochondrial superoxide production compared to APOE E2 and APOE E3-derived cells. We also found that APOE E4 iPSC-derived astrocytes and neurons had decreased mitochondrial respiration compared to APOE E3-derived cells. Lastly, we found that APOE E4 iPSC-derived neurons had increased intercellular and mitochondrial calcium levels compared to APOE E2 and APOE E3-derived cells. We conclude that cells that possess APOE E4 alleles have decreased mitochondrial health compared to cells that possess APOE E2 or E3 alleles leading us to continue to investigate the mitochondria and its role in AD.

A-1. Added Sugars are Present in US Infant Formulas
Audrey R. Rips-Goodwin[1], Daiil Jun[1], Dr. Adrianne Griebel-Thompson[2], Dr. Kai Ling Kong[3], Dr. Tera Fazzino[1]
[1]Department Psychology, University of Kansas, [2]School of Family and Consumer Sciences, University of Idaho, [3]Department of Pediatrics, University of Missouri-Kansas City School of Medicine

A-2. Using CRISPR-mediated Gene Silencing to Observe Differences in Toxin Production and Sporulation in Clostridioides difficile
Aliyana Reed[1], Kamrul Hasan[2], Oluchi Alaribe[2], Revathi Govind[2]
[1]Langston University, Langston, Oklahoma [2]Kansas State University, Manhattan, Kansas

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Zoie P. Liska[1], John X. Bourget[1], Anna C. Brake[1], Ahmed F. Alsoudi[1], Zeeshan Asmi[1], Clara Whitaker[1], Feras Awas-Eljied[1], Joseph Neri[1], Harry Nhat Nguyen[1], Yungeong Kim[2], Kyeong-Ok Chang[2], William C. Groutas[1]*. [1]Department of Chemistry & Biochemistry, Wichita State University, Wichita, KS and [2]Department of Diagnostic Medicine & Pathobiology, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

A-4. The role of premotor cortex in spontaneous recovery following injury to primary motor cortex
Lisa S. Hild[1], Heather M. Hudson[2], David J. Guggenmos[2]
1. Department of Psychology, University of Kansas
2. Department of Physical Medicine and Rehabilitation, University of Kansas Medical Center

A-5. Reprogramming the let-7 miRNA to bypass canonical biogenesis pathways
Heather Crawshaw, Jeff Medley, and Anna Zinovyeva
Division of Biology, Kansas State University

A-6. Structural Elucidation of the Ig3 Domain of Myopalladin by NMR
Julie Tran, Asha Rankoth Arachchige, Moriah Rene Beck
Department of Chemistry and Biochemistry, Wichita State University, Wichita KS 67260

A-7. Comparison of bacterial and human phosphoglycerate dehydrogenase
Lyon, Catherine J., Ella P. Ruliffson and Kim T. Simons
School of Science and Mathematics, Emporia State University, Emporia, KS, USA

A-8. Modulation of RBP-J as a novel strategy to control latent HIV-1 in chronic kidney disease
Owen Gier, Nicole Sommer, Sagine Placide and Madhulika Sharma. Department of Internal Medicine, the Jared Grantham Kidney Institute, University of Kansas Medical Center, Kansas City, KS

A-9. Exploring the potential use of RGD peptide expressing *Salmonella typhimurium* against human Pancreatic adenocarcinoma.
Brett Gibson, Hannah Eckstein, and Phillip Harries
Department of Biology, Pittsburg State University

A-10. AlphaFold 3 In Action: Demystifying Hnrnp C-RNA Binding Specificity
Gavin Doubrava, Zachary Todd, Irene Zegar, James McAfee
Pittsburg State University Department of Chemistry
Pittsburg Kansas

A-11. Development of a Course Based Undergraduate Research Experience (CURE) for a Bacterial Infectious Disease Laboratory Course
Breanna Wuerzberger,[1] Eileen Hotze, [1] Rosana Ferreira[2]
[1]Department of Undergraduate Biology, University of Kansas, [2]Department of Molecular Biosciences, University of Kansas

A-12. Unpacking Individual Amino Acid Contributions of the RGD-motif mediating αvβ3 Integrin Function
Dil Ranaweera[1], Justin Carbone[2] and Krzysztof Kuczera,[1,2]
[1]Department of Chemistry, University of Kansas
[2]Department of Molecular Biosciences, University of Kansas

A-13. The Role of DNA Topoisomerase IIα as a Component of the Chromosomal Scaffold
Regan Krueger[1], Yoshiaki Azuma[1]
[1]The University of Kansas Department of Molecular Biosciences

A-14. Developing A Wearable Fetal Heart Monitor: A Practical Evaluation of Fetal Electrocardiogram Extraction Algorithms
Emma Simmons, Yongkuk Lee
Department of Biomedical Engineering, Wichita State University
Supported by Kansas INBRE, P20GM103418

A-15. Reintroduction of functional Adenomatous Polyposis Coli reduces human colon tumor growth in xenograft model
Carly Gagnon, Bikash Pokhrel, Kristi Neufeld
Department of Molecular Biosciences, University of Kansas, Lawrence, KS 66045

A-16. Investigation of Capillin and Its Effects Microbial Growth using *Artemisia absinthium* from Wormwood
Ziyyah Gardner[1] and Lindsay Davis, Ph.D.[2]
[1]Department of Biology and [2]Department of Chemistry
Langston University, Langston, OK 73050

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Camille Carrier, Pankaj Baral
Kansas State University, Division of Biology

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Alex (Patrick) Swider, Dr. Joanna Gress,
Emporia State University Department of Science

A-19. Role of Antipsychotic Drugs in Enhancing Lifespan and Healthspan in C. elegans
Jane Oltjen[1], Shelby Innes[1], Iryna Graham[1], Aashna Srivastava[1], Shijiao Huang[1]
Department of Biochemistry, Kansas State University

A-20. Identifying novel candidate genes contributing to differences in visceral pain sensitization between C57BL/6 sub strains.
Sebastian Meriano[1], Leena Kader[2,3], Audie Rodriguez[2,3], Kyle Baumbauer[3], and Erin E. Young[2,3,4]
[1]University of Kansas, Lawerence, Kansas, United States, [2]Department of Anesthesiology, Pain, and Perioperative Medicine, KU Medical Center, Kansas City, KS, United States, [3]Neuroscience Graduate Program, KU Medical Center, Kansas City, KS, United States, [4]Department of Cell Biology and Physiology, KU Medical Center, Kansas City, KS, United States, **Funding**: Kansas IDeA Network for Biomedical Network Research Excellence (K-INBRE)

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Tucker, Kelsie, Carlie Payne and Peter A. Chung
Department of Biology, Pittsburg State University

A-22. Do parrotfish solve the cylinder task?
Morgan Leeper and Laurent Prétôt
Department of Psychology and Counseling, Pittsburg State University

A-23. Impact of CRISPR-induced mutations on *Arabidopsis thaliana* gene APETALA3 sites AP3-3 and AP3-5
Hazel Frans[1], Tara Phelps-Durr[1]
[1]Department of Biological Sciences at Fort Hays State University

A-24. Potential efficacy of immunotoxin expressing *Salmonella typhimurium* against human cancers
Auditya Jain, and Phillip Harries
Department of Biology, Pittsburg State University

A-25. Alzheimer-mutant γ-secretase complexes stall amyloid β-peptide production
Parnian Arafi[1], Sujan Devkota[1], Emily Williams[2], Masato Maesako[2] and Michael S. Wolfe[1]*
[1]Department of Medicinal Chemistry, University of Kansas, Lawrence, KS, USA
[2]Alzheimer Research Unit, MassGeneral Institute for Neurodegenerative Disease, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

A-26. Impact of antimicrobial resistance on virulence in *Pseudomonas fluorescens*
Tiffany Chan, Kervens Accilien, Robert Unckless PhD
Department of Molecular Biosciences, University of Kansas, Lawrence, KS

A-27. Glycosylation unveiled: Exploring the structure and function of FSH hormone glycoforms
Yara Abdine, Alan R. Brown, Viktor Y. Butnev, William K. White, Jeffrey V. May and George R. Bousfield
Department of Biological Sciences, Wichita State University

A-28. Leisure Interests in Individuals with Intellectual and Developmental Disabilities
Loryn Moser[1], Laura Covert-Miller[1]
[1]Pittsburg State University, Pittsburg, Kansas

A-29. Pair bonding in prairie voles requires VTA-NAc dopamine neurons
Caroline Hybl[1], Kyle R. Gossman[1], Adrianna Kirckof[2], Camryn S. Lowe[1], and Adam S. Smith[1,2]
[1]Pharmacology and Toxicology, The University of Kansas, Lawrence, KS, [2]Neurosciences, The University of Kansas, Lawrence, KS

A-30. Using DNA repair inhibitors to increase the efficacy of chemotherapy in cervical cancer
Allison Sandoval, Sebastian Wendel, Nicholas Wallace
College of Health and Human Sciences, Kansas State University

A-31. Using eDNA to detect Craspedacusta sowerbii (freshwater jellyfish) in aquatic ecosystems
Nadia Valdez Saravia, Rachel Bowes
Emporia State University

A-32. Impact of *APOE4* and high-fat diet on skeletal muscle morphology in a mouse model of Alzheimer's disease
Chelsea N. Johnson[1,2], Colton R. Lysaker[2], Elaine C. Gast[1], Colin S. McCoin[1,3], Riley E. Kemna[2], Kelly N. Z. Fuller, Benjamin A. Kugler[1], Edziu Franczak[1], Vivien Csikos[2], Julie Allen[1], Casey S. John[2], MaryJane A. Wolf[1], Matthew E. Morris[1,3], John P. Thyfault[1,3], Heather M. Wilkins[2,4], Jill K. Morris[2,4]/Paige C. Geiger[1,3], [1]Department of Cell Biology and Physiology, University of Kansas Medical Center, Kansas City, KS, USA, [2]University of Kansas Alzheimer's Disease Center, University of Kansas Medical Center, Fairway, KS, USA, [3]University of Kansas Diabetes Institute, University of Kansas Medical Center, Kansas City, KS, USA, [4]Department of Neurology, University of Kansas Medical Center. Kansas City, KS, USA, Kansas City, KS, USA

A-33. Carminic Acid from Cochineal Insects as Color Sensor for Monitoring the Freshness of Cheese
Megan Abdilla[1], Asher Freiburger[1], Mazeyar Parvinzadeh Gashti[1,2*]
[1]Department of Chemistry, Pittsburg State University, 1701 South Broadway Street, Pittsburg, KS, 66762, USA, [2]National Institute for Materials Advancement, Pittsburg State University, Pittsburg, KS, 66762, USA

A-34. Effect of glyphosate and bacterial community structure on cellulose metabolism
Alexa McGann, Ella Rulliffson and Stephen Fields
School of Science and Mathematics, Emporia State University

A-35. Flow Cytometry Core: A Chemical Biology of Infectious Disease COBRE Core Laboratory.
Peter R. McDonald [1], Robin C. Orozco [1 2], and P. Scott Hefty [1 2].
1 *Flow Cytometry Core, Chemical Biology of Infectious Disease, The University of Kansas, Lawrence, KS, USA and* ***2*** *Department of Molecular Biosciences, The University of Kansas, Lawrence, KS, USA*

B-1. Molecular Effects of PDGFR*β* in the Gain of Function in Mice
Genesis M Dambreville[1], Jang H Kim[1,2], Lorin E Olson[1,2]
[1] Langston University: Oklahoma Medical Research Foundation.
[2] Department of Cell Biology, University of Oklahoma Health Sciences Center.

B-2. Comparison of the Thermal Ecology of Ornate Box turtles (Terrapene ornata) across two distinct populations
Kaitlyn Scott, Ally Huber, Megan Norris, Abigail Trautman, Benjamin Reed
Washburn University

B-3. Understanding the role of gut microbiota in the development visceral hypersensitivity – A literature review
Colby Riddle[1], Levi Carrico[1], Erin Young[2], Kyle Baumbauer[2], Sree Chintapalli[3], and Anuradha Ghosh[1]
[1]Biology Department, Pittsburg State University, Pittsburg, KS;
[2]Departments of Cell Biology and Physiology, and Anesthesiology, University of Kansas Medical Center, Kansas City, KS;
[3]Department of Pediatrics, University of Arkansas for Medical Sciences, Little Rock, AR

B-4. Breast Cancer's Toll on Exercise Capacity
Alauna A. Parker[1], Ramona E. Weber[2], Kiana M. Schulze[2], Timothy I. Musch[2], David C. Poole[2]
Department of Biology, Langston University, Langston, OK[1]
Department of Kinesiology, Kansas State University, Manhattan, KS[2]

B-5. A Study of the Morphological Effects of Potential Detox Hair Treatments Using Polarized Light Microscopy
Mia Wendt, Holly O'Neill, Department of Chemistry Washburn University

B-6. Controlled Release Studies of Quercetin Encapsulated in Chitosan and N-Phthaloyl Chitosan Microparticles
Caitlynn D. Tate and Alessandro F. Martins
Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762, USA

B-7. Downregulation of the nutrient-sensitive post-translational modification, O-GlcNAcylation, attenuates Autosomal Dominant Polycystic Kidney Disease and increases survival in a mouse model
Jenna E. Jurgensmeyer[1, 4], Matthew A. Kavanaugh[1, 4], Casey Blades[1,4], Darren P. Wallace[2, 4], Stephen C. Parnell[3, 4], Chad Slawson[3, 4], Pamela V. Tran[1, 4]
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B-8. *Early Overexpression of the Kinase RsbW Impacts Growth and Progeny Production of Chlamydia trachomatis*
Diego Prieto, Alexandra P. Cutter, and P. Scott Hefty
Department of Molecular Biosciences, University of Kansas

B-9. Exploring Fertility Trends of Ornate Box Turtles via Ultrasonography in Western Nebraska
Powell, Brookelynn[1], Adam Schuhmacher[2], Octave Kurth[2], Katie Brighton[1], Keetan Munsell[1], Erica Guldner[1], Benjamin Reed[1]
[1]Department of Biology, Washburn University, Topeka, Kansas, USA
[2]School of Biological Sciences, University of Nebraska-Lincoln, Lincoln, Nebraska, USA

B-10. Antioxidants and Neonicotinoids: A Further Look into the Impact of Plant Antioxidants and Cannabinoids
David A Claridge, Dr. Joanna Gress
Emporia State University, Department of Science and Mathematics

B-11. Floristic Summary of Bates County, Missouri
Mason, Rylan,[1] and Neil Snow[1]
[1]Department of Biology, Pittsburg State University

B-12. Advancing the Use of eDNA to Monitor Plant-Pollinator Communities
Anna Hovious[1], Laura Budke[1], Nadia Valdez Saravia[1], Rachel Bowes[1], Darren Rebar[1]
[1]School of Science and Mathematics, Emporia State University

B-13. Analysis of antimicrobial activity of phytochemicals in *S. officinalis*
James Sweger and K.J. Abraham, Dept. of Biology Langston, OK.

B-14. How APOE Genotypes Affect Mitochondrial Phenotypes In Alzheimer's Disease
Caleb Gilmore, Colton Lysaker, Heather Wilkins PhD
Department of Alzheimer's Disease KUMC

B-15. Modulating T-Cell Immune Responses via Keap1-Nrf2 Pathway Regulation Using PROTACs
Gary Tran[1], Dr. Nadine Santana-Magal[1], Emily Burt[1], Aprajita Tripathi[1], Debolina Asgupta[1], Dr. Stefan H. Bossmann[1], Dr. Kalyani Pyaram[1]
[1]Department of Cancer Biology, University of Kansas Medical Center

B-16. The impact of thyroid hormone on microglia morphology
Alana Garcia[1], Matthew Zupan[2], Meredith Hartley[1]
[1-2]Department of Chemistry, University of Kansas, Lawrence, Kansas

B-17. Muscle Fiber Types and Cross-Sectional Area of Intrinsic Rat Tongue Muscles as a Function of Age and Intrinsic Aerobic Capacity
Paige R. Morefield[1], Elaine C. Gast[1], Lauren G. Koch[2], Steven L. Britton[3], John A. Stanford[1]
[1]Department of Cell Biology and Physiology, University of Kansas School of Medicine
[2]Department of Physiology and Pharmacology, The University of Toledo
[3]Department of Anesthesiology, University of Michigan

B-18. Cystic Cestodes: Utilizing qPCR for Species Identification of Parasitic tapeworms in sheep and cattle
Jordan L. Jackson[1], Amanda L. Roth[2], Theresa
A. Quintana[2,3], Jeba R. J.Jesudoss Chelladurai[2,3]
[1] Langston University, Langston, OK; [2] Kansas State University, Manhattan, KS; [3] Auburn University, Auburn, AL

B-19. Transcriptomic Analysis of EGFR and Downstream Pathway Expression in MCF7 and Healthy Breast Epithelium
Sara Akhtar[1], Christopher Ward[1]
1-Pittsburg State University

B-20. Identifying Factors that Regulate miRNA Strand Selection in *Caenorhabditis elegans*
Jensen Brull, Jeff Medley and Anna Zinovyeva
Division of Biology, Kansas State University. Manhattan, KS

B-21. Optimizing *C. elegans* for high throughput chemical screening
Ariana Siddique[1], Dr. Lisa Timmons[1]
[1]Department of Molecular Biosciences, The University of Kansas, Lawrence, Kansas

B-22. CDKs-1 and -2 Enhance HSV-1 Immediate-Early Gene Expression
Drew Honeycutt[1], Maxim Rodzkin[1], and David Davido[1]
[1] Department of Molecular Biosciences, University of Kansas, Lawrence, Kansas, USA

B-23. What's Linker Have to Do With It? Examining the Structure and Stability of Palladin's Ig3-4 Linker Region
Lauren Hughes, Rachel Sargent, Nathan Ta, Colby Bradford, Dr. Moriah Beck
Department of Chemistry and Biochemistry, Wichita State University

B-24. Comparing the efficacy of hand brushes versus electric brushes on teeth and evaluating the whitening power of two different kinds of toothpaste.
Junqiao An, Qiyang Zhang Department of Physical Science.
University: Emporia State University.
Department: Physical Science.

B-25. Role of Beta-2 Adrenergic Receptors in Innate Immune Response to Intracellular Bacterial Infection.
Abigail Judd[1], Prabhu R. Joshi[1], and Pankaj Baral[1]
[1]Division of Biology, Kansas State University

B-26. RNA Interference of TOR1A, CREB3, HSP90B1, and HSA1L genes in *Acyrthosiphon pisum*
Braden Allphin, Griffin Davies, James Balthazor
Department of Chemistry, Fort Hays State University

B-27. Characterization of *AT5G16120*: A Putative Monoacylglycerol Lipase
America Zarate[1,2], Zolian S. Zoong Lwe[1,3], and Ruth Welti[1,2]
[1]Division of Biology, [2]Kansas Lipidomics Research Center, [3]Department of Biochemistry & Molecular Biophysics, Kansas State University, Manhattan, KS 66506

B-28. Carbon sequestration across ecological and urban gradients in Kansas
Wedman, Clarissa,[1] and Christine C. Brodsky[1]
[1]Department of Biology, Pittsburg State University

B-29. Developmental origins of aberrant neurological trajectories in Down syndrome
Katherine I. McCarthy[1], Lexe West[1], Abhik Saha[1], Greta Foye[1], Keith P. Smith[2], Heather M. Wilkins[2], and Katherine A. Waugh[1]
Departments of Cell Biology and Physiology[1] and Neurology[2], University of Kansas Medical Center, Kansas City, KS 66160

B-30. Gene expression during the development of plastic somatic cells in the volvocine green algae
Lidia S. Lopez Vazquez[1] , Dinah R. Davison[1] , Bradley J.S.C. Olson[1]
[1]Division of Biology, Kansas State University, Manhattan, KS

B-31. Zinc Hydroxide/Biotin/Gelatin Composite Particles Take Aim at Human Breast Cancer Cells
Noah Freiburger[1], Sebastian Henry[3], Mazeyar Parvinzadeh Gashti[1,2*], Chris Ward[3], Anuradha Ghosh[3], Alessandro F. Martins[1,2]
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2- National Institute for Materials Advancement, Pittsburg State University, Pittsburg, KS, 66762, USA
3- Department of Biology, Pittsburg State University, 1701 South Broadway Street, Pittsburg, KS, 66762, USA

B-32. Metabolites derived from bacterial isolates of the human skin microbiome inhibit *Staphylococcus aureus* biofilm formation
Viet Hoang Le, Breanna Wuerzberger, Olivia R. Bauer, Eileen M. Hotze, and Rosana B. R. Ferreira.
Department of Molecular Biosciences, The University of Kansas, Lawrence, Kansas, USA

B-33. Study of ASF1a Protein's Biological Significance in Arabidopsis thaliana via Phenotypic Manifestations of CRISPR/CAS Induced A. thaliana ASF1a 1081 Gene Mutagenesis
Yue Fan1, Tara Phelps-Durr2
1 Biology Department, Kansas Academy of Mathematics and Science/Academy of Mathematics and Science and Fort Hays State University, 600 Park Street, Hays, Kansas, United States, 67601
2 Biology Department, Fort Hays State University, 600 Park Street, Hays, Kansas, United States, 67601

B-34. Synthesis of Sterically Hindered Catechol Ligands for Hydrophobic Anti-Cancer Vanadium Complexes
Colson Browning[1], Hannah Nimmo[1], Levi Ausherman[1], Maggi Braasch-Turi*[1]
[1]Department of Chemistry, Fort Hays State University, Hays, KS.
*Corresponding Author: mmbraaschturi@fhsu.edu

B-35. The Synthetic Chemical Biology Core (SCB): A Resource for Research in Chemical Biology
Chamani Perera

C-1. Unveiling the Architecture of hnRNP C Proteins Through AI-Powered Prediction
Zachary Todd, Gavin Doubrava, Irene Zegar, and James McAfee
Pittsburg State University, Department of Chemistry, Pittsburg, Kansas

C-2. Exercise-Induced Lactate as an Alternative Energy Source in Alzheimer's Disease
Carter Stanley and Stephanie Hall
Department of Anatomy and Physiology, Kansas State University, Manhattan, KS

C-3. The Link Between Cardiovascular Function and Life-Threatening Diseases: A model to understand cardiac dysfunction
McKinley D Reagor[1], Ramona E Weber[2], Kiana M Schulze[2], Timothy I Musch[2], David C. Poole[2], and Alauna A Parker[2]
[1]School of Arts and Sciences, Langston University
[2]Department of Anatomy and Physiology, Kansas State University

C-4. Impact of Microgravity on Ovarian Follicle Counts in Mice
Lauren Higgins, Cassandra Juran, Eduardo A.C. Almeida, V. Praveen Chakravarthi, Lane K. Christenson
Department of Cell Biology and Physiology, University of Kansas Medical Center, Kansas City, Kansas
Department of Human Factors and Behavioral Neurobiology, Embry-Riddle Aeronautical University, Daytona Beach, Florida
Space Biosciences Research Branch, NASA Ames Research Center, Mountain View, California

C-5. CRISPR Analysis of HIRA in Plants
Kelly Chen, Claire Shippy, and Tara Phelps-Durr
Fort Hays State University, Department of Biological Sciences

C-6. Transcriptomic Analysis of EGFR and Downstream Pathway Expression in A549 and Healthy Lung Epithelium
Sebastian Henry[1], Christopher Ward[1]
[1]-Pittsburg State University

C-7. Patterns of brain Ferritin expression in the Drosophila divalent cation transporter mutant *Malvolio*.
Breanna Leach[2*], Prabriti Neupane[1*], Mary Short[1], Jai Scarboro[1], and Rajprasad Loganathan[1,2]
1) Department of Biological Sciences and 2) Department of Biomedical Engineering. Wichita State University, KS 67260.
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C-8. Passive rates of heating and cooling in a paper wasp from Mediterranean scrub habitat
Edgar Nickols[1], Ashley Lee[2], Kiara Lopez[3], Brendon Blake[4], John M. Hranitz[5], John F. Barthell[6]
[1]Haskell Indian Nations University, Lawrence KS, [2]Loyola Marymount University, Los Angeles CA, [3]University of Central Florida, Orlando, [4]Oklahoma State University, Stillwater, [5]Commonwealth University of Pennsylvania, Bloomsburg, [6]University of Central Oklahoma, Edmond

C-9. Investigating the Molecular Function of the *Arabidopsis* AS2 Gene
Cadee Haugsness[1], Tara Phelps-Durr[2]
1-Kansas Academy of Mathematics and Science 2-Fort Hays State University Department of Biological Science

C-10/ The functional regulation of protein-based nanofiber bioscaffolds on human astrocyte for neural regeneration
Karen Bustamante-Fuchs, Kayla Cantu, Li Yao
Department of Biological Sciences, Wichita State University

C-11. Using FRET to Assess Conjugate Binding of Anthrax Toxin's PA and Antigen Spy0469
Mark White and James G. Bann
Department of Chemistry and Biochemistry, Wichita State University, Wichita, KS

C-12. Variance of Cancer Mortality in the United States
Aiyanna Davis, Trendon Edwards, and Sharon Lewis
Langston University, Chemistry Department

C-13. Understanding the Role of Mutant KRAS in Colorectal Cancer Metastasis
Alexa Magstadt[1], Andrew E. Evans[1], Dan Dixon[1,2]
[1]Department of Molecular Biosciences, University of Kansas, Lawrence, KS, [2]University of Kansas Cancer Center, Kansas City, KS

C-14. Analysis of Ibuprofen and Aspirin in Artificial Urine samples Using RP-HPLC-UV/Vis with A New QuEChERS Approach
Sofia Steigner & Qiyang Zhang, Emporia State University

C-15. Mechanisms of quorum sensing-dependent inter-species communication in the soil bacterium *Chromobacterium subtsugae*
Tate Nicholson (University of Kansas), Eryk Yarkosky (University of Kansas), Dr. Josephine Chandler (University of Kansas)
Department of Molecular Bioscience at the University of Kansas

C-16. Role of asparagine synthetase and other transcription factor targets in epidermal morphogenesis
Jillian K. Rockley, Bibek Subedi, Bradley J.S.C. Olson, Aytug Ulutas and Kathrin Schrick
Division of Biology, Kansas State University

C-17. Structure Guided Design of Broad-Spectrum Inhibitors of Coronavirus 3CL Proteases
Zoie P. Liska[1], John X. Bourget[1], Anna C. Brake[1], Ahmed F. Alsoudi[1], Zeeshan Asmi[1], Clara Whitaker[1], Feras Awas-Eljied[1], Joseph Neri[1], Harry Nhat Nguyen[1], Yungeong Kim[2], Kyeong-Ok Chang[2], William C. Groutas[1]*. [1]Department of Chemistry & Biochemistry, Wichita State University, Wichita, KS and [2]Department of Diagnostic Medicine & Pathobiology, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

C-18. Do cultured human astrocytes secrete thrombospondin-2 protein?
Greeves, Emmalyn M.[1], Kumari, M.[2]
1 Department of Biology, Kansas State University, Manhattan, KS 66506,
2 Department of Anatomy and Physiology, Kansas State University, Manhattan, KS 66506

C-19. GPNMB and SPP1 Activation in Macrophages: Implications for Pulmonary Fibrosis Pathobiology
Vincent Vo,[1] Chandrashekhar Prasad,[2] Nicolas Steele[3], Santhosh Kumar Duraisamy, [2] and Isaac Kirubakaran Sundar [2]
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[2]Department of Internal Medicine, Division of Pulmonary, Critical Care and Sleep Medicine, University of Kansas Medical Center, Kansas City, KS, USA
[3]Kansas City University, College of Osteopathic Medicine, Kansas City, MO, USA

C-20. Synthesis of Porphyrins with β-azo Linkage to Other Conjugated Systems
Shanafelt, Elizabeth, Price Kramer, and Sam Leung
Department of Chemistry, Washburn University, Topeka, KS

C-21. Preclinical evaluation of carnosic acid (CA) and rosemary extract standardized to 40% carnosic acid (RE) for the prevention of breast cancer
Ava Gartelos[1], Yan Hong[1], Darlene Limback[1], Aditi Rastogi[1], Aryamitra Banerjee[2], Brandon L. Plattner[3], Matthew Lindeblad[2], Alexander V. Lyubimov[2], Tim Fields[1], Seema A. Khan[4], Altaf Mohammed[5], Jeremy J Johnson[6], Michael J. Hageman[7], Mei Feng[7], Devin C. Koestler[8], Fariba Behbod[1*].
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C-22. Cutaneous human papillomavirus E6 impairs the cGAS-STING pathway.
Emily Tolbert, Dalton Dacus, Rose Pollina, Nicholas A. Wallace
College of Health and Human Sciences, Kansas State University

C-23. Activated Maple Carbon as a Bio-Based Cathodic Material in Lithium-Sulfur Batteries for Electrochemical Energy Storage Applications
Alexandra Robinson[2], Anjali Gupta[2], Wang Lin[1] and Ram K. Gupta[1,2*]
[1]National Institute of Material Advancement, Pittsburg, KS 66762, USA
[2]Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762, USA

C-24. Trail camera monitoring and GIS applications to investigate species distribution and habitat use in the Haskell Wetlands
Shawn Bird, Dr. Bridgett Chapin, Aysa Benally. Department of Natural and Environmental Sciences, Haskell Indian Nations University. Lawrence, Kansas.

C-25. The Minor Allele of *Ptpn22* Changes Pro-Inflammatory Cytokine Production in Dendritic Cells
Jenna R. Barnes, Anam Shaikh, Alec Bevis, Tammy Cockerham, Nancy Schwarting, Robin C. Orozco
Department of Molecular Biosciences, University of Kansas, Lawrence, KS

C-26. Qualitative Analysis of Hair Samples Treated with Over-the-Counter Hair Products using GC/MS
Gaea Gratiae Tradio, Holly O'Neill
Washburn University Chemistry Department

C-27. Interdisciplinary and Comprehensive Evaluations to Increase Service Access for Children Impacted by Autism Spectrum disorder
[1]Halle Panter [1]Paige Boydston [1]Department of Psychology and Counseling Pittsburg State University

C-28. The Impact of Dry Cupping on Pain Relief and Distal Circulation, Along the Meridian Chain.
Zane H. Busick,[1] Erin Blocker [1]
[1]Health and Human Performance Department of Emporia State University

C-29. Purification and characterization of stimulators of programed cell death protein 1 (PD-1) and granzyme B expression in T Lymphocytes.
Caden Blake[1], Sarah DeVader[1], Susumu Ishiguro[1], Brian Geisbreght[2], Chao An[3], Ping Li[3], and Masaaki Tamura[1]
1, Department of Anatomy and Physiology, Kansas State University College of Veterinary Medicine, Manhattan, KS 66506
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C-30. The Function of Human CLPB in Apoptosis of Breast Cancer Cells
Mia Thompson[1], Anna Zolkiewska[1]
[1]Department of Biochemistry, Kansas State University, Manhattan, Kansas

C-31. Role of Mitochondrial Protein CLPB in Apoptosis of Breast Cancer Cell Lines
Lexi Ziolo[1], Anna Zolkiewska[1]
Department of Biochemistry, Kansas State University, Manhattan, Kansas

C-32. Characterization of a Novel Peptide Computationally Designed to Inhibit the CD80/CD86 and CTLA-4 Interaction in T Lymphocytes
Logan Glover, Sarah DeVader, Susumu Ishiguro, Geraldine Magnin, Jeffery Comer, and Masaaki Tamura
Department of Anatomy and Physiology, Kansas State University College of Veterinary
Medicine, Manhattan, KS 66506

C-33. The structural landscape of pharmaceutically relevant 5-amino-1H-1,2,4-triazole building blocks and co-crystals thereof
Marrissa A. Raynesford,[1] Boris B. Averkiev, [1] Christer B. Aakeröy[1]
[1]Department of Chemistry, Kansas State University, Manhattan, KS 66506, USA
1. O'Sullivan, A., Long, B., Verma, V., Ryan, K. M., & Padrela, L. (2022). Solid-state and particle size control of pharmaceutical co-crystals using atomization-based techniques using atomization-based techniques. International Journal of Pharmaceutics, 621(0378–5173), 121798. https://doi.org/10.1016/j.ijpharm.2022.121798

C-34. Next Generation Sequencing at KU Genome Sequencing Core
Hackett, Jennifer L.[1,2,3], Kristen M. Cloud-Richardson[1,2,3], Erik A. Lundquist[1,2,3], Susan M. Lunte[1,4,5]
[1]Center for Molecular Analysis of Disease Pathways, [2]Genome Sequencing Core, [3]Department of Molecular Biosciences, [4]Department of Chemistry, [5]Department of Pharmaceutical Chemistry, University of Kansas, Lawrence KS, USA

C-35. University of Kansas Nanofabrication Facility: Equipment and Services
Ryan Grigsby[1] and Susan M. Lunte[1,2,3,4]
[1]The Center for Molecular Analysis of Disease Pathways, [2]The Ralph N. Adams Institute for Bioanalytical Chemistry, [3]Department of Pharmaceutical Chemistry, [4]Department of Chemistry, University of Kansas

C-36. RpoN-dependent phosphotransferase systems in *Enterococcus faecalis*
Tolulope I. Ade, Christian D. Decker and Lynn E. Hancock
Department of Molecular Biosciences, University of Kansas, Lawrence, KS

D-1. The Computational Chemical Biology Core: A Chemical Biology of Infectious Disease COBRE Core Laboratory
David K. Johnson[1,2] (dkjohnson@ku.edu)
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D-2. Assessing depth and evenness of sequencing coverage across hundreds of archived plastid genomes
Michael Gruenstaeudl[1] and Gregory Smith[2]
[1]Department of Biological Sciences, Fort Hays State University; [2]Department of Computer Science, Fort Hays State University

D-3. DNA barcoding of bloom-forming cyanobacteria in western Kansas.
Louisa Acquah, Dr. Michael Gruenstaeudl
Department of Biological Sciences, Fort Hays State University

D-4. Development of PROTAC-based Cellular Probe to Protein N-Terminal Methylation
Chao An, [1] Wei Wu,[1] Ping Li[1]
[1] Department of Chemistry, Kansas State University, Manhattan, Kansas, 66506, U.S.A

D-5. The Autoimmunity-Associated Minor Allele of *PTPN22* Enhances Innate Antiviral Immunity During Coronavirus Infection
Alec M. Bevis[1,2], Catherine Kerr[1,2], Kate Rosa[1], Tammy Cockerham[1], Nancy Schwarting[1], Anthony R. Fehr[1], Robin C. Orozco[1].
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D-6. Molecular target for the tamoxifen-refractory breast cancers
Bretches, Morgan[1], Riley Drees[2], Shang-You Yang[1,2]
[1]Department of Orthopedic Surgery, University of Kansas School of Medicine – Wichita, [2]Department of Biological Sciences, Wichita State University

D-7. Extracellular Vesicles (EVs) as a Prognostic Biomarker for Molecular Therapy Targeting RNA-binding Protein HuR in Cancer Immunotherapy
Alfred Buabeng, Sunghae Kim, Qi Zhang, Xiaoqing Wu, and Liang Xu.
University of Kansas/ Department of Molecular Biosciences

D-8. FINDING A GUT-PAINCONNECTION IN NEUROGENIC BOWEL PAIN AND DISORDERS
Sonali Choudhury[1], Adam Willits[1], Audie Rodriguez[1], Leena Kader[1] and Erin Young[1]. [1]University of Kansas Medical Center, Department of Anesthesiology, Kansas City, KS.

D-9. Analyzing Common Molecular Targets in Diabetic Brain and Kidneys
Henrietta Ehirim, Nicole Sommer, Sagine Placide, Aditi Gupta and Madhulika Sharma. Department of Internal Medicine, the Jared Grantham Kidney Institute, University of Kansas Medical Center, Kansas City, KS

D-10/ Pleiotropic Prioritization: Unraveling Shared Genetic Threads in Insomnia and Chronic Pain Through an Advanced Gene Prioritization Pipeline
Morgan A. Ewald,[1,2,3] Olivia J. Veatch [2] and Erin E. Young [1]
[1]Department of Anesthesiology and Perioperative Medicine, [2]Department of Psychiatry, [3]Neuroscience Program, University of Kansas Medical Center

D-11. *Caenorhabditis elegans* models of neurodevelopmental disorder-associated *AGO1/AGO2* variants
Belén Gaete Humada[1] and Anna Zinovyeva[1]
[1] Kansas State University, Manhattan, KS, United States

D-12. RNA binding protein HRPK-1 coordinates with miRNAs to regulate *C. elegans* development
Mika Ghosh[1], Li Li[1], Anna Zinovyeva[1]
[1]Division of Biology, Kansas State University

D-13. Evolutionary history of two X chromosome meiotic drivers in *Drosophila affinis*
Gupta, Anjali[1]* and Unckless, Robert L.[2]
[1]Department of Ecology and Evolutionary Biology, University of Kansas, Lawrence, KS
[2]Department of Molecular Biosciences, University of Kansas, Lawrence, KS
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D-14. Nuclear APC Maintains Colon Homeostasis and Mitigates Inflammation
Anika James, Kristi Neufeld
University of Kansas, Lawrence, KS, USA

D-15. Advanced Insights into Non-Adiabatic Dynamics and Ring-Opening Mechanisms of Oxazole and Isoxazole
Paul Javed[1], Briony Downes-ward[3], Arthur G. Suits[3], Daniel Rolles[2], Christine M. Aikens[1]
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[3]Department of Chemistry, University of Missouri, Columbia, MO 65211, USA

D-16. Platform generation for precisely monitoring the targeted degradation of endogenous NTMT1
Sahadev Khadka, Wei Wu, Ping Li
Department of Chemistry, Kansas State University, Manhattan, Kansas, 66506

D-17. SPECC1L C-terminal truncation results in behavioral differences and cerebellar Purkinje cell loss
Michael Kuehn[1], Jeremy Goering[1], Luke Wenger[1], Yomna Badawi[1], Marta Stetsiv[1], Preethi Kunchala[1], An Tran[1], Brittany Martinez[1], Dana Thalman[1], Iman Dilower[1], Olivia Veatch[1,3], Hiroshi Nishimune[1,4], Irfan Saadi[1,2]
[1]Department of Cell Biology and Physiology, [2]Institute for Reproductive and Developmental Sciences, [3]Department of Psychiatry and Behavioral Sciences, University of Kansas Medical Center, Kansas City, KS. [4]Tokyo Metropolitan Hospital, Tokyo, Japan.

D-18. Integrating Machine Learning-Assisted Protein Design and Analysis into Rosetta
Samuel Lim[1], Joanna Slusky, PhD[1,2]
[1]Center for Computational Biology, University of Kansas, Lawrence, KS 66047
[2]Department of Molecular Biosciences, University of Kansas, Lawrence, KS 66045

D-19. Dopamine signaling modulates partner-seeking behavior in the socially monogamous prairie vole (*Microtus ochrogaster)* during social loss.
Lowe, Camryn S.[1], Vitale, Erika M.[1], Ahad, Nicole T.[2], Guillen Sauceda, Nicolle[3], Smith, Adam S.[1,4]
[1]Department of Pharmacology and Toxicology, School of Pharmacy, [2]Department of Biology, College of Liberal Arts & Sciences, [3]Department of Psychology, College of Liberal Arts & Sciences, [4]Program in Neuroscience, University of Kansas, Lawrence, KS, USA

D-20. FORCE: Feature-Oriented Representation with Clustering and Explanation
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D-21. The three-component signal transduction system YesLMN of *Enterococcus faecalis* senses host glycans to activate expression of an ABC transporter required for host glycan import
Abdulrahman M. Naeem, Janie B. Rainer, Tolulope I. Ade, Zakria H. Abdullahi, and Lynn E. Hancock
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D-22. A Novel Cancer Therapeutic Treatment: Up-Regulating the cGAS-STING Pathway via HuR Inhibition in Prostate Cancer
Ngoc Huan Nguyen[1], Sunghae Kim[1], Xiaoqing Wu[1], and Liang Xu[1]
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D-23. Unlocking the Gut-Brain Connection: Targeting the Microbiome to Relieve Visceral Hypersensitivity and Restore Function in Irritable Bowel Syndrome
Audie Rodriguez[1,2], Leena Kader[1,2], Adam Willits[5], Sonali Chodhury[1,2], Sebastian Meriano[2], Ashleen Toor[2], Julie A. Christianson[2,3], Kyle Baumbauer[2,3], Sree V. Chintapalli[7], Anuradha Ghosh[6], Erin. E. Young[1,2,3]
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D-24. THE ROLE OF TUMOUR MICROENVIRONMENT(TME) IN GLIOBLASTOMA THERAPEUTIC RESISTANCE
Kwesi E. Taylor, Dr. Ifelayo Adefuye
Department of Biological Sciences, Fort Hays State University

D-25. Loss of *Specc1l* Disrupts the Development of the Blood-CSF Barrier resulting in Embryonic Edema and Ventriculomegaly
Dana Thalman[1], Brittany M. Hufft-Martinez[1,2], An Tran[1], Jeremy Goering[1], Luke Wenger[1], Zaid Umar[1], Benjamin Kelm[1], Sarah C. Wilson[1], Marta Stetsiv[1], Timothy C. Cox[3], Irfan Saadi[1,2]
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D-26. Nrf2 regulates the activation-driven expansion of CD4⁺ T-cells by differentially modulating glucose and glutamine metabolism
Aprajita Tripathi[1], Debolina Dasgupta[1,2], Anil Pant[2,3], Ashlyn Bugbee[3], Nanda Kumar Yellapu[4], Emily Burt[1], Ben H. Y. Choi[1], Shailendra Giri[5], Viveka Nand Yadav[6] and Kalyani Pyaram[1]
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D-27. SUSTAINED EXPRESSION OF DCLK1-S PROMOTES INFLAMMATION AND TUMORIGENESIS IN THE COLON
Kafayat Yusuf[1,2], Badal C. Roy[1,2], Shrikant Anant[2], Shahid Umar[1,2]
[1]Department of Surgery, University of Kansas Medical Center
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D-28. Oxidative stress regulator NRF2 inhibits inflammatory CD4 T-cell differentiation and protects against inflammatory bowel disease progression.
Debolina Dasgupta[1], Aprajita Tripathi[1], Emily Burt[1], Nadine Santana Magal[1], Rachel Griffard[2], Nandakumar Yellapu[2], Kalyani Pyaram[1].
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D-29. How do new cell types arise?
Davison, Dinah R.[1,2], Richard E. Michod[2], Bradley J.S.C. Olson[1]
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[2]Department of Ecology and Evolutionary Biology, University of Arizona, Tucson, Arizona

D-30. *Thm1* heterozygous female mice protect against cleft palate in offspring due to uterine cytoskeletal changes and increased expression of nutrient receptors
Hufft-Martinez, Brittany M. [1,2], Tran, An [1], Goering, Jeremy P. [1], Wilson, Sarah C. [1], Thalman, Dana N. [1], Rekowski, Michaella[3], Washburn, Michael [3,4], Tran, Pamela V. [1,5], Saadi, Irfan [1,2].
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D-31. 3' Nucleotide Asymmetry Directs miRNA Strand Selection
Jeff Medley[1], Sumire Kurosu[1], Huiwu Ouyang[2], Heather Crawshaw[1], Sarah Zhang[1], Ganesh Panzade[1], Will Sydzyik[1], Joel Sydzyik[1], Mira Bhandari[1], Christopher Hammell[2] and Anna Zinovyeva[1]
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D-32. *De novo* Generation of Outer Membrane β-Barrel Sequences with Bidirectional LSTM
Daniel Montezano, Rachel Kolodny, Joanna S. G. Slusky
Computational Biology Program, University of Kansas, Lawrence, KS 66047

D-33. Optimizing Tissue Clearing of Embryonic Palatal Shelves to Visualize the Actin Cytoskeleton in 3D
An J Tran[1], Brittany M. Hufft-Martinez[1,2], Jeremy P. Goering[1], Andras Czirok[3], and Irfan Saadi[1,2]
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D-34. Infectious Disease Assay Development Core: High Throughput Screening Laboratory at the University of Kansas
Anuradha Roy, PhD
IDAD-HTS Laboratory, University of Kansas, Lawrence, KS, USA

D-35. Using Camera Trapping to examine the Effects of Wetland dissection on Vertebrate Activity and Community Structure.
Benally, Aysa,1 Bridgett Chapin1, Courtney King1, Department of Environmental Science

A-1. Added Sugars are Present in US Infant Formulas

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Objective: Added sugar consumption is contraindicated for infants < 2 years due to potential metabolic risks; however recent research shows that most U.S.-produced infant formulas contain added sugars. This study examined differences across three types of formulas in (1) the proportion of six added sugars out of total sugars, and (2) the proportion of naturally occurring lactose out of total carbohydrates.
Methods: Data were obtained from the Nutrition Data System for Research (NDSR) and represented infant formulas on the U.S market in 2022 (N=73). Formula products were categorized into three mutually exclusive types based on their marketing labels: standard (n=31), gentle (n=27), and lactose-free (n=15). Differences in the percent of total sugars from lactose, glucose, sucrose, fructose, and maltose, and the percent of total carbohydrates from naturally occurring lactose (distinguished from refined added lactose) were examined across formula types using Bayesian modeling.
Results: The median percent added sugars was high in all formulas (M=59-90%, IQR=22.0-54.3) Compared to standard formulas, gentle formulas contained less lactose (OR=0.22, 95% HDI=[0.11, 0.44], pd=99.3%) and had >2 fold higher proportional added sugar contents (ORs=2.11 to 2.57, 95% HDI > 0, pd=99.1-99.99%). Lactose-free formulas had 4-8 times higher sucrose (OR=8.92, 95% HDI=[3.86, 20.8], pd=100%) and maltose (OR=4.91, 95% HDI=[2.51, 9.56], pd=100%) relative to standard and gentle formulas (sucrose 95% CI=[-1.67, -0.26], maltose 95% CI=[-0.35, -0.04]). Gentle formulas contained less naturally occurring lactose than standard formulas (gentle OR=0.21, HDI=[0.12, 0.40], pd=100%)
Conclusions: US-produced infant formulas present a high risk for added sugar exposure among infants.

A-2. Using CRISPR-mediated Gene Silencing to Observe Differences in Toxin Production and Sporulation in Clostridioides difficile

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Clostridioides difficile is a major cause of antibiotic-associated diarrhea and severe infections in patients with compromised immune system. In *C. difficile*, spore formation and toxin production are the major factors that contribute to infection transmission, host colonization, and infection outcome. A key regulator of these processes in *C. difficile* and other bacterial pathogens is cyclic diguanylate monophosphate (cyclic-di-GMP), a second messenger moleule whose intracellular levels is tightly regulated by two classes of enzymes, diguanylate cyclases (DGCs) and phosphodiesterases (PDEs). Previous studies have shown that *C. difficile* encodes multiple cyclic-di-GMP turnover genes in its genome. However, it is not yet clear how intracellular levels of cyclic-di-GMP control *C. difficile* physiology and the exact regulatory networks involved. This study aimed at determining how intracellular levels of cyclic-di-GMP controls major physiological processes in *C. difficile.* We used an RNA-based gene editing system (CRISPRi) to knockdown the expression of two cyclic-di-GMP turnover genes (a DGC and a PDE) and investigated the effects on *C. difficile* colony and cell morphology, motility, spore formation, and biofilm formation using in *vitro assays*. Knockdown of the DGC increased sporulation while knockdown of the PDE affected cell morphology and reduced sporulation. We believe that uncovering the exact role of these genes in intracellular levels of cyclic-di-GMP in *C. difficile* could help discover novel treatment targets that will help improve patient outcome from *C. difficile* infections.

A-3. Structure Guided Design of Broad-Spectrum Inhibitors of Coronavirus 3CL Proteases

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Periodic transmissions of β-coronaviruses (SARS-CoV, MERS-CoV, and SARS-CoV-2) from animals to humans underscore the need to devise rapid response strategies that can be cost-effectively deployed to minimize the pervasive harmful effects on global health. Commonly originating from bats, zoonotic β-coronaviruses from the Merbecovirus and Sarbecovirus subgenera have shown to be particularly dangerous to humans. The evolution of new viral variants remains a significant concern, particularly the potential emergence of pathogenic strains displaying high mortality rates, like MERS-CoV (~30%). With each new virus and variant, millions of dollars have historically been invested in the development of effective treatments; however, each year a therapeutic remains in development, thousands of deaths occur. Although β-coronaviruses and their variants have shown to have similar active site topographies, a broad-spectrum antiviral treatment remains elusive, and numerous β-coronaviruses lack established therapeutics. Our research aims to use a structure-guided approach to design a broad-spectrum antiviral therapeutic and prophylactic for zoonotic β-coronaviruses that acts by inhibiting the 3CL protease, an enzyme essential for coronavirus replication. The results of preliminary studies suggest the design of such an antiviral is feasible.

A-4. The role of premotor cortex in spontaneous recovery following injury to primary motor cortex

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Following an injury to primary motor cortex (M1) there is often a varied degree of spontaneous recovery of motor function. While neuroplastic reorganization in the premotor cortex (PM) has been shown to contribute to this recovery, the critical time period for reorganization remains unknown. This study aims to determine the time period when PM contributes the most to spontaneous recovery following M1 injury. Long-Evans rats (7 male, 8 female) were trained on a skilled reach task before random assignment to five groups. Rats were then given an ischemic injury to M1 (controls received a sham injury) and PM was inactivated (using a constant infusion of muscimol, a GABA-A receptor agonist) for 0, 1, 2, or 3 weeks depending on group assignment. Behavioral reaching data was collected bi-weekly for 8 weeks post-injury to assess the impact of the duration of PM inactivation on spontaneous motor recovery. Results show rats are unable to perform the reach task while PM is inactivated regardless of whether they have an M1 injury or not. Within one week after the cessation of muscimol infusion, rats began reaching. By 8-weeks post-injury, all rats recovered to >80% of their baseline activity. These preliminary results suggest that the critical time period for PM's involvement in motor recovery after an M1 injury may be delayed beyond the three weeks that we expected. Further investigation into the correlation of behavioral recovery with anatomical reorganization may enhance our understanding of PM's role in motor recovery.

A-5. Reprogramming the let-7 miRNA to bypass canonical biogenesis pathways
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Regulation of gene expression enables diverse cellular functions, controlling the magnitude, timing and location of different gene activities. Abnormal gene expression is associated with numerous human diseases including several cancers and developmental defects. microRNAs (miRNAs) are non-coding RNAs that regulate gene expression, typically by repressing the activity of target genes. During miRNA biogenesis, miRNAs are loaded into Argonaute proteins to form a functional complex that recognizes target genes. Similar to disruptions in miRNA function, loss of Argonaute function leads to broad disruption of gene regulation. In the nematode *Caenorhabditis elegans*, small RNAs are sorted into different Argonautes based on structural features of the small RNA. For example, miRNAs are primarily associated with the Argonaute ALG-1, while other small RNAs are sorted to different Argonautes. The let-7 miRNA is absolutely required for animal development and is evolutionarily conserved across nematodes and humans. Loss of let-7 or ALG-1 function results in larval lethality due to aberrant gene regulation of let-7 target genes. We hypothesized that modifying the structure of let-7 could redirect let-7 to a different Argonaute and bypass the requirement for let-7 in *alg-1* mutants. To address this, we used CRISPR/Cas9 genome editing to introduce mutations that altered the let-7 miRNA structure without impacting the predicted targeting of downstream genes. These mutations are viable, suggesting that let-7-dependent gene regulation is not disrupted. We are currently in the process of creating *let-7 alg-1* double mutations to determine if altering the structure of let-7 bypasses the let-7-dependent phenotypes observed in *alg-1* mutants.

A-6. Structural Elucidation of the Ig3 Domain of Myopalladin by NMR
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Myopalladin (MYPN) is a recently described actin-binding protein located at the Z and I lines of striated muscle. MYPN is hypothesized to anchor to other structural proteins, such as actin, nebulin, and titin, which work together to facilitate contractile motion at the sarcomeres of muscle cells. However, its specific role in regulating the actin cytoskeleton is largely unknown, and its structure is yet to be solved. Based on sequence homology with palladin, MYPN comprises five immunoglobulin-like domains (Ig1-5). Additionally, the Beck Lab has established that the Ig3 domain is the minimum required for binding and bundling interactions with filamentous actin. The ultimate purpose of the study is to elucidate the structure of the Ig3 domain of MYPN with 2D and 3D NMR experiments. The first phase of the study consists of optimizing existing protocols for expression and purification of uniformly ^{13}C and ^{15}N-labelled MYPN Ig3 for maximum yield of high purity protein from minimal media. Purified, $^{13}C/^{15}N$- labelled protein will then be used for a series of NMR experiments on the department's new 500 MHz NMR instrument. Processing and analysis of the data will involve a significant computational effort to assign chemical shifts before structure calculations can be carried out. Future experiments will also examine six point mutations in the Ig3 domain of MYPN that have been previously associated with various types of cardiomyopathies, as the Beck Lab has examined that these mutations have limited binding and bundling interactions with actin.

A-7. Comparison of bacterial and human phosphoglycerate dehydrogenase
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In the major pathway for serine production, phosphoglycerate dehydrogenase (PHGDH) catalyzes the conversion of 3-phosphoglycerate (3PG) into 3-phosphopyruvate (PHP). PHP is then transaminated and dephosphorylated to produce serine. The PHGDH gene, when overexpressed, has been implicated in oncogenesis. *E. coli* PHGDH catalyzes the oxidation of other molecules and is feedback inhibited by serine. *H. sapiens* PHGDH is more specific for 3PG and is not feedback regulated by serine. To better understand enzyme specificity and regulation, the structure of both *E. coli* and *H. sapiens* PHGDH active sites were analyzed, and thirteen amino acid differences in the active site were identified. Site-directed mutagenesis has been successfully used to change six of these *E. coli* PHGDH positions into the *H. sapiens* version. The wild-type and mutant recombinant enzymes were purified, and enzyme activities were monitored using the cofactor NADH. To date, the substrates α-ketoglutarate and oxaloacetate were used as substitutes for 3PG. Continued mutagenesis will be used to identify the critical positions in the sequence that confer specificity with the eventual goal of understanding how the enzyme is involved in oncogenesis.

A-8. Modulation of RBP-J as a novel strategy to control latent HIV-1 in chronic kidney disease
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Antiretroviral therapy (ART) has drastically reduced HIV-1 related deaths, but HIV elimination poses a challenge due to latently infected cells. Latently infected epithelial cells (podocytes) produce viral proteins and promote inflammation. This ultimately leads to chronic kidney disease (CKD). We need strategies that act on the HIV-LTR (long terminal repeat) promoter and can either permanently kill/eliminate (shock and kill) the virus, or permanently suppress the virus (lock and block). RBP-J is a transcriptional repressor that binds on the same sites of HIV-LTR as the NFkB-p65 sites. While NFkB-p65 is a well-known trans-activator of HIV-LTR, RBP-J is poorly studied. We hypothesize that RBP-J counteracts the actions of NFkB-p65 on the HIV-LTR and deletion of RBP-J will lead to disease aggravation in a mouse model of CKD (Tg26 mice). Using breeding strategies, we generated Tg26 mice with podocyte specific deletion of RBP-J (Tg-RBP-J-Podko). Compared to Tg26, Tg-RBP-J-Podko mice displayed histological abnormalities, increased NFkB-p65, compromised renal function and inflammation. In vitro, RBP-J overexpression suppressed LTR activity while knockout/pharmacological inhibition led to massive activation. Our studies suggest that RBP-J overexpression can tightly suppress HIV-LTR and prohibit its transactivation, while RBP-J inhibition can activate the LTR to an extent that can be lethal to latent cells. Thus RBP-J may fulfill the requirements of lock and block and shock and kill strategies, respectively. We conclude that RBP-J is a strong repressor of the HIV-LTR in podocytes, which can be targeted for the benefit of HIV-CKD patients.

A-9. Exploring the potential use of RGD peptide expressing *Salmonella typhimurium* against human Pancreatic adenocarcinoma.
Brett Gibson, Hannah Eckstein, and Phillip Harries
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In an attempt to mitigate the frequent limitations of traditional radiation and chemotherapy in treatment of certain cancers, an auxotrophic strain of *Salmonella* (D2) has been engineered with shortened surface lipopolycaccharide (LPS) to allow for expression and display of arginine-glycine-aspartate (RGD) peptide on the bacteria's surface. This peptide binds to alpha v beta 3 ($\alpha v\beta 3$) integrin heterodimer which is known to be overexpressed in certain cancers. RGD-displaying *Salmonella* has been found to strongly bind to $\alpha v\beta 3$ overexpressing cancer cells and increase survival in mouse models of human breast cancer and melanoma. Despite these successes, there has been no published discussion of other potential human cancer targets for this engineered *Salmonella*. In this analysis, we utilize cancer gene expression data to show that pancreatic adenocarcinoma overexpresses both alpha v and beta 3 integrin subunits and would likely make an attractive target for this *Salmonella* based therapy. We further examine survival curves for high and low integrin expressing pancreatic cancer and look at promoter methylation as a possible explanation for existing integrin expression patterns.

A-10. AlphaFold 3 In Action: Demystifying Hnrnp C-RNA Binding Specificity
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We have used the artificial intelligence program AlphaFold2 to determine the quaternary structure of the hnRNP C family of proteins which includes the differentially spliced isoforms C1 and C2 (C protein) as well as hRaly and hRalyl. The results of that study indicated that the C proteins, hRaly, and hRalyl are structurally redundant. The C proteins are two of the most abundant proteins in the nucleus and have been reported to play critical roles in RNA splicing, RNA polyadenylation, regulation of mRNA turnover, internal ribosome entry site mediated translation, as well as being a component of the telomerase holoenzyme. There exists controversy with regard to the RNA binding properties of the hnRNP C protein primarily with regard to RNA binding specificity. The research presented here uses AlphaFold3 to investigate the RNA binding specificity of hnRNP C in its tetrameric structural context. More specifically we have compared the binding specificity and organization of short RNA oligonucleotides compared to a variety of RNA molecules that are much longer in length.

A-11. Development of a Course Based Undergraduate Research Experience (CURE) for a Bacterial Infectious Disease Laboratory Course
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Participation in scientific research has been shown to positively impact undergraduate students in STEM fields, but capacity constraints in faculty research laboratories at large universities limit access. Implementation of Course-based Undergraduate Research Experiences (CUREs) in laboratory courses is an effective strategy to give undergraduates the opportunity to conduct authentic research within a teaching laboratory. Therefore, we are transforming the curriculum of the Bacterial Infectious Disease (BID) Laboratory course at the University of Kansas into a CURE "MicrobioME: A CURE for Staphylococcus aureus". Students will determine if bacterial skin microbiome isolates can influence the pathogenic mechanisms of the human skin pathogen Staphylococcus aureus. This CURE, grounded in Dr. Rosana Ferreira's ongoing research in the Molecular Biology Department, aligns seamlessly with course objectives by introducing students to molecular and media-based techniques for identifying bacterial pathogens and exploring their disease-causing mechanisms. We implemented a piloted study of this CURE in the spring 2024 semester and identified several challenges that needed to be met prior to full implementation in the 2025 offering of the course. These barriers included identifying strains of S. aureus appropriate for BSL2 laboratory settings as well as overcoming bottlenecks in the experimental procedures. Preliminary student survey feedback reflected positive student experiences and highlighted the relevance, practical application, autonomy, and engagement of this project.

A-12. Unpacking Individual Amino Acid Contributions of the RGD-motif mediating αvβ3 Integrin Function
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Arginine-glycine-aspartate (RGD) is a highly conserved amino acid motif found in all three domains of life. This motif plays a significant role in regulating processes such as cancer progression, metastasis, and angiogenesis, acting especially through its interaction with RGD-binding integrins. The $\alpha_V\beta_3$ integrin, a member of RGD-binding integrin subfamily, binds to the RGD motif via coordination of a divalent metal ion at the Metal Ion-Dependent Adhesion Site (MIDAS), hydrogen bonding and van der Waals interactions at the interface of the α and β subunits. RGD is present in various Extracellular Matrix (ECM) proteins including fibronectin, vitronectin, and osteopontin. To help understand the molecular mechanism of RGD:$\alpha_V\beta_3$ interactions, we perform a computational docking study using the AutoDock vina program, aimed at elucidating the importance of the three individual amino acids – arginine, glycine and aspartate – to the complex formation, and thus to the underlying biological processes. Our initial results, based on alanine scanning, show a significantly greater binding contributions from arginine and aspartate than from glycine. In further work, we will explore the possibility of identifying sequences that bind $\alpha_V\beta_3$ integrin more strongly than the native RGD. Insights derived from these computer models may lead to the development of new and improved integrin-targeted cancer therapies.

A-13. The Role of DNA Topoisomerase IIα as a Component of the Chromosomal Scaffold
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DNA Topoisomerase II (Topo II) is an enzyme that functions to decatenate genomic DNA. TopoIIα is a known component of the chromosomal scaffold but its mechanism as such has yet to be elucidated. Both functions of TopoIIα require its C-terminal domain (CTD). Our group established the critical role the chromatin binding ability, within the CTD, has for TopoIIα's function in the separation of mitotic chromosomes. In this study, I investigated the role the chromatin binding ability of TopoIIα has as a component of the chromosomal scaffold.
The auxin-inducible degron (AID) system and subsequent tetracycline (Tet) inducible system is utilized to study TopoII in CRISPR/Cas9 genome-edited cell lines. These systems allow for the depletion of endogenous TopoIIα and expression of exogenous TopoIIα or the desired TopoII mutant of interest. These systems are essential in the study of TopoII as TopoIIα serves such a crucial role in mitosis that classical knockout systems cannot be used.
We have observed distinct protein pattern differences in the isolated chromosomal scaffold fraction of the TopoII mutant without its chromatin binding ability. Mass Spectroscopy analysis of the isolated chromosomal scaffold fractions identified proteins that are affected by TopoIIα binding to mitotic chromosomes. This study is ongoing to further analyze these proteins of interest.

A-14. Developing A Wearable Fetal Heart Monitor: A Practical Evaluation of Fetal Electrocardiogram Extraction Algorithms
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Supported by Kansas INBRE, P20GM103418

Congenital heart defects (CHDs) are the leading cause of infant mortality. Early detection of CHDs enables healthcare providers to intervene timely, reducing fetal and infant death rates. The current gold standards for fetal observation include Doppler sound and invasive fetal electrocardiogram (I-fECG). However, these techniques are either too imprecise for effective CHD detection or pose significant risks to the mother and fetus. The non-invasive fetal electrocardiogram (NI-fECG) offers a safer alternative for detecting the fetal QRS (fQRS). This technique utilizes a configuration of electrodes on the maternal abdomen to capture fetal signals. However, it also captures maternal signals and noise, making the fQRS difficult to analyze. Therefore, developing an accurate extraction algorithm is essential to enable reliable, non-invasive, and ambulatory fetal heart monitoring. In this study, we analyzed simulated and practical abdominal ECG (aECG) data. For the simulated data, an open-source database (FECGSYN) was utilized to synthesize 1 minute-long, 160 samples of aECG recordings containing fetal signals, maternal signals, and noise. For the practical data, aECG signals were collected from 20 participants for comparison. Each sample was analyzed with three main extraction algorithms (BSS, TS, and AFM) along with various electrode configurations. Average F1 values were used for statistical analysis. Our findings indicate that BSS provides the most consistent results, but low F1 values. In contrast, AFM produced higher F1 values but more variable outcomes. TS compromised with higher F1 values than BSS and more consistent results than AFM. These trends were consistent across both simulated and practical data sets.

A-15. Reintroduction of functional Adenomatous Polyposis Coli reduces human colon tumor growth in xenograft model
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Eighty percent of colorectal cancers exhibit mutations in the APC (adenomatous polyposis coli) gene, with APC loss of function often initiating and driving tumor formation. Prior studies used shRNA to decrease wildtype APC levels in mouse models and showed that the resulting intestinal tumor growth was reversed upon restoring wildtype APC. However, it was unclear if this effect applies to human colon cancer cells harboring additional mutations and truncated APC, which has gain-of-function properties. To address this, we used the DLD1 human colorectal cancer cell line, which expresses nonfunctional truncated APC, and inserted a doxycycline (DOX)-inducible full-length, wildtype APC gene. Initially, we found that introducing full-length APC significantly reduced DLD1 cell proliferation in vitro. Next, we assessed the effect of full-length APC expression on the tumor-forming capability of these DLD1 cells in a xenograft mouse model. DLD1 cells were injected into each flank of nude mice, which were given doxycycline (DOX) in their food. DOX-treated mice exhibited a substantial reduction in both the size and growth rate of tumors, indicating that DLD1 cell proliferation depends on the absence of functional APC protein. In another cohort, tumors were allowed to grow to ~200 mm^3 over 16 days before inducing functional APC expression. Remarkably, functional APC introduction also reduced the growth rate of pre-existing tumors, suggesting that restoring functional APC reduces tumor growth even in established tumors. Future work should explore methods to reintroduce APC as a potential therapeutic strategy for colorectal cancer.

A-16. Investigation of Capillin and Its Effects Microbial Growth using *Artemisia absinthium* from Wormwood
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The Center for Disease Control and Prevention (CDC) estimates that at least 2.8 million antimicrobial-resistant infections continue to occur in the United States each year, killing more than 35,000 people. Globally, the World Health Organization projects that these infections will cause up to 10 million deaths annually by 2050 if new antibiotics are not developed. Capillin is an organic compound known for its cytotoxic properties, which can induce cell apoptosis. It also exhibits antitumoral, antibacterial, antimicrobial, and antifungal activities. This study investigates the inhibition of bacterial growth using *Artemisia absinthium* extract from wormwood, isolating capillin as a key active component. By examining the antibacterial effects of capillin, this research contributes to the understanding of natural compounds in medicine, offering insights into anti-microbial-resistant patients through the development of novel treatments and therapeutic options. The research findings support our hypothesis that the extract displays antibacterial properties, as evident through our preliminary bacterial growth trials. Our future studies will involve using GC-MS and HPLC techniques to verify capillin purity and concentration. Furthermore, we will investigate of the mechanism of capillin in inhibiting bacterial and fungi growth.

A-17. The Effect of Age on Lung Nerve Density and Neuroimmune Interactions
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This study aims to investigate age-related changes in nerve innervation within lung tissue across five developmental stages in mice: neonatal (Day 0), juvenile (Day 14), early adolescent (Day 30), late adolescent (Day 60), and mature adult (Day 182). At each stage, mice will be humanely euthanized, and their lungs will be harvested for nerve innervation analyses by immunohistochemical method. We will employ specific nerve markers—Tuj1 (general neuronal marker), TH (sympathetic nerves), and CGRP (Sensory fibers)—alongside a macrophage marker (F4/80), combining each neuronal marker with macrophage staining overalap to map cellular interactions. Using Super Resolution Confocal Microscopy, we will analyze spatial distribution and density changes in nerves and macrophages around the lung airways and parenchyma to assess the extent of nerve innervation and neuroimmune interactions over time. The relevance of this study lies in the observed decline in immune cell abundance, but increase in nerve density, within aging lung tissue. This may be linked to altered immune functions, inflammation, or shifts in neuronal maintenance. Such age-associated changes in lung innervation could contribute to respiratory health issues commonly observed with age, such as increased susceptibility to allergic reactions, which affect approximately one-third of adults. Insights gained from this study could pave the way for targeted therapeutic approaches aimed at mitigating age-related respiratory complications by addressing neural and immune function alterations in lung tissue.

A-18. Detection of Phytocannabinoids In Hive Products and Bees
Alex (Patrick) Swider, Dr. Joanna Gress,
Emporia State University Department of Science

Agricultural pesticides can be harmful to non-target insects, including pollinating bees. When foraging bees encounter these chemicals, unknowingly bringing toxic pesticide residues back to the hive, exposing other bees in the colony. Managed honey bee hives experienced a 48% loss in 2022-2023. Exposure to pesticides, including the neonicotinoid imidacloprid, is one co-morbidity factor associated with hive loss. Exposure at sub-lethal levels can negatively impact honeybee olfactory memory and learning. Imidacloprid is also linked to gene regulation of many honeybee detoxification genes that metabolize toxic molecules or minimize their fatal effects. Industrial hemp, (Cannabis sativa L.), one of the earliest crops spun for fiber, is now used for various commercial products derived from fiber or seeds and is used as a pollen source by bees. Hemp pollen contains several pharmacologically active phytocannabinoids including CBD, CBG, and CBN that can exert beneficial effects, including antioxidant and anti-inflammatory activity. These compounds can affect the production of ROS and therefore may combat the harmful effects of these neonicotinoids. We have been investigating if foraging on hemp pollen can protect against imidacloprid stress at a whole hive level. Hives were allowed to freely forage on hemp flowers, either in the absence or presence of imidacloprid. Foragers were collected for qPCR analysis of the antioxidant pathway in the bee gut. In addition, hive sundries were collected to determine what phytocannabinoids were incorporated into the hive using HPLC. These results may lead to an additional role for hemp pollen in IPM strategies for hive survival.

A-19. Role of Antipsychotic Drugs in Enhancing Lifespan and Healthspan in C. elegans
Jane Oltjen[1], Shelby Innes[1], Iryna Graham[1], Aashna Srivastava[1], Shijiao Huang[1]
Department of Biochemistry, Kansas State University

Aging is the leading risk factor for developing neurodegenerative diseases like Alzheimer's, Parkinson's, and Huntington's disease. These diseases can cause behavioral issues, mood disorders, and psychosis, often requiring antipsychotic medications to alleviate symptoms. Interestingly, research has shown that antipsychotics enhance life longevity. We are studying olanzapine and trifluoperazine, two antipsychotics previously shown to enhance the expression of the longevity gene *fmo-2* and extend lifespan. First, we tested various concentrations of olanzapine (0 µM, 5 µM, 10 µM, 25 µM, and 50 µM) in wildtype C. elegans for lifespan extension. We determined that the 10 µM concentration showed the largest increase in lifespan extension. We then tested this concentration in Alzheimer's, Huntington's, and Parkinson's disease models. This experiment is ongoing, and the conclusion as to whether olanzapine increases lifespan of disease models will be drawn after the completion of the experiment. To explore the mechanisms of the healthspan effects of trifluoperazine, we are currently examining whether fatty acid metabolism genes are required for *fmo-2* induction and motility improvement. In the imaging experiments, we analyzed nine gene knockdown conditions (EV, fat-2, fat-5, fat-7, Ibp-5, Ibp-6, Ibp-8, acs-7, and DR as a control) using RNAis, both with and without 100 µM trifluoperazine. In the thrashing assays, we compared wildtype and Huntington's disease models, as well as control conditions against 0.1 µM trifluoperazine, using these same knockout gene conditions. We expect to uncover key genes involved in *fmo-2* induction and healthspan improvement in C. elegans.

A-20. Identifying novel candidate genes contributing to differences in visceral pain sensitization between C57BL/6 sub strains.
Sebastian Meriano[1], Leena Kader[2,3], Audie Rodriguez[2,3], Kyle Baumbauer[3], and Erin E. Young[2,3,4]
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Functional abdominal pain (FAP) can severely affect the quality of life in patients with disorders of gut brain interactions (DGBIs), such as irritable bowel syndrome (IBS). A major factor that contributes to FAP is visceral hypersensitivity (VH), which refers to increased sensitivity to mechanical stimulation of visceral organs. VH is present in up to 90% of IBS patients, yet the etiology remains completely unknown. Therefore, understanding mechanisms underlying VH can reduce visceral pain and increase quality of life. In previous data collected in our lab, researchers found differences in visceral sensitization between two sub strains of mice, C57BL/6J and C57BL/6NTac, even though they are similar mice. We know that we can study the minor differences in genes between these two groups to understand their differences in pain-related phenotypes. Researchers also performed a mini systematic review to identify all genes associated with pain, nociception, and/or hypersensitivity. From their candidate gene list, only one gene in our lab has been studied and further identified as a possible gene contributing to VH. In this study, we furthered our systematic review by creating and validating primers for other genes that were identified. We successfully identified primer sets with adequate efficiency for quantification of four candidate genes identified in genomic comparisons. Subsequent development and validation of primers for the remaining candidate genes are underway. These findings are critical to the continuation of ongoing studies but also support the need for validation and efficiency testing for newly developed primers and primers taken from prior studies.

A-21. Understanding the Mechanisms of Tumor Resistance in Murine Cell Lines
Tucker, Kelsie, Carlie Payne and Peter A. Chung
Department of Biology, Pittsburg State University

Our lab has been interested in understanding how activated macrophages discriminate between normal and tumor cells. Two SV-40 transformed murine fibroblast cell lines, F5m and F5b, have been used to investigate this discrimination. Unpublished microarray data provided by Kansas State University has shown alterations in the expression of many genes. Through the years, our lab has performed various analysis on some of the more statistically significant target genes that were identified via mRNA chip analysis (Aff3, Cbr3, PlagL1, Zfp521, Pde3b, Tesc, CD47, CD81). These genes were either upregulated or downregulated in the macrophage-sensitive cell line F5b compared to the F5m cell line. (unpublished data, collaboration with Kansas State University). Although gene expression analysis as well as Western blot analysis of some target genes have yielded some encouraging results (CD47, CBR3) show some promise, data has mostly been inconclusive. Western analysis has confirmed some solid targets based on differences in protein expression: CBR3, CD99, PTGDS, KCNN4, and CD47. Future directions will utilize RT-PCR to look into discrepancies between mRNA and protein expression results.

A-22. Do parrotfish solve the cylinder task?
Morgan Leeper and Laurent Prétôt
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ManyFishes 1 is a large-scale collaboration that uses a standardized version of the cylinder task to investigate inhibitory control capacities across the fish taxon. In the task, subjects must successfully retrieve a food reward placed in a transparent cylinder without touching the cylinder first. Here, we tested parrotfish—an understudied group of marine herbivores that inhabit coral reefs and seagrass—in the task. As a group, parrotfish performed poorly, failing to retrieve the food without touching the cylinder first in most trials (94%; two-tailed binomial test, $N = 9$, $P < 0.001$). Compared to Lamarck's angelfish—an omnivorous coral reef fish previously tested in the same procedure—parrotfish showed a tendency towards failing more often than angelfish (two-tailed Fisher's exact test, $P = 0.075$). When examining whether success or failure in the task was associated with decision making, we found that parrotfish were more likely to make a decision than angelfish, regardless of the outcome (two-tailed Fisher's exact test, 90% vs. 70%; $P = 0.002$). We discuss the implications of these findings for ManyFishes 1 and, more broadly, our understanding of fish cognition and behavior.

A-23. Impact of CRISPR-induced mutations on *Arabidopsis thaliana* gene APETALA3 sites AP3-3 and AP3-5
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The goal of this project is to create CRISPR-CAS9 mutations in the APETELA3 (AP3) gene of the model plant *Arabidopsis thaliana*. AP3 is a gene required for normal flower development. While several alleles of AP3 already exist, these alleles are strong alleles that knockout gene function. The advantage of creating weak alleles is that they often reveal functions of a gene that are overlooked by knockout mutations. Thus, examining the phenotypes caused by weak alleles allow researchers to fully characterize gene function. Using CRISPR, we will create weak alleles at position sites AP3-3 and AP3-5. The CRISPR-CAS9 system can make a variety of mutations including small insertions, deletions, or substitutions. We will specifically choose and examine weak alleles for this project. The first generation of potentially mutated AP3-3 and AP3-5 plants has been planted, and some plants have progressed to a second generation where DNA will be isolated and examined for mutations in the AP3 gene. Consistent with AP3's role in floral development, we have already seen plants with abnormal flowers and sterile fruit. These observations suggest that we have generated mutations in the AP3 gene which will be confirmed by genomic sequencing. The potential mutant plants will be further analyzed for new phenotypes and how the mutations affect protein structure and function.

A-24. Potential efficacy of immunotoxin expressing *Salmonella typhimurium* against human cancers
Auditya Jain, and Phillip Harries
Department of Biology, Pittsburg State University

The use of engineered bacteria and viruses to attack cancer cells has become a viable anti-cancer therapy in recent years, particularly for cancers that have proven resistant to traditional treatments. In this project we focus on an attenuated strain of *Salmonella typhimurium* that expresses a modified version of the *Pseudomonas* exotoxin PE38 lacking its cell binding domain and fused to transforming growth factor alpha (TGFα). TGFα is a ligand for the epidermal growth factor receptor (EGFR) that is known to be highly overexpressed in certain cancer tissues. The strain of TGFα-PE38 expressing *Salmonella* has been tested on colon and breast cancers overexpressing EGFR but other possible targets have not been explored. In this study we utilize cancer gene expression tools to examine other human cancers that might be suitable targets for this *Salmonella* strain based on EGFR expression. We further analyze survival curves for high and low EGFR expressing cancers and look at promoter methylation patterns as a possible explanation for altered EGFR expression in these same cancers.

A-25. Alzheimer-mutant γ-secretase complexes stall amyloid β-peptide production
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Missense mutations in the amyloid precursor protein (APP) and presenilin-1 (PSEN1) cause early-onset familial Alzheimer's disease (FAD) and alter proteolytic production of secreted 38-to-43-residue amyloid β-peptides (Aβ) by the PSEN1-containing γ-secretase complex, ostensibly supporting the amyloid hypothesis of pathogenesis. However, proteolysis of APP substrate by γ-secretase is processive, involving initial endoproteolysis to produce long Aβ peptides of 48 or 49 residues followed by carboxypeptidase trimming in mostly tripeptide increments. We recently reported evidence that FAD mutations in APP and PSEN1 cause deficiencies in early steps in processive proteolysis of APP substrate C99 and that this results from stalled γ-secretase enzyme-substrate and/or enzyme-intermediate complexes. These stalled complexes triggered synaptic degeneration in a *C. elegans* model of FAD independently of Aβ production. Here we conducted full quantitative analysis of all proteolytic events on APP substrate by γ-secretase with six additional PSEN1 FAD mutations and found that all six are deficient in multiple processing steps. However, only one of these (F386S) was deficient in certain trimming steps but not in endoproteolysis. Fluorescence lifetime imaging microscopy in intact cells revealed that all six PSEN1 FAD mutations lead to stalled γ-secretase enzyme-substrate/intermediate complexes. The F386S mutation, however, does so only in Aβ-rich regions of the cells, not in C99-rich regions, consistent with the deficiencies of this mutant enzyme only in trimming of Aβ intermediates. These findings provide further evidence that FAD mutations lead to stalled and stabilized γ-secretase enzyme-substrate and/or enzyme-intermediate complexes and are consistent with the stalled process rather than the products of γ-secretase proteolysis as the pathogenic trigger.

A-26. Impact of antimicrobial resistance on virulence in *Pseudomonas fluorescens*
Tiffany Chan, Kervens Accilien, Robert Unckless PhD
Department of Molecular Biosciences, University of Kansas, Lawrence, KS

The rapid emergence of antimicrobial resistance in bacteria is a worldwide crisis that puts the effectiveness of antibiotics at risk. Several studies suggest, however, that where there is an adaptive advancement in one trait, in this case, antimicrobial resistance, there is a decreased performance in another, such as growth or virulence. This is referred to as resistance-virulence tradeoff. This study investigates the relationship between those traits in experimentally evolved resistant *Pseudomonas fluorescens* using *Drosophila melanogaster* as an *in vivo* infection model. The resistant strains showed a decreased within-host growth rate compared to the wild type. This data suggests there is a tradeoff between bacterial growth and antimicrobial resistance. Future studies examining other phenotypic traits will be conducted to clarify aspects of the tradeoff, which could provide insight into how bacteria adapt to their environment and potential ways to combat drug resistance.

A-27. Glycosylation unveiled: Exploring the structure and function of FSH hormone glycoforms
Yara Abdine, Alan R. Brown, Viktor Y. Butnev, William K. White, Jeffrey V. May and George R. Bousfield
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Follicle-stimulating hormone (FSH), luteinizing hormone (LH), and human chorionic gonadotropin (hCG) play pivotal roles in human reproduction. FSH initiates the growth, differentiation, and maturation of ovarian follicles. FSH causes fully developed follicles to develop & release a mature oocyte in response to the mid-cycle surge in LH concentration. The mature oocyte then resumes meiosis I and advances to metaphase II, resulting in an oocyte that is capable of fertilization by sperm. In the testes, FSH supports spermatogenesis. FSH is a glycoprotein composed of a common alpha (shared with thyroid-stimulating hormone, hCG, and LH) and hormone-specific FSH-beta (exclusive to FSH) subunits. Our research focuses on unraveling the intricate roles of naturally occurring FSH variants called glycoforms. Glycosylation, the attachment of oligosaccharide molecules (carbohydrates) to the protein backbone, is crucial for the structural stability and functionality of glycoproteins. Both alpha and beta subunits of FSH possess two N-linked glycosylation sites; however, the occupancy of glycans on the beta-subunit sites may vary. The three-dimensional structures of LH and FSH are modulated by the addition of N-linked glycans, along with the degree of glycosylation, giving rise to diverse glycoforms with distinct structural stability and functionality. This research aims to develop a FSH18-specific ELISA, an FSH21-specific ELISA, an FSH24-specific ELISA, and a Pan FSH ELISA (recognizes all 3 glycoforms equivalently). FSH glycoform measurement will contribute to a deeper understanding of the glycosylation patterns in FSH and their implications in reproductive processes.
Keywords: Follicle-stimulating hormone, Glycosylation, Glycoforms, Reproductive biology, ELISA

A-28. Leisure Interests in Individuals with Intellectual and Developmental Disabilities
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Individuals diagnosed with Intellectual and Developmental Disabilities participate in fewer leisure activities as age increases (King et. al, 2022). It is important to keep those diagnosed with an ID/DD active throughout their lives. Active lifestyles can decrease potential health risks and increase quality of life (Heister, 2023). Identifying leisure interests within the population can help determine potential leisure interventions that will keep them active throughout their lives. **Methods**: In the spring of 2025, fifteen adults with ID/DD will be recruited from a day service program to complete the Leisure Assessment Inventory. The assessment will collect data on leisure interests and barriers to leisure. Inclusion criteria for this study is adults over the age 18 diagnosed with an ID/DD that can communicate verbally or non-verbally with the researchers. Trained undergraduate student lab assistants will collect the data. Based on previous research predicted outcomes the ID/DD sample group will participate in more sedentary activities compared to physically active activities (Heister, 2023). Additionally, barriers predicted for this sample group will show a lack of knowledge and lack of opportunity (de Leeuw et. al, 2022).

A-29. Pair bonding in prairie voles requires VTA-NAc dopamine neurons
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Prairie voles (*Microtus ochrogaster*) are socially monogamous rodents that form pair bonds with their mate. Once a pair bond is formed, prairie voles demonstrate preference towards their partner and stranger aggression. Dopamine, specifically in the nucleus accumbens (NAc), plays a critical role in the formation of pair bonds between prairie voles, and it is suggested that the VTA regulates dopamine in the limbic reward system. However, little is known about the regulation of bond formation by the VTA. We hypothesize that dopamine projections from the VTA into the NAc promote the formation of pair bonds and the maintenance of these pair bonds, because it is known that dopamine in the NAc regulates these behaviors. Presently, we have applied the immunotoxin anti DAT-SAP into the NAc to determine its effects on partner preference formation in male and female prairie voles after cohabitation with a new opposite-sex partner. Thus far, we have found that this manipulation inhibits the formation of pair bonds in female prairie voles after a twenty-four-hour cohabitation, a period typically sufficient to form partner preference. To further investigate how this toxin manipulates the dopamine VTA-NAc projection, we plan to run an immunohistochemistry to analyze the presence of tyrosine hydroxylase (TH) fibers, D1 and D2 receptors in the NAc, and CRFR1 receptors in the VTA. This research will determine if elimination of VTA-NAc dopamine neurons prevents pair bonding, demonstrating the involvement of the dopamine-rich VTA in social attachment formation.

A-30. Using DNA repair inhibitors to increase the efficacy of chemotherapy in cervical cancer
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Cervical cancer (CaCx) caused by HPV takes the life of someone every 90 seconds, making it the fourth most common cancer among women globally. Effective treatment options are limited. The standard chemotherapy for CaCx is Cisplatin, which damages DNA in all cells. Cisplatin is, in part, effective because CaCx has a reduced capacity to repair DNA damage in comparison to untransformed cells. However, there are two frequently arising issues: (i) development of resistance and (ii) dose limiting side effects. Thus, there is a critical need to develop alternative approaches to CaCx chemotherapy. AOH1996 is a proliferating cell nuclear antigen (PCNA) inhibitor that targets a cancer specific isoform of PCNA, in turn, giving specificity for CaCx treatment. PCNA is a critical component of DNA repair. I hypothesize that the combination treatment of AOH1996 and cisplatin will synergize effectively in CaCx models. I will use MTT assays to define the ability of AOH1996 to sensitize CaCx cells to a range of cisplatin concentrations. I will also determine the ability of AOH1996 to enhance the efficacy of cisplatin *in vivo* by implanting CaCx cells into an immunocompetent mouse. Moving to an animal model will allow me to assess survival, tumor size, and systemic toxicity. With supporting work, moving to immunocompetent models will allow me to better compare this treatment in a pre-clinical setting.

A-31. Using eDNA to detect Craspedacusta sowerbii (freshwater jellyfish) in aquatic ecosystems
Nadia Valdez Saravia, Rachel Bowes
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Craspedacusta sowerbii, a freshwater hydrozoan species native to Asia, has successfully adapted to diverse freshwater environments across North and South America, Europe, Asia, and Australia. Its life cycle alternates between asexual polyps and sexually reproducing, free-swimming medusae, the latter becoming more prominent during warmer seasons. These temperature-driven blooms often occur in summer and early fall but are intermittent and irregular, complicating detection with traditional sampling methods. Although endangered in its native habitat, *C. sowerbii* is considered invasive in North America, with unknown impacts on native ecosystems and limited knowledge of its non-native distribution.
Environmental DNA (eDNA) offers a promising solution for monitoring this invasive species. eDNA, comprising genetic material shed by organisms into their surroundings, has become a widely accepted tool for detecting aquatic species, including those difficult to observe or capture. It enables species identification and mapping of invasive ranges, providing critical data for ecological monitoring and management. This study explores the potential of eDNA to detect and map the invasion range of *C. sowerbii* in Kansas reservoirs. By utilizing eDNA techniques, it can overcome traditional detection challenges, offering a cost-effective and non-invasive approach to track the spread of *C. sowerbii* and inform conservation strategies. This approach holds promise for identifying the full extent of the species' invasive range and understanding its ecological implications.

A-32. Impact of *APOE4* and high-fat diet on skeletal muscle morphology in a mouse model of Alzheimer's disease
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Accounting for 60-70% of dementia cases, Alzheimer's Disease (AD) is a type of dementia characterized by severe memory loss and cognitive decline. The only genetic risk factor for late-onset AD is the E4 mutation on the APOE gene. APOE4 carriers present with increased muscle degeneration after diagnosis of AD. Skeletal muscle serves as a major site of carbohydrate and lipid oxidation, and skeletal muscle atrophies, type II fibers (fast-twitch) decrease more significantly. Prior research demonstrated that improved cardiovascular health is correlated with greater hippocampal volume and memory improvement. This study investigates the skeletal muscle morphology of APOE4 and APOE3 in male and female targeted replacement (TR) mice on high-fat (HFD) and low-fat (LFD) diets through skeletal muscle fiber-typing and lipid droplet staining. A HFD led to a decrease in oxidative muscle fibers in APOE4 transgenic (TR) mice compared to APOE3 TR mice. This was evident from the reduced percentage of type I (slow oxidative) and type I/IIA hybrid fibers in both male and female APOE4 mice. In HFD females, APOE4 was also associated with fewer type IIA fibers (fast oxidative/glycolytic) compared to APOE3. Lipid droplet accumulation, observed through BODIPY 493/503 staining, was prominent in HFD mice regardless of APOE genotype or sex and was absent in low-fat diet (LFD) mice. These lipid droplets were predominantly found in type IIA fibers, as shown by fiber-type-specific staining.

A-33. Carminic Acid from Cochineal Insects as Color Sensor for Monitoring the Freshness of Cheese
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The application of natural dyes in food safety has gained significant attention due to their biocompatibility and eco-friendliness. This study focuses on the application of carminic acid from cochineal insects (*Dactylopius coccus*) as a color sensor for monitoring the freshness of cheese. Carminic acid is sensitive to pH changes, making it an ideal indicator for detecting spoilage in cheese storage caused by microbial activity and acidity fluctuations. The corn starch, chosen as a carrier for carminic acid due to its biodegradability and non-toxicity, was utilized as a robust biomaterial. For this purpose, carminic acid was dissolved in aqueous solution followed by the addition of starch. Various salts including zinc chloride, lithium chloride and strontium chloride were also added into the final dispersion, to study the color sensing properties. The synthesized particles were centrifuged, dried in an oven and pressed in tablet forms for the cheese monitoring.
FTIR analytical characterization confirmed the stability of carminic acid and various salts within the starch matrix and their responsiveness to pH variations in the range typically observed during cheese spoilage. Spectro-colorimeter tests demonstrated that the tablets successfully detected early spoilage in various cheese samples, with visible color changes correlating to pH thresholds indicative of spoilage onset.
The results suggest that carminic acid-loaded starch tablets are a reliable, cost-effective, and consumer-friendly solution for monitoring cheese freshness. This approach highlights the potential of natural dye-based sensors in advancing food safety, providing a practical solution for producers and consumers to ensure high-quality standards in dairy products.

A-34. Effect of glyphosate and bacterial community structure on cellulose metabolism
Alexa McGann, Ella Rulliffson and Stephen Fields
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Soil bacteria play a crucial role in the degradation and cycling of materials and nutrients within the ecosystem. Cellulose, a polysaccharide found in plant cell walls, is the most prevalent organic molecule found in soil. Many soil bacteria have enzymatic pathways for breaking down cellulose, which also aids soil health by releasing nutrients for plants and other microbes. Our lab has isolated, sequenced and annotated eight bacterial species from a soil community. Some of these species contain many cellulases, while others do not have enough to metabolize cellulose on their own. We are testing the ability of this sub-community to synergistically survive on carboxymethylcellulose (CMC). These species have been plated together and individually on agar gradients that reduce glucose and increase CMC until CMC is the only carbon source at the end. In theory, some or all the bacterial species will work together as a community, utilizing their cellulases to synergistically metabolize cellulose. Our goal is to eventually re-sequence all eight species after continuous growth cycles on CMC to look for adaptive mutations and horizontal gene transfer. Many herbicides, including glyphosate, have major effects on soil microbial communities. Glyphosate inhibits a key enzyme in the shikimate pathway that is common to both plants and microorganisms. Therefore, it is hypothesized that this herbicide will disrupt the growth of microorganisms present in the soil. We are currently testing these same soil species for sensitivity to glyphosate and will also measure the effect of glyphosate on cellulose metabolism.

A-35. Flow Cytometry Core: A Chemical Biology of Infectious Disease COBRE Core Laboratory.
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The University of Kansas Flow Cytometry Core (FCC) provides access to flow cytometry and cell sorting instrumentation and expertise to researchers. Services and training are provided for flow cytometry: cell sorting and multi-parametric analysis of individual cells in solution, calculated from their fluorescent or light scattering characteristics. The FCC provides assistance in sample processing, data analysis, instrument training, software support, method and grant assistance, manuscript support, and consulting. The FCC is a 980 ft2 BSL-2 facility equipped with a BD FACSymphony™ S6 Cell Sorter, a BD FACSAria™ Fusion cell sorter, a Cytek™ Aurora spectral flow cytometer, an Agilent NovoCyte Advanteon conventional flow cytometer, and other supplemental assay instrumentation. The flow cytometry analyzers provide users with tube- and plate-based, conventional and spectral flow cytometry. The BD FACS instruments allow measurement and sorting of up to 6 resolved populations of cells simultaneously, based on up to 50 parameters of detection using 18 simultaneous fluorochromes. The facility is equipped to handle BSL-2 samples and perform aseptic and single cell sorting into tubes or 96-well plates. The facility provides instrument training for users who desire to become self-operators of the facility instruments. The FCC will equip CBID researchers with tools directly applicable to infectious disease research, such as identifying and characterizing infectious agents such as bacteria and parasites, quantification and sorting of cells infected with microbial pathogens, and assessing chemical probe efficacy against infectious agents. The University of Kansas Flow Cytometry Core seeks to assist the academic community in achieving their research goals.

B-1. Molecular Effects of PDGFR*β* in the Gain of Function in Mice
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Platelet-derived growth factor (PDGF) signals through two receptor tyrosine kinases, PDGFRα and PDGFRβ, playing a role in epithelial malignancies by promoting tumor growth, angiogenesis, invasion, and metastasis. PDGFRβ mutations have been linked to connective tissue disorders, such as Penttinen syndrome (characterized by early aging, lipoatrophy, and skin atrophy) and Kosaki overgrowth syndrome (marked by skeletal overgrowth, fragile skin, and neuropsychiatric symptoms). However, the precise role of PDGFRβ mutations remains unclear.
This study investigates the molecular effects of PDGFRβ gain-of-function mutations in mice. Previous work in the lab found elevated levels of total and phosphorylated STAT1 in these models, suggesting STAT1 as a modulator of the observed phenotypes. However, the regulation of activated STAT1 is not fully understood. The aim of this study was to assess whether disrupting downstream signaling could reduce STAT1 activation.
Using doxycycline-inducible NIH 3T3 cells transfected with the V665A PDGFRβ mutation or wild-type PDGFRβ, the study found that V665A-induced PDGFRβ activation did not rely on JAK2, as treatment with the JAK2 inhibitor Ruxolitinib did not reduce STAT1 phosphorylation. However, treatment with Rapamycin, an mTORC inhibitor, reduced phosphorylated STAT1 levels. These results suggest that STAT1 phosphorylation can be modulated by alternative downstream pathways, offering insights into potential therapeutic targets for disorders linked to PDGFRβ mutations.

B-2. Comparison of the Thermal Ecology of Ornate Box turtles (Terrapene ornata) across two distinct populations
Kaitlyn Scott, Ally Huber, Megan Norris, Abigail Trautman, Benjamin Reed
Washburn University

Thermal ecology includes investigating interactions between temperature and organismal function. These temperature interactions can influence an organism's physiology, their relationship with the environment, as well as their behavioral patterns. Body temperature also plays a role in an organism's metabolism, digestion rate, growth rate, heart rate, and immune function. Studying the bioenergetics of an animal can provide more information on their overall health and performance (including movement, disease spread/parasitism, mating, and reproduction). Currently, the effects of climate change are being studied in agriculture, species range shifting, and changing ocean dynamics, yet little is known about the effects of temperature change with respect to some of Kansas' local fauna, especially with respect to ectotherms. In our study, we investigated the thermal ecology of two distinct populations of Ornate Box Turtles (*Terrapene ornata*) by closely monitoring their shell temperature (as a proxy for body temperature) simultaneously with their movement patterns, microhabitat use, range size, and mating behaviors. Our findings show clear differences in average temperature and variability within and between populations of turtles and distinctly different temperature profiles during their primary morning active period which likely has important consequences for individual fitness and population-level persistence in the face of changing climate. This data will also serve as an important baseline when examining box turtle thermal ecology in future studies.

B-3. Understanding the role of gut microbiota in the development visceral hypersensitivity – A literature review
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Persistent abdominal pain is one of the most impactful symptoms across various gastrointestinal disorders. Increased sensitivity to bowel distention, termed visceral hypersensitivity (VH), is a primary driver of chronic abdominal pain. Differences in microbial colonization are associated with disease, but how bacteria drive pain and the potential therapeutic targets remain poorly understood. This review illustrates applications of microbiome-based diagnostics referring to currently available literature and also describes the aims of the proposed research. Using metagenomic analysis, the role of microbial communities in many infectious diseases can be ascertained including response to cancer immunotherapy and transplant safety. Investigating the effects of microbiota on VH will supplement our current understanding of how these milieus change over a given treatment regime. This background knowledge leads to our first specific aim: to study how the composition of the gut microbiome evolves temporally when VH is induced in both male and female mice by sequencing the collected fecal material and comparing using 16S metagenomics. Future research may be focused on developing precise methods for manipulating the microbiome and creating targeted treatments for specific conditions. This frames our second specific aim: to study the microbiota after treatment with broad spectrum antibiotics or spermidine-supplemented diet employing molecular diagnostics such as multiplex qPCR technique to detect selected bacterial species (e.g. *Bacteroides, Enterococcus, Dorea, Clostridium and/or Ruminococcus*). The ultimate goal of this research project is to systematically assess the impact of altered microbial colonization on the host and examine novel therapeutic approaches to restoration/stabilization of the microbial response.

B-4. Breast Cancer's Toll on Exercise Capacity

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Introduction: Breast cancer (BC) is the leading cancer in women nationwide and is characterized by uncontrolled cell growth in the breast. Despite improvements in treatment strategies, there are currently 4 million BC survivors nationwide that experience cancer-related exhaustion, decreased exercise tolerance, and a very low maximal oxygen uptake during exercise ($\dot{V}O_2$max). These effects are largely attributed to the effect of chemotherapy on cardiac and skeletal muscle. However, the tumor itself is profoundly pro-inflammatory and may also contribute to reduced exercise capacity. We hypothesized that tumor-bearing alone would reduce $\dot{V}O_2$max and exercise tolerance in BC rats. **Methods:** Female Fischer-344 rats were used in this study. We measured $\dot{V}O_2$max in all rats prior to intraductal injections of MATBIII adenocarcinoma cells (6 x 10^3 in 0.5 µL saline) and following tumor growth (~24 days). **Results:** Alternative to our hypothesis, there was no significant change in $\dot{V}O_2$max between pre-BC and post-BC measurements (72.1 ± 2.7 ml/kg·min vs. 70.0 ± 3.1 ml/kg·min; $P > 0.05$). **Conclusion:** These results suggest that anti-cancer treatment, rather than the pro-inflammatory tumor itself, contributes to reductions in $\dot{V}O_2$max. Altogether, emphasis must be placed on the effects of chemotoxicities on cardiorespiratory health, and effective treatment strategies for individuals with BC that do not impair cardiac and skeletal muscle O_2 transport are needed to improve longevity and quality of life for the 4 million BC survivors nationwide.

B-5. A Study of the Morphological Effects of Potential Detox Hair Treatments Using Polarized Light Microscopy

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Hair samples are often used for drug testing to identify the presence and habitual use of controlled substances, especially since hair will retain controlled substances for a longer time than bodily fluids. However, there is not much research regarding whether "detoxifying" agents or other hair treatments can potentially remove the drug trace or alter the morphological features of human hair during the detox process. This study aimed to use polarized light microscopy and scale casting to determine any morphological changes that may be due to a variety of chemical hair treatments.
In this study, hair samples were collected from a third-party source and treated with a variety of over-the-counter products: Heads & Shoulders Shampoo, Olaplex Shampoo, Paul Mitchell Shampoo, Shimmer Lights lightener and developer, and L'Oreal Paris Excellence Cream in Medium Brown. The physical structure of the hair was examined both before and after treatment. It was apparent that the hair dyes and bleaching, which results in oxidation of the hair (especially under alkaline conditions), lead to significant changes in the morphology of hair. The hair samples that were treated with dye or bleaching chemicals resulted in some differences in their point of extinction, inference colors, and general color appearance.

B-6. Controlled Release Studies of Quercetin Encapsulated in Chitosan and N-Phthaloyl Chitosan Microparticles

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Quercetin is a flavonoid found in fruits and is well-known for its health benefits, including antioxidant, anti-inflammatory, and potential anti-cancer properties. Despite these properties, quercetin presents low aqueous solubility, light-induced degradation, and poor bioavailability. To address these limitations, this study aims to develop and characterize chitosan and N-phthaloyl chitosan microparticles (drug delivery systems) stabilized with *iota*-carrageenan. The degree of N-phthaloylation will be optimized to support interactions with quercetin, primarily through π-π interactions. This could lead to sustained quercetin release in intestinal environments. Microparticles will be prepared using a microemulsion method. The oil phase will consist of sunflower oil, while the aqueous phase will include chitosan or N-phthaloyl chitosan, poly(ethylene glycol)-b-poly(propylene glycol)-b-poly(ethylene glycol), iron oxide, and quercetin. Once the emulsion is formed, aliquots of an *iota*-carrageenan solution will be added under high-speed stirring, leading to the formation of quercetin-loaded microparticles via an in-situ process. Alternatively, microparticles can be prepared first and subsequently loaded with quercetin using an ex-situ strategy. Iron oxide (Fe_3O_4) may be incorporated to promote magnetic properties in the drug delivery system. Release studies will be conducted in simulated gastric and intestinal fluids, both with and without lysozyme. Mathematical models will be applied to the release profiles to investigate the release mechanisms. The primary objective is to demonstrate that these microparticles can protect quercetin from degradation, promote its sustained release, and enhance its solubility and bioavailability. Additional assays, including antimicrobial, antioxidant, and cytocompatibility tests, may be performed to evaluate the retention of quercetin's bioactive properties after encapsulation.

B-7. Downregulation of the nutrient-sensitive post-translational modification, O-GlcNAcylation, attenuates Autosomal Dominant Polycystic Kidney Disease and increases survival in a mouse model

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Autosomal dominant polycystic kidney disease (ADPKD) is the 4th leading cause of kidney failure worldwide. This genetic disease causes the progressive growth of fluid-filled kidney cysts resulting in irreparable damage. A major component of ADPKD pathogenesis is altered cell metabolism, although drivers of these alterations are not understood. Addition of O-linked β-N-acetylglucosamine (O-GlcNAc) onto protein substrates by O-GlcNAc transferase (OGT) is a nutrient-sensitive post-translational modification that integrates multiple metabolic signals. We have reported that protein O-GlcNAcylation is increased in kidneys of ADPKD patients and mouse models. Further, *Ogt* deletion in the collecting ducts of a juvenile ADPKD mouse model, *Pkd1* conditional knock-out (cko);*HoxB7-Cre* mice, markedly reduces disease severity and improves kidney function. Here we show that while *Pkd1* cko;*HoxB7-Cre* mice die between postnatal day (P)14-P20, strikingly, *Pkd1;Ogt* double ko (dko);*HoxB7-Cre* mice continue to thrive beyond 1 year of age. Remarkably, kidney weight:body weight (KW/BW) ratios of *Pkd1;Ogt* dko;*HoxB7-Cre* mice are lower at 1 year of age than at P14. While immunostaining of kidney sections revealed an increase in inflammation and myofibroblasts in the renal medulla of *Pkd1;Ogt* dko;*HoxB7-Cre*, the renal cortex largely resembled kidneys of control littermates. In an adult ADPKD mouse model, *Pkd1* cko*;Pax8rt-TA;LC1-Cre* mice, deletion of *Ogt* in the nephron similarly attenuated disease severity, with reduced KW/BW ratios, inflammation and fibrosis, and reduced alteration of signaling pathways. Finally, OGT inhibition reduced *in vitro* cyst formation of cultured ADPKD patient-derived cells, indicating clinical significance. These data indicate *Ogt* is a critical node in ADPKD pathogenesis with strong therapeutic potential.

B-8. *Early Overexpression of the Kinase RsbW Impacts Growth and Progeny Production of Chlamydia trachomatis*
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Chlamydia trachomatis is the most prevalent sexually transmitted bacterial infection worldwide, representing a major public health concern. This obligate intracellular bacterium has a phylum-defining biphasic developmental cycle characterized by the organism's interconversion between infectious and replicative forms. One protein system that has been shown to govern the cycle's regulation is the Rsb system. This system works based on the phosphorylation state of an intermediate, $RsbV_1$, which determines the activity of a periplasmic sensor phosphatase and a terminal protein partner kinase, RsbU and RsbW respectively. Although genetic disruption of RsbU has been shown to cause a dramatic decrease in progeny production, alteration of RsbW's expression during the last stage of development does not, making the Rsb system's role in *C. trachomatis*' development unclear. We studied the temporal specificity of the Rsb system by studying the effect of RsbW's overexpression on progeny production and growth. We performed these experiments at different developmental time points with a tetracycline inducible system, using immunofluorescent microscopy and ddPCR to determine effects on morphology and genome replication. Starting overexpression at 0- or 8-hours post-infection led to a dramatic decrease in progeny production. This also resulted in the production of smaller and aberrant inclusions, as well as decreased genome replication. A kinase-defective RsbW mutant is currently being transformed into *C. trachomatis* to validate these results and confirm the importance of the protein's phosphorylating activity, clarifying the system's specific role in development regulation and potential as a therapeutic target

B-9. Exploring Fertility Trends of Ornate Box Turtles via Ultrasonography in Western Nebraska
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Investigating and understanding reproductive ecology for any animal is important for numerous reasons, including: identifying individual and species needs, individual fitness (relative reproductive output), population trends (growth/decline), and ultimately should better inform management decisions in conservation. In many species, especially ectotherms, the reproductive ecology is widely unknown and/or understudied. The goal of this research was to examine the reproductive ecology and fertility trends in an understudied/lesser known species, the Ornate Box Turtle (*Terrapene ornata*) via ultrasonography. In this study, we monitored and documented the reproductive cycle of 24 female box turtles on a four-day rotation (six per day). Here, we characterized the reproductive development cycle (ovum to hardshell egg) in these female turtles. We also investigated whether female *T. ornata* individuals were capable of reproducing over consecutive years, and also if they are able to have two nesting periods in a single season. This study is important as it broadens our understanding of reproductive ecology and how reproductive success relates to fitness. Our research could spark future interest in understanding why certain females are having more reproductive success than others, which may potentially be related to variation across individuals in home range size, personality traits, and health. Finally, comparing egg-bearing trends across separate populations may be highly informative for identifying populations at risk or those that are performing well across their range.

B-10. Antioxidants and Neonicotinoids: A Further Look into the Impact of Plant Antioxidants and Cannabinoids
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Over 130 commercial crops are pollinated by *Apis mellifera*, adding $15 billion to the U.S. economy each year. However, in 2023, beekeepers lost 48.2% of their hives due to stressors including pesticide exposure. Imidacloprid, a neonicotinoid pesticide, makes up ⅕ of the global insecticide market. This prevalence in both agricultural and private environments leads to high levels of exposure to forager honey bees. Exposure to this pesticide causes elevated levels of oxidative stress, which reduces the ability to break down toxins resulting in cell damage and death in the gut. This class of pesticides is also linked to genetic down-regulation of many *A. mellifera* detoxification genes. Antioxidant compounds that are naturally present in many plant species may be able to counteract the harmful effects of this pesticide exposure by increasing the expression of the detoxification pathway. In a previous study by this lab 2 plant antioxidants and 2 cannabinoids showed promise for short term alleviation of Imidacloprid exposure. epigallocatechin-3-O-gallate, peppermint essential oils, CBD, and CBN were all analyzed. To assess if these plant-based antioxidants/cannabinoids contain a protective effect for neonicotinoid poisoning, a survivorship protocol was followed with modified containers for long-term incubation. The bees were incubated from pupa to workers and separated into 30 modified containers, each fitting 20-30 bees and given a regular food source which contained a control, experimental dosage of Antioxidant/cannabinoid, or Antioxidant/cannabinoid-Imidicloprid mix. Survivors were counted over a 8 day period and flash frozen at the end for RNA sequencing.

B-11. Floristic Summary of Bates County, Missouri
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Overall human health is directly tied to the health of the local environment. The health of local environments depends in some measure on what native and non-native plants occupy the land. Recurring censusing of species in an area is an effective way to monitor the ecological health of an ecosystem. However, many counties in the USA have never had their plant (= floristic) diversity fully documented. Previous research suggested that Bates County, Missouri, was less surveyed than other counties in the state. Our hypothesis is that at least a dozen or more plant species will be documented for the first time in Bates County. Our research involves multiple collecting trips to survey floristic diversity, and we have permission to include areas managed by the Missouri Department of Conservation. Of the approximately 350 specimens collected thus far in the first field season, eighteen were documented for the first time in Bates County, including the aggressively invasive understory Amur honeysuckle. This project will continue through the summer of 2026. Even our early results corroborate the hypothesis that Bates County is incompletely documented. It is well known that plant distributions change through time, and climate change likely is exacerbating such shifts. Our project is providing baseline data for future ecological comparisons for things such as flowering times, the presence of invasive species, and monitoring the occurrences of rare taxa.

B-12. Advancing the Use of eDNA to Monitor Plant-Pollinator Communities
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In North America, over 99 percent of prairie has been destroyed in most states, negatively impacting pollinators and their affiliated animal communities. As 90 percent of flowering plant species, including 35 percent of agricultural crops, depend on pollinators for reproduction, the loss of pollinator diversity has drastic implications on plant diversity, food crop production, and overall ecosystem stability. Here, we use a non-invasive means to monitor insect communities in concert with characterizing the floral community in a newly established prairie undergoing active restoration. DNA can be deposited in detectable amounts on flowers during plant-animal interactions as environmental DNA (eDNA). In recent years, eDNA has been successfully extracted as a means of detecting floral visitors. As DNA is a water-soluble molecule, weather events can disrupt the detection of eDNA. To monitor communities, we experimentally controlled precipitation as a factor leading to loss in detection. Additionally, we used transect surveys to describe the plant community, extracting DNA from those flowers to characterize the insect pollinator community. Floral samples were collected from set transects; surveys of flowering plant species along these transects were performed to track plant community shifts. Using DNA extraction and metabarcoding, we aim to characterize these pollinator communities. Through understanding detection of eDNA under variable environmental conditions, we seek to monitor ecosystem health.

B-13. Analysis of antimicrobial activity of phytochemicals in *S. officinalis*
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Natural products from plants are good substitutes for current antibiotics. Currently antibiotic resistance in humans is leading to exploration of better alternative antibiotics. Phytochemicals are natural compounds and better for human health. The objective of the research was to test and isolate phytochemicals from ***Salvia officinalis*** for antimicrobial activity. The significance of this study is to possibly identify new antimicrobial compounds to combat resistant strains of microorganisms that are becoming more prevalent. Methanol was used as a solvent for Soxhlet extraction from stems and leaves of ***S. officinalis***. Disc diffusion method with the extract showed inhibition against ***Escherichia coli*** in all applications. Further testing and phytochemical screening are in progress to characterize bioactive constituents in ***S. officinalis***.

B-14. How APOE Genotypes Affect Mitochondrial Phenotypes In Alzheimer's Disease
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Alzheimer's disease (AD) is the most common form of dementia, causing memory loss and behavioral changes. Genetic variation in the lipid transporter protein, apolipoprotein E (APOE), can increase (APOE E4) or decrease (APOE E2) the risk of AD, however, the mechanism is not yet fully understood. Past research has proposed the mitochondria to be a likely candidate for triggering AD progression and some studies suggest APOE E4 can affect mitochondrial function. Therefore, we investigated the effect of APOE genetic variation on mitochondria. We hypothesized that APOE E4 would lead to decreased mitochondrial health. We found that astrocytes and neurons derived from APOE E4 iPSCs had increased hydrogen peroxide production, increased mitochondrial membrane potential, and increased mitochondrial superoxide production compared to APOE E2 and APOE E3-derived cells. We also found that APOE E4 iPSC-derived astrocytes and neurons had decreased mitochondrial respiration compared to APOE E3-derived cells. Lastly, we found that APOE E4 iPSC-derived neurons had increased intercellular and mitochondrial calcium levels compared to APOE E2 and APOE E3-derived cells. We conclude that cells that possess APOE E4 alleles have decreased mitochondrial health compared to cells that possess APOE E2 or E3 alleles leading us to continue to investigate the mitochondria and its role in AD.

B-15. Modulating T-Cell Immune Responses via Keap1-Nrf2 Pathway Regulation Using PROTACs
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T-lymphocytes, or T-cells, are essential components of the adaptive immune system, orchestrating immune responses against infections and tumors. Their function is tightly regulated by intracellular pathways, including the Keap1-Nrf2 antioxidant pathway, which maintains cellular redox balance. Under basal conditions, Nrf2 is suppressed by Keap1 and directed for ubiquitination. However, oxidative stress triggers conformational changes in Keap1, releasing Nrf2 to activate antioxidant gene expression, thereby supporting immune cell survival and function.
Proteolysis Targeting Chimeras (PROTACs) are an innovative therapeutic strategy for selectively degrading target proteins, allowing precise cellular pathway modulation and expanding the range of druggable targets. This study investigates the potential of Keap1-targeting (Keap1 PROTAC) and Nrf2-targeting (Nrf2 PROTAC) molecules to influence Nrf2 levels and T-cell cytokine production.
Experiments were conducted using Jurkat T-cell lines and human peripheral blood mononuclear cells (PBMCs). In Jurkat cells, Keap1 PROTAC treatment increased Nrf2 protein levels, as confirmed by Western blot. In PBMCs, Keap1 PROTAC enhanced interleukin-2 (IL-2) production, a critical cytokine for T-cell proliferation, while Nrf2 PROTAC reduced IL-2 levels, as measured by ELISA. Similarly, trends for interferon-gamma (IFN-γ) supported the cytokine-modulatory effects of these PROTACs, demonstrated through ELISA and flow cytometry.
These findings establish PROTACs as promising tools for manipulating the Keap1-Nrf2 pathway and modulating cytokine production in T-cells, with potential applications in immunotherapy. Ongoing studies aim to validate these results across additional donor samples and refine PROTAC efficacy, paving the way for advanced T-cell-based immunotherapies. Keywords: Keap1-Nrf2 pathway, T-cell modulation, PROTACs, cytokine regulation, immune therapy, T-cell cytokines, adaptive immunity

B-16. The impact of thyroid hormone on microglia morphology
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Demyelination, characterized by damage to the myelin sheath, is a hallmark of many neurological diseases, including multiple sclerosis. In the central nervous system, microglia serve as the resident immune cells and are closely linked to demyelination and remyelination processes. Specifically, microglia phagocytose myelin debris consequent of demyelination. Microglia can assume various morphological states depending on the environmental and pathological contexts. In response to their environment, they generally take on three distinct forms: ramified, amoeboid, and phagocytic. Thyroid hormone (TH) agonists are another key component to neurological health through their promotion of myelination. Our lab has recently demonstrated that TH agonists and myelin debris induce phagocytosis in microglia. Additionally, in demyelinating diseases, morphology shifts from ramified to phagocytic during peak demyelination in animal models. However, the specific effect of TH on microglial states has yet to be fully understood. To address this, we utilized primary cultured microglia from rat pups and treated them with T3, an active form of TH, and Sobetirome, a TH receptor-β agonist, in both the presence and absence of myelin. Overall, we hope to establish a relationship between the morphology and function of microglia treated with TH agonists, providing further insights into the mechanism of myelin debris clearance by microglia.

B-17. Muscle Fiber Types and Cross-Sectional Area of Intrinsic Rat Tongue Muscles as a Function of Age and Intrinsic Aerobic Capacity
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Physiological changes with aging can result in decreased motor function. Although most studies of motor function focus on locomotion or limb movements, we are interested in orolingual motor function due to its relationship to dysarthria and dysphagia. We recently found that rats that are selectively bred for low or high aerobic capacity exhibit differences in orolingual motor function. Specifically tongue motility is significantly lower in rats will low aerobic capacity (LCR) than in their age-matched high aerobic capacity (HCR) counterparts. In order to determine the role of muscle fiber properties in these differences, we examined intrinsic tongue muscles in young adult (8 month-old) and aged (25 month-old) female LCR and HCR rats. Tongues were fixed in formalin and then cut in thirds (0.25 cm from the anterior and posterior regions) and placed in ethanol. Tongues were then processed and embedded in paraffin with the anterior or posterior regions facing out. A microtome was used to cut 6 micron-thick sections. We then used an immunofluorescence staining procedure to detect different muscle fiber types. So far we have found non-specific binding with all antibodies, making it difficult to differentiate the different fiber types. Our next steps are to optimize the protocol using different dilutions and more washes. We will then use Muscle J (ImageJ) to count fiber types and perform cross-sectional analysis. Finally, we will compare to gastrocnemius muscle from the same animals.

B-18. Cystic Cestodes: Utilizing qPCR for Species Identification of Parasitic tapeworms in sheep and cattle
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Echinococcus granulosus and Taenia hydatigena cause cysts in pigs, cattle, and sheep. It is important to identify these cysts accurately to understand how these parasites are transmitted, mainly because E. granulosus is zoonotic and T. hydatigena is non-zoonotic. Differentiating these species enables veterinarians to implement effective infection control measures. Previously gel based PCR with Sanger sequencing was the standard technique used for cyst identification but the procedure can take several days. Our research aims to utilize Quantitative PCR for quicker differentiation of cyst types. We tested four different primer sets on DNA extracted from cysts obtained from cattle and sheep in the US, validating qPCR performance against known E. granulosus and T. hydatigena samples through dilution and melt curve analysis. We hypothesized that one qPCR method would yield higher amplification and specificity for detecting these cysts. Our validated qPCRs were then applied to 12 unknown cyst samples. Primers designed by Deghani accurately differentiated the parasites in 100% of samples tested, while primers designed by Maksimo did so in 66%. Two other primer sets that we tested were unable to amplify cyst DNA. This qPCR method proves to be a reliable and rapid diagnostic tool for identifying cysts caused by these parasites in animals.

B-19. Transcriptomic Analysis of EGFR and Downstream Pathway Expression in MCF7 and Healthy Breast Epithelium
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Background: Clinical tests utilizing EGFR isoforms as a method of cancer screening have had an been unreliable history to date. Our lab has an interest in this gene's isoforms and downstream activation. We used an In-silco characterization and assessment of an Invasive Breast Ductal Carcinoma adenocarcinoma cell line (MCF7) in association with expression patterns of EGFR and associated pathways relative expression. **Research Question**: What proteins associated with downstream pathways are up or downregulated in MCF7 compared to the normal breast epithelium? **Methods**: The expression pipeline was applied to breast adenocarcinoma and healthy breast epithelium from publicly available short reads. This pipeline involved sequence alignment with a splice-aware aligner (HISAT2) and feature counting algorithm (featureCounts) and normalization/filtering/plotting with limma. Feature counts were plotted in a log of counts per million with heteroscedasticity adjustment (voom). **Results**: Transcriptomic expressions trended in favor of under-expression, with the log fold change in gene expression of EGFR being nuanced among multiple samples. **Discussion:** The breast ductal carcinoma cells show a general trend towards under-expression. However, the expression of EGFR, being comparable to healthy breast epithelium, remains nuanced. This suggests EGFR isoforms may indeed be useful for possible detection of disease states.

B-20. Identifying Factors that Regulate miRNA Strand Selection in *Caenorhabditis elegans*
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Regulation of gene expression allows cells to maintain normal cellular structure and function. microRNAs (miRNAs) are small, non-coding RNAs that are key regulators of gene expression. Typically, miRNAs base-pair to specific mRNA transcripts and repress their expression. During miRNA biogenesis, miRNAs are initially formed as double-stranded precursors that are enzymatically processed into a double-stranded duplex. One strand is loaded into an Argonaute protein and becomes functional, while the other stand is degraded. As each strand is expected to have different mRNA targets, strand selection determines which genes are regulated by miRNAs. Abnormal strand selection is often observed in human diseases, although it is not clear what factors lead to dysregulation of strand selection. Ongoing work from our lab has determined that 3' nucleotide identity influences miRNA strand selection in the nematode *Caenorhabditis elegans*. However, it is unclear how the 3' nucleotide is recognized during miRNA strand selection. We hypothesize that factors influencing strand selection interact with factors important for miRNA biogenesis, such as Argonaute. To identify regulators of miRNA strand selection, we will deplete the levels of candidate proteins using RNAi. We will then quantify the relative levels of each miRNA strand using quantitative PCR assays to determine whether strand selection was affected. Identifying factors that regulate miRNA strand selection will be important towards understanding how miRNA strand selection is dysregulated in human disease.

B-21. Optimizing *C. elegans* for high throughput chemical screening
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This study explores RNAi strategies to enhance membrane permeability for improved high-throughput drug screening in *Caenorhabditis elegans*. As a small, genetically tractable multicellular animal, *C. elegans* is an excellent system for compound library screening; however, the cuticle poses a formidable barrier for many chemicals. Traditional chemical library screens rely on ingestion to deliver drugs to internal cells in the animal, limiting cellular exposure to the drug. We are undertaking a new approach to drug delivery by using RNAi to reduce cuticle integrity gene expression. *The C. elegans* system facilitates large-scale RNAi experiments, affecting treated animals and their progeny. *C. elegans* ingest bacteria as a food source, which can easily be manipulated to express gene-targeted double-stranded RNA. This bacterial-mediated feeding protocol for dsRNA delivery is used to reduce the level of cuticular proteins and lipids. Permeability will be assessed by the time to lethality for small molecules (e.g., bleach, boric acid) in treated versus untreated animals. We will also extend this approach using small molecule dyes and larger fluorescent nanoparticles to determine the extent of permeability. To obtain reproducible breaches in cuticle integrity in live animals, we will manipulate the environment using osmotic agents and physical forces. The goal is to devise a protocol that reproducibly produces a large population of worms that are permeabilized but remain viable and fertile. This experiment's results will improve the efficiency of toxicity assessments and establish a pathway for the entry of larger molecules to penetrate the cuticle barrier.

B-22. CDKs-1 and -2 Enhance HSV-1 Immediate-Early Gene Expression
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Cyclin-dependent kinases (CDKs) are a group of cellular kinases that modulate, in part, the host cell cycle. Herpes simplex virus 1 (HSV-1) is a large DNA virus that requires host factors for replication. Specific CDKs have been reported to interact with HSV-1 proteins. Previous research showed that inhibition of CDK-1 and CDK-2, greatly reduces immediate-early (IE) gene expression and viral titers 24 hours post infection (hpi) by 100- to 1,000-fold. Published reports have also linked the activities of CDKs-1 and -2 to host RNA polymerase II (RNAPII). We hypothesized that CDKs -1 and -2 are required for HSV-1 IE gene expression by interacting with host RNAPII. We used specific CDK -1 or -2 inhibitors and examined IE mRNA and protein levels at 4 hpi. IE transcript levels for ICP0, ICP4, and ICP27 and protein levels for ICP0 and ICP4 were appreciably reduced upon inhibition of CDK-1 or -2. We then performed phosphoproteomics to determine if the phosphorylation of cellular factors associated with transcription, were altered upon inhibition of CDK-1. Our results show sites of phosphorylation on RNAPII were reduced upon CDK-1 inhibition. We are currently determining if the binding of RNAPII is diminished at HSV-1 IE genes when CDK-1 is inhibited by performing chromatin immunoprecipitation (ChIP). Future studies will focus on validating our results with RNAPII and other cellular targets for CDK-1 and performing phosphoproteomics with a CDK-2 inhibitor. Overall, our results indicate that the activities of CDK-1 and CDK-2 regulate HSV-1 during lytic infection by stimulating IE gene expression.

B-23. What's Linker Have to Do With It? Examining the Structure and Stability of Palladin's Ig3-4 Linker Region
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The protein actin is integral to movement and cytoskeleton function within the human body. Actin participates in more protein-protein interactions than any other known protein and one such relationship involves palladin. Palladin is a lesser-known binding protein that is required for embryonic development. Palladin is also associated with increased expression in metastatic cancer cells.
To understand the role of palladin in cancer metastasis, we first must understand its structure. Palladin is comprised of five immunoglobulin-like domains (Ig), each connected via an unstructured linker region. Previous research has proven that Ig3 is the minimal actin-binding domain, however, binding affinity is significantly increased when the Ig3-4 linker domain is present. To examine the effects of this Ig3-4 linker on overall actin binding, the Beck lab introduced several mutations. These mutations include DELinkerA, QELinkerA, RLinkerA, and a scrambled linker sequence. The most prominent mutation was RLinkerA, as the conversion of the linker region's ten arginines to alanines completely disrupted the binding ability of both actin and palladin. Our current research seeks to understand the structural and stability changes that occur when binding affinity is disrupted within palladin's Ig3-4 linker region using circular dichroism and fluorescence spectroscopy.

B-24. Comparing the efficacy of hand brushes versus electric brushes on teeth and evaluating the whitening power of two different kinds of toothpaste.
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Department: Physical Science.

Toothpaste serves a dual function in the cleaning and whitening of discolored teeth, employing both chemical and mechanical mechanisms. Generally, commercial toothpastes incorporate peroxide compounds, such as hydrogen peroxide, which are known for their bleaching properties, along with abrasive agents that facilitate the physical removal of surface stains. The efficacy of these whitening agents can significantly vary based on their concentration within the formulation, leading to differing results in terms of tooth discoloration removal. Furthermore, the degree of discoloration present on the teeth is influenced by various factors, including dietary habits, oral hygiene practices, and the intrinsic properties of the dental enamel itself. This study seeks to investigate the comparative whitening effects of different types of toothbrushes when used in conjunction with the same toothpaste formulation, as well as to explore the impact of varying toothpaste formulations when applied with a consistent toothbrush type. By systematically examining these variables, the research aims to provide insights into the optimal combinations of toothbrush and toothpaste that maximize whitening effects, thereby contributing to more effective oral care regimens.

B-25. Role of Beta-2 Adrenergic Receptors in Innate Immune Response to Intracellular Bacterial Infection.
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Burkholderia thailandensis is an opportunistic intracellular bacterium that can cause pneumonia. Due to its similarities in pathogenic mechanisms to *Burkholderia psuedomallei*, a more virulent species, it can serve as a model to better understand their intracellular survival in host immune cells. The bacterium evades phagolysosome formation, allowing it to proliferate within macrophages. This intracellular survival leads to the formation of multinucleated giant cells (MNGCs), making the infection antibiotic resistant. The lungs are densely innervated with sympathetic neurons responsible for maintaining homeostatsis through the release of noradrenaline (NA). Resident innate immune cells express b-adrenergic receptors (ADRB), but the immunomodulatory role of the sympathetic neurons during *B. thailandensis* infection is unknown. This project utilizes both *in vitro* and *in vivo* approaches to explore the role of the NA-ADRB signaling pathway during *B. thailandensis* infection. We performed intracellular killing assays by coculturing bone marrow-derived macrophages (BMDMs) and *B. thailandensis* and tested the effects of NA, ADRB-2 short-acting agonist (albuterol), and ADRB-2 long-acting agonist (Salmeterol). All three treatments had significant effects on the intracellular killing abilities of BMDMs. Additional assays were conducted with ADRB2 knockout (ADRB-/-) BMDMs, which showed significantly enhanced intracellular survival of *B. thailandensis*. For *in vivo* experiments, we infected control and ADRB2-/- mice intranasally with as sub-lethal dose of *B. thailandensis.* Then measured core body temperature and bacterial load after 24 hours. The ADRB2-/- mice exhibited a more severe drop in temperature. This data suggests that b,2-adrenergic receptor signaling may play a critical role in host defense against *B. thailandensis* infection.

B-26. RNA Interference of TOR1A, CREB3, HSP90B1, and HSA1L genes in *Acyrthosiphon pisum*
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Acyrthosiphon pisum, more commonly known as pea aphids, are a pest to many species of Febaceae (legumes) mainly due to the species being prone to carrying Febaceae diseases. Protection against A. pisum currently includes insecticides and natural predators, both of which bring potential negative effects to other organisms in the surrounding area. In this study, the use of RNA interference (RNAi) provides an alternative and species-specific elimination of A. pisum. The targeted genes in this study, HSPA1L, HSP90B1, HSA1L, and TOR1A, are involved with the unfolded protein response (UPR) which includes the stabilization of existing proteins, mediation of folding the newly translated proteins in the cytosol and organelles and catabolizing misfolded proteins. Targeting these genes could potentially result in an increase in improperly folded proteins in the cytosol and other organelles, which would eventually result in increased apoptosis (cell death) and death of the organism. In this study, RNA was isolated from A. pisum and reverse transcribed into cDNA. This cDNA was combined with HSPA1L and TOR1A primers to synthesize HSPA1L and TOR1A dsRNA that would be fed to multiple groups of A. pisum. In preliminary studies, this method has shown reasonable evidence of increased death rate of A. pisum.

B-27. Characterization of *AT5G16120*: A Putative Monoacylglycerol Lipase
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Lipases are enzymes that hydrolyze lipids. The substrates and products of these enzymes are diverse; the biological functions of lipases are just as varied. They are involved in processes ranging from seed oil composition remodeling to the breakdown of fats in food for nutrient absorption in the intestine. The gene product of *AT5G16120* has been annotated as a monoacylglycerol (MAG) lipase (Kim et al., 2017, Plant J., 85, 758). The authors first identified the AT5G16120 protein as likely having MAG-lipase activity because the modeled protein structure is similar to that of a known MAG lipase, and they named the protein AtMAGL15. Heterologous expression of AtMAGL15 in *E. coli* demonstrated MAG lipase activity for the enzyme, and, using confocal microscopy, AT5G16120 localizes to the cytosol in tobacco leaves. Lipid profiles of two independent T-DNA insertion mutants in *AT5G16120* showed a buildup of not only MAG but also of digalactosylmonoacylglycerol (DGMG), suggesting that DGMG may also be an AtMAGL15 substrate. We are currently characterizing the effect of the T-DNA insertions on *AT5G16120* mRNA levels in the mutants. We will also create complementation lines in the mutant lines to further validate that the mutations in *AT5G16120* are responsible for the lipid phenotypes we observed. Because the gene expression level of *AT5G16120* is highly upregulated under osmotic stress, we hypothesize that AT5G16120 is involved in the osmotic-stress response and that the mutants will not adjust to the stress as well as the wild type plants. We also plan to test this hypothesis.

B-28. Carbon sequestration across ecological and urban gradients in Kansas
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Carbon sequestration is the process of capturing and storing carbon dioxide from the atmosphere into photosynthetic organisms. Beyond carbon sequestration, trees provide many ecosystem services, particularly in urban areas, such as cleaning the air and water; cooling temperatures, preventing pollution and soil erosion, and providing wildlife habitats. Our research objective was to determine how carbon sequestration differs across 1) a forest - grassland ecological gradient, and 2) urban areas in Kansas. We focused our efforts to 17 Kansas cities with populations of at least 1,000 people along the I-70 corridor to maintain longitudinal consistency. Using i-Tree, we downloaded carbon sequestration rates, common tree species, tree basal area, and developed land cover data for each city. We analyzed trends in carbon sequestration and urban features with a series of correlation analyses. The most common tree genera found in Kansas cities were elms (*Ulmus* spp.) and hackberry (*Celtis* spp.). We found that tree basal area was strongly correlated to carbon sequestration. Within our target cities, human population size was also positively correlated to carbon sequestration; however, developed land cover (low to high intensity) was surprisingly not correlated to carbon sequestration. Thus, we assumed that the relationship between carbon sequestration and urbanization (population density) is due to an east-west population and basal area gradient that corresponds to a forest-grassland ecotone in Kansas. Future research on this topic should explore which tree species have the greatest rates of carbon sequestration in cities, and the preferences of urban residents for those trees.

B-29. Developmental origins of aberrant neurological trajectories in Down syndrome

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Key inflammatory pathways are conserved across diverse neurodevelopmental and degenerative conditions, such as autism spectrum disorder (ASD), Alzheimer's disease (AD), and Down syndrome caused by trisomy 21 (T21). T21 represents the most common genetic cause of intellectual and developmental disability and can range from mild to severe. The distinct neurocognitive profile uniquely experienced across the lifespan by individuals with T21 also features an increased risk for ASD diagnosis. ASD affects approximately 1/3rd of people with T21 who additionally exhibit a complete penetrance of AD, though there are highly variable timelines of disease progression to dementia through largely unknown mechanisms. Emerging evidence strongly supports our hypothesis that overlapping networks of immunomodulatory cytokines shape lifelong neurocognitive profiles through microglia mediators with hyperactivation beginning early in neurodevelopment in human fetuses with T21 which corresponds to aberrant AD etiology in a mouse model of Down syndrome. This emerging dogma has been largely established with preclinical animal models and epidemiological clinical data, so we aimed to incorporate a novel translational pipeline to begin dissection of mechanisms underlying the developmental origins of aberrant neurological trajectories heterogeneously experienced by people with T21. To this end, we used both T21 and control disomic human induced pluripotent stem cells (hiPSCs), differentiated them into *yolk-sac* progenitors of microglia for exposure to titrated levels of the interferon family of immunomodulatory cytokines for assessment of karyotype-specific impacts by flow-cytometry, then sequentially into microglia for future study. Defining the immunomodulatory mechanisms that underly T21-associated neurodiversity would provide a foundation for personalized interventions.

B-30. Gene expression during the development of plastic somatic cells in the volvocine green algae

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One of the central questions in the study of cellular evolution has been how new cell types emerge. Genetic changes can lead to the evolution of novel cell types in multicellular species, but it remains unclear whether differentiation can be ancestrally plastic and then be stabilized. We are currently identifying how environmental stimuli can cause changes in a cell's state and cause it to turn into a different type of cell. To do this, we will use the volvocine green algae as a model system. Specifically, we will work with *Eudorina*, a species that was previously characterized as undifferentiated but which can develop plastic somatic cells after cold shock. We will be identifying and analyzing the mechanisms underlying this response. We will be subjecting *Eudorina sp. NIES 3984* to cold shock and then collecting and sequencing cells at important developmental stages every few hours for the span of two generations. We will be extracting RNA from the collected tissue and then carrying out RNA sequencing. To characterize the gene expression changes tied to the development of plastic somatic cells, we will use transcriptomics and investigate the genetic mechanisms. We will also be comparing this to other species within the volvocine green algae family who have somatic cells but different differentiation such as *Pleodorina* and *Volvox*. By combining phenotypic, developmental data, and transcriptomics, we aim to understand how gene interactions with the environmental conditions can lead to a new trait's emergence.

B-31. Zinc Hydroxide/Biotin/Gelatin Composite Particles Take Aim at Human Breast Cancer Cells

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Composite particles have emerged as a promising multifunctional platform for cancer therapy due to their biocompatibility, targeted delivery potential, and bioactivity. This study focuses on the synthesis and evaluation of zinc hydroxide/biotin/gelatin composite particles for the potential antimicrobial and targeted cancer treatment. Zinc hydroxide serves as an antimicrobial and therapeutic agent with intrinsic cytotoxic properties, while studies have shown that biotin, also known as vitamin B7, helps with the metabolic processes of cells. Some studies also showed that biotin can enhance tumor-targeting specificity through receptor-mediated uptake. On the other hand, gelatin, a natural biopolymer, was used as a stabilizing matrix, providing biocompatibility and controlled release properties.
In this study, synthesis process involved co-precipitation of zinc hydroxide within a biotin-functionalized gelatin matrix, forming microparticles. Characterization techniques included scanning electron microscopy (SEM), Fourier-transform infrared spectroscopy (FTIR), and X- ray diffraction (XRD), confirmed the structural integrity and functionalization of the composite particles. In vitro cytotoxicity assays using MCF-7 (breast cancer cell lines), demonstrated the enhanced efficacy of the composite particles in the following order: zinc hydroxide/biotin < zinc hydroxide < zinc hydroxide/gelatin < Zinc Hydroxide/Biotin/Gelatin, attributed to the synergistic effects of biotin targeting and gelatin-mediated controlled release.
Our composite particles were also tested against Staphylococcus aureus and revealed an excellent antibacterial property. These findings underscore the potential of zinc hydroxide/biotin/gelatin composite particles as an innovative therapeutic platform for targeted and efficient cancer treatment, paving the way for further preclinical and clinical investigations.

B-32. Metabolites derived from bacterial isolates of the human skin microbiome inhibit *Staphylococcus aureus* biofilm formation

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The human skin microbiome is a diverse microbial ecosystem with an established role in preventing host colonization by producing small molecules and peptides that inhibit the growth and virulence of human bacterial pathogens. *Staphylococcus aureus* is a major human pathogen responsible for human diseases ranging from acute skin and soft tissue infections to life-threatening septicemia. The ability of *S. aureus* to form biofilm on biotic and abiotic surfaces is a key virulence factor that favors its success as a pathogen and contributes to increased antimicrobial resistance. The lack of interventions and limited availability of effective antibiotics creates an urgent need to further explore the human skin microbiome for therapeutically viable compounds capable of combatting *S. aureus* infections. Here, we investigated the ability of bacterial skin commensals to produce molecules that inhibit *S. aureus* biofilm formation. Using MALDI-TOF analysis, 77 human skin microbiome bacterial isolates were identified from the *Staphylococcus* and *Bacillus* genera. Metabolites from cell-free concentrated media (CFCM) obtained from 26 representative isolates were evaluated for the ability to reduce biofilm formation by both methicillin-resistant and methicillin-sensitive *S. aureus* strains in 96-well plate biofilm inhibition assays. CFCM derived from the majority of our bacterial isolates demonstrated significant inhibition of biofilm formation to varying extents, while not affecting planktonic growth. These findings indicate that several bacterial constituents of the human skin microbiome may serve as novel sources of therapeutic agents against skin infections. Further research into these commensal bacteria and their biofilm inhibitory mechanisms could lead to innovative treatments targeting antibiotic-resistant infections.

B-33. Study of ASF1a Protein's Biological Significance in Arabidopsis thaliana via Phenotypic Manifestations of CRISPR/CAS Induced A. thaliana ASF1a 1081 Gene Mutagenesis

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Anti-silencing function 1A (ASF1a) is a well-characterized histone chaperone involved in critical biological processes across diverse organisms, including *Homo sapiens*, *Drosophila melanogaster*, and *Saccharomyces cerevisiae*. ASF1a regulates DNA replication through its interaction with the H3/H4 histone complex and plays a key role in DNA damage response and recovery by NHEJ. In humans, ASF1a also facilitates gene silencing by interacting with HIRA to promote the formation of SAHF. This study aims to investigate the role of ASF1a in *A. thaliana* by employing CRISPR/Cas9-mediated site-specific mutagenesis of the ASF1a 1081 gene. We hypothesize that induced mutations in this gene will result in leaf morphological alterations. At this stage, we successfully generated pDe-CAS9 constructs, which will be integrated into *A. thaliana* through *Agrobacterium tumefaciens* subsequently. Our goal is to provide valuable insights into the genetic mechanisms underlying plant dedifferentiation, a process critical for tissue regeneration and damage repair. Additionally, understanding ASF1a's role in *A. thaliana* could inform strategies for enhancing crop yield and resilience in economically significant species, such as *Brassica.*

B-34. Synthesis of Sterically Hindered Catechol Ligands for Hydrophobic Anti-Cancer Vanadium Complexes

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Hydrophobic vanadium complexes have recently shown greater anti-cancer activities than cisplatin, the gold standard of cancer chemotherapy. In the cell, vanadium ions oxidize to form vanadate (VO_4^{3-}), a known inhibitor of phosphatase enzymes. Vanadium complexes are used to treat diabetes to deliver vanadate into cells. Studies have shown that vanadium complexes must be sufficiently hydrophobic to prevent hydrolysis of the complex to ensure the release of vanadate within the cell. Recently, two vanadium complexes, [VO(HSHED)(cat)] and [VO(HSHED)(dtb)] ([Hshed= *N*-(salicylideneaminato)-*N'*-(2-hydroxyethyl)-1,2-ethanediamine, cat= catechol, and dtb= 3,5-di(tert-butyl)catechol)]) were evaluated against cisplatin for their anti-cancer activity. [VO(HSHED)(dtb)] demonstrated greater anti-cancer activity than both [VO(HSHED)(cat)] and cisplatin. The increased hydrophobic character of the (dtb) derivative is thought to be responsible for its increased anti-cancer activity. Thus, the hydrophobic properties of [VO(HSHED)(dtb)] have inspired the development of a library of hydrophobic ligands for vanadium complexes to evaluate their anti-cancer and physical properties. We aim to synthesize hydrophobic catecholate ligands as part of an international collaboration with the Crans/Lay team from Colorado State University and the University of Sydney, respectively. Specifically, our lab will prepare bulky catechol ligands through the alkylation of catechol with chiral terpene substituents and the synthesis of catechol-cycloalkane fused rings systems. Our approach and progress will be described.

B-35. The Synthetic Chemical Biology Core (SCB): A Resource for Research in Chemical Biology

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The Synthetic Chemical Biology Core strives to provide comprehensive synthetic chemistry capabilities to investigators under one roof. The synthetic expertise of the core includes, but is not limited to, novel and commercially unavailable small molecules, fluorescent molecules and custom peptides. The core assists in identifying hits for medicinal chemistry optimization in infectious disease targets and provides synthesis capabilities for structure activity studies of said hits. The core staff will work with investigators to design and synthesis novel molecular probes to facilitate their research. SCB core provides purification, analysis and quality control of compounds via LC/MS. The SCB core also offers MALDI-TOF analysis of biomolecules.

C-1. Unveiling the Architecture of hnRNP C Proteins Through AI-Powered Prediction

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Heterogeneous nuclear ribonucleoproteins (hnRNPs) are very abundant proteins associated with mediating the transformation of nuclear pre-mRNA into mature cytosolic mRNA. One of the most abundant hnRNP proteins within this group are the hnRNP C proteins which consist of two differentially spliced isoforms, C1 and C2 (C proteins). Two additional proteins, hRaly and hRalyl have been theorized to belong to the C protein family based upon primary sequence homology with the C proteins. A variety of functions have been assigned to the C proteins including their involvement in mRNA splicing, RNA polyadenylation, regulation of mRNA stability, mediating internal ribosome entry site translation, as well as being a component of the telomerase holoenzyme. The amino terminal domain of hnRNP proteins contains a canonical RNA recognition motif (RRM), followed by a basic region that precedes a leucine zipper and terminates with a highly acidic carboxy terminus. The structure of the C protein's amino terminal RRM has been solved, and its structure is consistent with the βαββαβ structure observed in RRMs of other RNA binding proteins. However, recombinant C1 and C2 expressed in *Escherichia coli* form stable homo-tetramers presumably mediated by the leucine zipper. Structural investigation of the C proteins, hRaly, and hRalyl outlined here, using AlphaFold2, has provided us with insight into the functional relevance of having four RRMs arrayed in a tetrameric context. These data also confirm the structural redundancy between the C proteins and hRaly/hRalyl.

C-2. Exercise-Induced Lactate as an Alternative Energy Source in Alzheimer's Disease

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The brain is a highly energy-dependent organ, requiring lots of energy to support neuronal signaling, synaptic plasticity, and cognitive processes. As aging progresses maintaining energy homeostasis is important for proper cognitive function and mitigation of neurodegenerative brain diseases such as Alzheimer's Disease. Lactate is produced during exercise and was previously thought to be a metabolic waste product. However, further research has indicated that lactate is used as an alternative energy substrate in the brain and does so through the astrocyte-neuron lactate shuttle. In this study we plan to evaluate the role of Lactate as an alternative energy substrate in a transgenic model of Alzheimer's Disease in rats. Twelve-Month-old TgF344-AD rats were randomly stratified into either sedentary or exercise groups. Rats were treadmill trained for 5 days a week for 6 months. After the exercise protocol, brain tissue, skeletal muscle, and blood serum were collected for protein analysis. Using ELIZA and western blot we expect Lactate Dehydrogenase, MCT2, and MCT4 to be increased in the hippocampus and cortex in the exercise group ($p < 0.05$). Our results will show whether the astrocyte-neuron lactate shuttle is utilized in an Alzheimer's Disease model. This research will further the understanding of brain metabolism in Alzheimer's Disease pathology.

C-3. The Link Between Cardiovascular Function and Life-Threatening Diseases: A model to understand cardiac dysfunction
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Cardiovascular dysfunction contributes to more than 20% of all causes of death. Globally, cardiovascular disease leads all causes of death. Diseases affecting the heart lead to cardiac dysfunction, which is the inability of the heart to eject sufficient O2-carrying blood throughout the body in order to sustain increases in metabolic demand. Breast cancer (BC), pulmonary hypertension (PH), and heart failure with reduced ejection fraction (HFrEF) are related to cardiac dysfunction and impaired systemic O2 transport. These three diseases represent the leading cause of death amongst women nationwide and using animal models of BC, HFrEF, and PH. We can preclude the effect of diet, nutrition, and exercise and investigate the direct relationship between these diseases alone and cardiac dysfunction. We hypothesize that HFrEF will induce cardiac dysfunction in comparison to BC and PH.

C-4. Impact of Microgravity on Ovarian Follicle Counts in Mice
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The impact of microgravity during spaceflight is a prevalent concern for astronauts as it pertains to reproductive function. To determine if spaceflight and microgravity affect the ovarian follicular development, four experimental groups composed of ~15-week-old mice were euthanized and frozen following 28/29 days of spaceflight (FLT mice) on Rodent Research 10 mission, similarly handled Habitat Ground Control (HGC), Baseline, and Vivarium control mice were also processed. Upon thaw ovaries were dissected, embedded in paraffin, serial cross sectioned, and H&E stained to prepare for microscopic analysis. Follicles and corpora lutea were quantified on every fifth section at 10x and 40x magnification. Follicles were categorized based upon their developmental stage: primordial, primary, secondary, tertiary, early antral, and antral. Overall, primordial follicle numbers between HGC and FLT groups were not different. Whereas FLT mice had a greater number of developing primary, secondary, tertiary, and antral follicles. Count differences in early antral and corpora lutea groups were not statistically significant. Ongoing analyses with Vivarium and Baseline groups are in progress. In conclusion, it appears that spaceflight/ microgravity elevated the progression of ovarian follicular development, while not impacting the overall pool of primordial follicles. As early follicular development, the transition of primordial and growth to tertiary follicles, occurs in the absence of pituitary support, we could interpret that microgravity enhances this aspect of follicle development, which is currently not well understood. The ultimate impact of this could be an early loss of ovarian follicular reserve and ultimately a shortened reproductive lifespan.

C-5. CRISPR Analysis of HIRA in Plants
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The purpose of this study is to understand how *Histone Repressor A (HIRA)* functions in *Arabidopsis* by creating mutations via CRISPR. HIRA is a chromatin-remodeling protein essential for proper development in both plants and animals. In animals, HIRA interacts with Anti-Silencing Function 1 (ASF1) to assemble chromatin. During early animal development, this ASF1-HIRA complex silences early development genes by tightly packaging them into heterochromatin. Because development is fundamentally different in plants and animals, it can't be assumed that HIRA functions preciously the same in both. In plants, *HIRA* is known to be involved in regulating genes that promote cellular differentiation. HIRA interacts with a myb domain transcription factor which represses the *KNOTTED-like homeobox (KNOX)* genes during leaf development. We are using CRISPR to make mutations in the *Arabidopsis HIRA* gene. Phenotypic and molecular analysis of CRISPR mutant plants will provide information about what regions of the HIRA protein are critical for proper function. So far, we have successfully used CRISPR to transform a CAS9 vector into plants. The seeds from the initial transformants have been harvested and plated on media. This allows germination when the CRISPR tools are present in the seed. We are now working to determine if any of our T1 and T2 plants have phenotypes consistent with the involvement of HIRA in leaf development.

C-6. Transcriptomic Analysis of EGFR and Downstream Pathway Expression in A549 and Healthy Lung Epithelium
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Background: Clinical tests utilizing EGFR isoforms for cancer screening, primarily for lung, breast, and ovarian, have shown uncertain specificity and sensitivity. We have interest in intronic SNPs in intronic regions. This work is an Insilco characterization of the lung adenocarcinoma cell line (A549), focusing on EGFR expression patterns and related pathways.
Research Question: What associated proteins from downstream pathways are up- or down regulated in A549 compared to normal lung epithelium. What intronic SNPs in the EGFR coding sequence may affect splicing.
Methods: The expression pipeline was applied to lung adenocarcinoma and healthy lung epithelium from publicly available cDNA short reads. This pipeline involved sequence alignment with a splice aware aligner (HISAT2) and feature counting algorithm (featureCounts) and normalization/filtering/plotting with limma. Feature counts were plotted in a log of counts per million with heteroscedasticity adjustment (voom). The SNP pipeline applied typical genomic aligner (BWA) using GRCh38, Variant calling (Varscan), and Mpileup were used and annotated with vcf2maf. Variance in the coding sequence were plotted with the use of Gviz.
Results: Transcriptomic expressions trended in favor of under expression, with the log fold change in gene expression of EGFR being statistically insignificant. SNP mutations have been identified with the mutation, c.2625+196A>G seen as phenotypically benign.
Discussion: Lung adenocarcinoma cells show a general trend towards under expression. However, the consistent expression of EGFR, comparable to that of healthy lung epithelium, remain distinctive. The stability highlight's EGFR's potential for possible detection of different isoforms.

C-7. Patterns of brain Ferritin expression in the Drosophila divalent cation transporter mutant *Malvolio*.

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Malvolio (Mvl) is the Drosophila ortholog of the mammalian Solute Carrier Protein Slc11a2, which transports divalent metals, including iron. The function of Mvl in the developing Drosophila brain is unclear and the developmental anomalies of the brain in *Mvl* mutant, if any, have not been investigated. Our objective was to determine potential physiological defects, if any, in the brain of *Mvl* mutants. We tested iron availability in the brain of *Mvl* loss-of-function mutant, Mvl^{exc1}. Brain tissue from both the control and mutant animals were dissected and the Ferritin GFP levels at both the larval and adult stages were recorded. Ferritin 1 GFP intensity was used as the marker of iron availability for comparison between the mutants and controls. We confirmed that the loss of Mvl results in lack of iron storage in the midgut iron cells. Contrary to our expectation, we observed differential and sharply contrasting regions of Ferritin expression in the *Mvl* mutant brains compared to controls. The optic lobes expressed high levels of Ferritin (high GFP) in the *Mvl* mutants compared to the central brain lobe in a pattern that persisted during both larval and adult stages. The finding that *Mvl* mutant brain tissue (optic lobe) has higher Ferritin expression compared to the control suggests one or more of the following scenarios: (i) Despite the loss of Mvl, brain tissue can access iron, via non-Mvl dependent cellular uptake of iron, and/or (ii) Ferritin expression in brain tissue is uncoupled from cellular iron availability.

C-8. Passive rates of heating and cooling in a paper wasp from Mediterranean scrub habitat

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Bees are some of the most well-known pollinators in the world, they are also the most efficient pollinators in the world. The bulk of research involving heat tolerance and the effects of climate change have gone towards bees, as it should. However, with the field expanding to include areas such as critical thermal max, it is important that we include other pollinators into the overall picture we are starting to uncover. Through first-hand observations, the presence of *Vespa orientalis* and *Megascolia maculata* was noticeably lower than in years prior. This observation coupled with the heat wave that was present during our time on Lesvos brought into question how the other pollinators were dealing with the heat. Due to time constraints and some technical difficulties, I focused my efforts on observing the rate of heating on a common and plentiful species *Polistes dominula*, the European Paper Wasp. The overall results showed that there was no impact on passive thermal loss or gain in relation to body size. My hope with starting to look at other pollinators abilities to live with heat is that the research will expand into other areas. Climate change causing rising temperatures will most likely cause changes in behaviors that will affect others beyond bees. Ultimately, these changing interactions can be another form of disruption to bees that must be taken into account.

C-9. Investigating the Molecular Function of the *Arabidopsis* AS2 Gene

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This study aims to better understand the role of the *ASYMMETRIC LEAVES 2 (AS2)* gene in *Arabidopsis.* The *AS2* gene encodes a protein that regulates the *KNOX (KNOTTED-LIKE HOMEOBOX)* genes, which function to keep cells in an undifferentiated state. During leaf development, the AS2 protein suppresses the *KNOX* genes, allowing for normal leaf development. If AS2 fails to inactivate the *KNOX* genes during leaf development, cells remain undifferentiated and leaves do not develop properly. However, we still know very little about how AS2 functions to keep the *KNOX* genes turned off. To investigate, we use CRISPR technology to create mutations in the *AS2* gene that will alter its protein structure and function, potentially causing mutations that change leaf shape and development. By studying these mutations, we can uncover the mechanisms AS2 employs to turn off *KNOX* genes, improving our understanding of genetic regulation in plant development.

C-10/ The functional regulation of protein-based nanofiber bioscaffolds on human astrocyte for neural regeneration

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Nanofiber scaffolds showed promising applications in tissue regeneration and tissue engineering. Because their structure closely resembles the extracellular matrix (ECM) morphology, nanofiber scaffolds can be grafted in wounded neural tissues, thus enabling neural regeneration. Collagen is extracellular matrix molecule and soy protein can potentially modulate neural immune activity. Nanofiber scaffolds fabricated from collagen and soy protein may enhance the neural repair process. Studies reported that transplantation of fetal astrocytes may stimulate axonal regeneration and functional recover after spinal cord injury. After neural injury, different from adult astrocytes, fetal astrocytes can potentially create a more conducive environment for regeneration. Additionally, fetal astrocytes may produce factors for neuron protection and therefore promote neuron survival. In this study, we fabricated protein and polymer composite nanofibers by electrospinning and tested the fetal astrocyte response to the fibers. Soy protein isolate (SPI)-polycaprolactone (PCL), collagen-PCL and SPI-collagen-PCL nanofibers were generated and characterized by fourier transform infrared (FTIR) test to confirm the fiber component. We found astrocytes showed high viability on all the fibers in a LIVE/DEAD assay. However, SPI can reduce the astrocyte proliferation compared with collagen-based fibers. Flowcytometry test showed that the fiber component did not affect cell cycle. Time-lapse recording showed the dynamic migration of fetal astrocytes on those fibers. Our further study of RNA gene sequencing will explore the molecular mechanism that modulates the cellular function. The study demonstrated the potential of these protein and PCL copolymer nanofibers in the application of neural regeneration.

C-11. Using FRET to Assess Conjugate Binding of Anthrax Toxin's PA and Antigen Spy0469
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Anthrax toxin is an AB toxin, requiring the enzymatic A components edema factor (EF) and lethal factor (LF), and the B component protective antigen (PA) to successfully infiltrate and spread infection within the host organism. PA binds with high affinity to capillary morphogenesis protein 2 (CMG2). Previous studies in mice have shown that injection of a combination of LF and PA elicited a profound debilitation in the ability of dendritic cells to secrete cytokines. We hypothesized that CMG2 would be present on human dendritic cells, and would facilitate binding of PA. Binding of PA would allow uptake, and potentially digestion and presentation on the dendritic cell surface to activate CD4+ T cells. To test this, we have created a conjugate between PA and Spy0469, a surface antigen of *S. pyogenes* that has a relatively high (55%) frequency of T-cell responders in the human population. In preliminary studies in human PBMCs, we observed CD4+ and CD8 cytokine T cell responses toward Spy0469 and PA-Spy0469. To confirm our conjugate can still bind CMG2, we utilized a gel-shift assay to monitor binding, and plan to also assess binding affinity through FRET usage. We have created a mutant of PA and PA-Spy0469 (E733C) and plan to label with the maleimide activated probe AF488. CMG2 R40C will be labeled with AF546 and can establish binding between PA-Spy0469 and CMG2 through the use of FRET. Further, we can assess specificity of binding using a monoclonal antibody to CMG2 to prevent binding of the conjugate to CMG2.

C-12. Variance of Cancer Mortality in the United States
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Disparities in cancer mortality persist among racial and ethnic minority groups. Our research endeavor was to investigate cancer mortality across diverse demographic groups, encompassing men and women. The study utilized the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Explorer dataset, which is a comprehensive compilation from population-based cancer registries covering approximately 48% of the U.S. population. I generated datasets for mortality cancers among Hispanic, Black, and Asian/Pacific Islanders, females, ages 15-39 from (2017-2022) and from (2000-2022). The research covered a range of 26 cancer types affecting diverse anatomical sites, including anus, bones/joints, brain, breast, colon/rectum, cervix uteri, esophagus, kidney/renal pelvis, larynx ,leukemia, liver/bile duct, lung/bronchus, melanoma, myeloma, non-Hodgkin lymphoma, oral cavity/pharynx, ovary, pancreas, small intestine, soft tissue including hard, stomach, thyroid, urinary bladder, vagina, and vulva. The significance of our research was to investigate disparities in cancer mortality among minority groups. Our findings highlight the urgent need for targeted interventions to address cancer disparities among young women of color. Further research is necessary to identify risk factors to include geographic proximity to nuclear waste dumps, landfills, mining, and fracking sites; socioeconomics; access to health care and cancer screening; generational occupations; and smoking habits. By sharing this critical information, we can work towards developing and implementing effective prevention and early detection strategies, to these specific populations. This collaborative effort will empower individuals, strengthen healthcare systems, and improve public health outcomes.

C-13. Understanding the Role of Mutant KRAS in Colorectal Cancer Metastasis
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In the United States, colorectal cancer (CRC) is the third-most diagnosed and second-most lethal cancer for men and women combined. When CRC is localized, patients have a predicted five-year survival rate of 90%. However, when CRC metastasizes, this predicted survival drops to 14%. The epithelial-to-mesenchymal transition (EMT) is a cellular transformation that enhances cell motility and enables metastasis. This project aims to understand the role of a common CRC mutation, Kirsten Rat Sarcoma (KRAS), in EMT reprogramming. To accomplish this, we generated a stable, KRAS G12D-transformed cell line using rat intestinal epithelial (RIE-1) cells. In our KRAS-mutants, we observed an elongated, layered phenotype suggesting EMT reprogramming had occurred. Through western blotting and functional assays, we demonstrated that KRAS-mutants differentially expressed EMT-related proteins E-cadherin and vimentin and had increased invasion and migration capabilities. We then treated KRAS-mutants with a selective KRAS-G12D inhibitor. After treatment, KRAS-mutants demonstrated WT phenotype recovery, decreased migration and invasion capabilities, and 95% and 86% recoveries of WT E-cadherin and vimentin expression, respectively. This data supports the hypothesis that KRAS mutations are implicated in EMT reprogramming and, ultimately, CRC metastasis. The next step of this project is to identify the cell signaling pathways affected by mutant KRAS that precipitate the EMT. To do so, RNA expression will be explored with next-generation RNA Sequencing and RT-qPCR. Data from this project will be used to further understanding of the cellular processes mediated by mutant KRAS that promote EMT reprogramming. This will help identify how specific KRAS inhibitors can be employed to prevent metastasis.

C-14. Analysis of Ibuprofen and Aspirin in Artificial Urine samples Using RP-HPLC-UV/Vis with A New QuEChERS Approach
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Ibuprofen, a nonsteroidal anti-inflammatory drug (NSAID), is commonly administered for pain relief, fever reduction, and inflammation management. Aspirin, another NSAID, serves a dual purpose, preventing blood platelet aggregation and reducing the risk of heart attacks when administered in low doses. However, a critical concern arises when people take both ibuprofen and low-dose aspirin simultaneously, either on purpose or by mistake. While these medications are beneficial individually, their combined use can lead to unintended consequences. Specifically, ibuprofen has the potential to interfere with the cardioprotective effects of low-dose aspirin, leaving patients vulnerable to heart attacks. Thus, it's important to be able to separate and analyze these two drugs and their concentrations in urine in case people take both drugs. There has been a lack for a quick, easy, cheap, effective, rugged, and safe (QuEChERS) analysis approach for analyzing both drugs in urine sample. To address this issue, our research aims to achieve the following objectives: Goal 1: To develop a quicker detection method for detection of ibuprofen and aspirin in artificial urine samples using HPLC with a new QuEChERS approach. Goal 2: To assess the short-term and long-term stability of ibuprofen and aspirin in artificial urine samples. To achieve quick and easy analysis of ibuprofen and aspirin, standards and samples will be directly run on the HPLC after extraction to minimize the sample preparation process. Standards will be analyzed to construct the calibration curves, which will be used to determine the concentrations of each drug in each sample.

C-15. Mechanisms of quorum sensing-dependent inter-species communication in the soil bacterium *Chromobacterium subtsugae*
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Many soil bacteria use quorum sensing (QS), a type of population density-dependent cell-cell communication, to coordinate behaviors important for competition with other species. A type of QS involves production and response to acyl-homoserine lactone (AHL) signals. AHLs specifically interact with cognate LuxR-family transcription factors and cause them to activate transcription of specific genes by binding a specific DNA sequence in the gene promoter. Some LuxR-type AHL receptors have relaxed specificity and are responsive to non-cognate AHLs. These promiscuous receptors might be used to sense and respond to AHLs produced by nearby competitors by eavesdropping. We are interested in understanding the role of eavesdropping during interspecies competition. The soil saprophyte *Chromobacterium subtsugae* has a single AHL circuit that produces and responds to *N*-hexanoyl-HSL (C6-HSL) via the LuxR-type receptor CviR. CviR is promiscuous and can respond to other AHLs such as *N*-octanoyl-HSL (C8-HSL). Here, we use transcriptomics to show that C6-HSL and C8-HSL activate distinct and overlapping regulons in *C. subtsugae*. CviR and either signal is sufficient to activate two of these gene promoters, *vioA* and *chiA*, in the heterologous host *Pseudomonas putida*. We show the *vioA* promoter responds more sensitively to C6-HSL whereas the *chiA* promoter has better response to C8-HSL. The results suggest that promoter selectivity of CviR might be dependent on the particular AHL interaction. Through these studies, we will better understand the role of QS in interspecies cross-talk and the mechanisms of eavesdropping.

C-16. Role of asparagine synthetase and other transcription factor targets in epidermal morphogenesis
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Asparagine synthetase is an enzyme responsible for generating asparagine and glutamine from aspartate and glutamate. Expression of this enzyme is tightly controlled in both plant and animal tissues. Mutations in the human *ASNS* gene lead to asparagine synthetase deficiency and neurological pathologies. Additional evidence supports a regulatory function for *ASNS* in several cancers. In the plant model *Arabidopsis*, asparagine synthetase controls nitrogen distribution throughout development. An RNA-sequencing experiment in our laboratory led to the discovery that *ASPARAGINE SYNTHETASE1/DARK INDUCIBILE6* (*ASN1/DIN6*) is upregulated in leaves displaying an abnormal “curly leaf” phenotype due to overexpression of the homeodomain leucine-zipper (HD-Zip) transcription factor GLABRA2 (GL2). In addition to *ASN1*, another dark-inducible gene, *DIN1*, was found to be upregulated in curly leaves. HD-Zip transcription factors are involved in cellular events ranging from stress responses to regulation of morphogenesis. To investigate the role of *ASN1* expression in HD-Zip mutants, we performed RNA extractions from *pdf2-4, gl2-5*, and wild-type leaves for reverse transcription-quantitative PCR (RT-qPCR) analysis. RT-qPCR experiments were additionally performed to validate the elevated expression of *ASN1* and *DIN1* in curly leaves. To address whether *ASN1* and/or *DIN1* are required for the curly leaf phenotype, *asn1* and *din1* T-DNA insertion mutants were acquired, and the mutant plants were genetically crossed to curly leaf plants. Understanding the function and role of asparagine synthetase and other light-regulated genes in plants may facilitate further understanding of the importance of similar genes in human cells.
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C-17. Structure Guided Design of Broad-Spectrum Inhibitors of Coronavirus 3CL Proteases
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Periodic transmissions of β-coronaviruses (SARS-CoV, MERS-CoV, and SARS-CoV-2) from animals to humans underscore the need to devise rapid response strategies that can be cost-effectively deployed to minimize the pervasive harmful effects on global health. Commonly originating from bats, zoonotic β-coronaviruses from the Merbecovirus and Sarbecovirus subgenera have shown to be particularly dangerous to humans. The evolution of new viral variants remains a significant concern, particularly the potential emergence of pathogenic strains displaying high mortality rates, like MERS-CoV (~30%). With each new virus and variant, millions of dollars have historically been invested in the development of effective treatments; however, each year a therapeutic remains in development, thousands of deaths occur. Although β-coronaviruses and their variants have shown to have similar active site topographies, a broad-spectrum antiviral treatment remains elusive, and numerous β-coronaviruses lack established therapeutics. Our research aims to use a structure-guided approach to design a broad-spectrum antiviral therapeutic and prophylactic for zoonotic β-coronaviruses that acts by inhibiting the 3CL protease, an enzyme essential for coronavirus replication. The results of preliminary studies suggest the design of such an antiviral is feasible.

C-18. Do cultured human astrocytes secrete thrombospondin-2 protein?
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Astrocytes, one subtype of glial cells, comprise around 30% of CNS cells in the mammalian brain. They are crucial for brain and spinal cord function, supporting neuronal development, neurotransmission, synapse formation and activity through release of proteins and small molecules. Astrocyte are known to secrete thrombospondin proteins (TSPs) *in vivo*. TSPs are important for formation of new synapses. Here, we examined whether astrocytes of human cortex origin secrete thrombospondin-2 (TSP-2) into the tissue culture medium. Commercially purchased human astrocytes were cultured in medium containing 2% fetal bovine serum according to instructions of the supplier. Conditioned medium was collected from cultured astrocytes. After precipitating serum albumin, CM was dialyzed against phosphate buffered saline, and concentrated by passing through centrifugal filters with MWCO 5kDa. Retentates were collected and a known volume of retentate proteins was analyzed by Western blotting to detect TSP-2 using anti-TSP2 on Azure 600. Expression of TSP-2 was analyzed in human astrocyte lysates. A 130 kilodalton TSP-2 protein was detected in astrocyte cell lysates and retentate. Although 60% serum albumin in CM was removed by ammonium sulfate precipitation, remaining 40% albumin in retentate interfered with migration of CM proteins on SDS-PAGE and distorted TSP-2 band. Running a separate Western blot at low voltage was helpful in detecting TSP-2 protein in the retentate. These data will be presented at the K-INBRE meeting.

C-19. GPNMB and SPP1 Activation in Macrophages: Implications for Pulmonary Fibrosis Pathobiology

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The circadian rhythm in mammals is a 24-hour cycle that regulates key physiological processes, including inflammation, immune response, and metabolism. Disruption of the circadian clock is associated with chronic inflammatory lung diseases, such as asthma, chronic obstructive pulmonary disease, and pulmonary fibrosis. However, its role in pulmonary fibrosis, a fatal interstitial lung disease, remains unclear. Environmental factors, such as chronic exposure to cadmium and silica, can induce pulmonary fibrosis, but the underlying molecular mechanisms are not fully understood. Dysregulated inflammation and myofibroblast activation are central to fibrosis progression. Recent evidence implicates macrophage-specific proteins glycoprotein non-metastatic melanoma protein B (GPNMB) and secreted phosphoprotein 1 (SPP1) in this process. This study investigates whether cadmium and silica exposure activate GPNMB and SPP1 signaling in macrophages and explores the role of REV-ERBα, a circadian clock transcription factor, in regulating these proteins and profibrotic responses. Human macrophage cells (Mono Mac 6, MM6) were treated with cadmium chloride (10 μM) or silica (50 μg) for 4, 24, and 48 hours. Cadmium and silica exposure increased intracellular levels of SPP1 and GPNMB, as confirmed by Western blot analysis. However, extracellular levels of these proteins were only slightly altered in the conditioned medium, as analyzed by slot blot. Cadmium exposure significantly increased IL-8 release, while silica exposure did not, as confirmed by ELISA. These findings suggest that cadmium and silica induce macrophage-specific proteins involved in pulmonary fibrosis pathobiology. Ongoing studies aim to determine if REV-ERBα directly regulates these pathways in macrophages.
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C-20. Synthesis of Porphyrins with β-azo Linkage to Other Conjugated Systems

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In photodynamic therapy (PDT) for cancer treatments, porphyrins are often used as photosensitizers. These photosensitizers, upon activation from light, transfer energy into oxygen present in body tissue leading to the generation of reactive oxygen species. These species then interact with various biological substrates within the target tissue; the resulting oxidative damage can subsequently effect cancer cell death. Photosensitizers are most effective at wavelengths longer than 620 nm. This project involves the synthesis of porphyrins with a β-azo linkage to another conjugated system in the hope of increasing the absorption wavelength of the porphyrin to greater than 620 nm. To achieve this, the porphyrin must be constructed via the combination of two dipyrromethane molecules. The current goal is to successfully attach an azo group to the *beta* position of one of these dipyrromethanes. Thus far, all attempts to attach the azo group to the already synthesized dipyrromethane have not been fully successful. We are now attempting to attach the azo group to the various precursors of the dipyrromethane molecule so as to subsequently synthesize the dipyrromethane with the azo group already attached.

C-21. Preclinical evaluation of carnosic acid (CA) and rosemary extract standardized to 40% carnosic acid (RE) for the prevention of breast cancer

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Ductal Carcinoma in Situ (DCIS) is the most common form of non-invasive breast cancer. If left untreated, it is estimated that only less than 50% of these non-invasive cancer diagnoses will make the transition to become invasive. By uncovering the molecular drivers and potential biomarkers that cause the progression, we aim to enhance treatment strategies and improve outcomes for women. The aim of this study was to evaluate the efficacy of CA and RE for the prevention of DCIS invasive progression using the Mouse-INtraDuctal (MIND) model (PMID: 34714554). By utilizing the DCIS MIND models, we demonstrated significantly elevated levels of B cell lymphoma-9 (BCL9) (protein and mRNA), a b-catenin coactivator, with a transition from DCIS to invasive ductal carcinoma (IDC). We chose to target BCL9 using RE and its active ingredient CA, previously identified in a screen for inhibitors of -catenin binding to BCL9. CA and RE were administered to DCIS.COM (basal) and SUM225 (HER2+) MIND models by oral gavage or mixed in a powder diet formulation. Following 6 weeks of treatment, mammary glands were excised and subjected to immunostaining to evaluate drug efficacy and the expression of clinically relevant biomarkers (i.e., Ki67). To evaluate efficacy, the extent of human DCIS and/or IDC growth (EOG), the number of invasive (IL) or micro-invasive lesions (MIL), tumor volume (TV), and tumor numbers (TN) were assessed for each mammary gland. These results will enable the clinical development of a novel and safe chemoprevention option for breast cancer, and for other cancers that show a similar expression of BCL9.

C-22. Cutaneous human papillomavirus E6 impairs the cGAS-STING pathway.

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Each year 3 million Americans are diagnosed with non-melanoma skin cancer (NMSC) and spend approximately $4.8 billion on treatments. Cutaneous human papillomaviruses (cHPV) are believed to promote NMSC by destabilizing the host genome. Supporting this, cHPV E6 expression results in increased DNA damage, including micronuclei. This should induce an innate immune response that eliminates damaged cells. Yet, cHPV E6 promotes rather than restricts proliferation. We hypothesize that cHPV E6 accomplishes this by attenuating the cyclic GMP-AMP synthase-stimulator of interferon genes (cGAS-STING) pathway, an innate immune pathway that becomes activated in response to micronuclei and can induce apoptosis. To test this hypothesis, we examine the cGAS-STING pathway after stimulation by transfection of double stranded DNA (dsDNA). cHPV E6 reduces the magnitude and intensity of cGAS-STING pathway activation. Further, RNA-sequencing of transfected cells demonstrated that cHPV E6 likely more broadly downregulates innate immune activation. These data support a role for cHPV in promoting NMSC development and suggest a mechanism by which cHPV E6 facilitates proliferation of cells destabilized by micronuclei.

C-23. Activated Maple Carbon as a Bio-Based Cathodic Material in Lithium-Sulfur Batteries for Electrochemical Energy Storage Applications
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Amid the energy crisis, there is extensive research going into the field of electrochemistry as scientists search for new methods of harnessing and storing clean energy in a sustainable way. Such methods have the potential to revolutionize technology across the board, including in medical devices and settings. This work focuses on lithium sulfur (Li-S) batteries, a type of electrochemical energy storage device with a complex working principle, termed the conversion mechanism, that retains efficiency through 16-step redox reactions. Li-S batteries are promising, primarily because they have an impressive theoretical potential for an energy density of 500 Wh/kg, electrochemically outperform current lithium-ion (Li-ion) batteries severalfold, and are made with economically favorable materials. Impediments such as the shuttling effect, volume expansion due to (de)lithiation process, and the formation of dendritic lithium are the reasons why Li-S batteries are not favored commercially yet. To prevent such impediments, a high surface area carbon material was synthesized in this work using maple leaves to facilitate incremental conductivity in the presence of structural pores to allow space for sulfur expansion. Different ratios of potassium hydroxide, the activating agent for this bio-based carbon, were compared to analyze its effect on the carbon's surface properties. Further, these carbons were used in the fabrication of Li-S batteries, which were tested using cyclic voltammetry, electrochemical impedance spectroscopy, galvanostatic charge-discharge measurements, and cyclic stability at different C-ratings to reveal the effect of surface area on the Li-S batteries' electrochemical properties.

C-24. Trail camera monitoring and GIS applications to investigate species distribution and habitat use in the Haskell Wetlands
Shawn Bird, Dr. Bridgett Chapin, Aysa Benally. Department of Natural and Environmental Sciences, Haskell Indian Nations University. Lawrence, Kansas.

The Haskell Wetlands located in Lawrence, Kansas are the subject of continual research to more completely understand the zoogeography of the area and the physical environmental factors that are of influence. Landscape features in the wetland tract consist of natural and human engineered elements; the area is bordered by a highway and other urban infrastructures such as bridges and walkways are present also. The aim of my study is to further investigate corridors in use by local fauna. The wetlands are bisected by highway K-10 and my aims include observation of the potential impact that this habitat fragmentation has had on local wildlife's preferred methods of the road's traversal. Ongoing studies have revealed the presence of numerous wildlife species via the use of motion activated trail cameras and I have used these data histories as well as newly gathered camera footage to shed light on the corridors in use by notable species of terrestrial mammals.

C-25. The Minor Allele of *Ptpn22* Changes Pro-Inflammatory Cytokine Production in Dendritic Cells
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Dendritic cells (DCs) produce cytokines with direct antiviral properties, such as type I interferon (IFN-I), as well as polarizing cytokines that promote T cell differentiation (e.g. IL-12). Previously, we found that the common minor allele of the immunoregulatory gene *Ptpn22* (PEP-R619W) promotes antiviral immunity in DCs and T cells. However, the mechanisms by which *Ptpn22*, and its minor allele, influence antiviral immune function remain largely unknown. Prior studies report that mice without *Ptpn22* expression (PEP-null) have reduced IFN-I production in DCs following LPS exposure. However, they did not investigate the impact of PEP-R619W on IFN-I production during virus infection. We hypothesize that mice expressing PEP-R619W possess an immunostimulatory cytokine profile which enhances viral clearance. To test this, we measured the production of IFNβ, and other pro-inflammatory cytokines including IL-2, IL-12, and TNF-α, following exposure to LPS, 3p-hp-RNA (RIG-I agonist), or virus. Contrary to previous studies, our results show *Ptpn22,* and its minor allele do not impact IFNβ production. However, PEP-R619W DCs produce more IL-2 and less TNF-α compared to wildtype DCs. This study demonstrates that PEP-R619W alters the production of various pro-inflammatory cytokines which may promote viral clearance. These results provide further insight into the mechanisms in which the minor allele of *Ptpn22* enhances the anti-viral immune response.

C-26. Qualitative Analysis of Hair Samples Treated with Over-the-Counter Hair Products using GC/MS
Gaea Gratiae Tradio, Holly O'Neill
Washburn University Chemistry Department

The analysis of different biological fluids and samples for detection of different drugs and their metabolites have been a forensic research focal point for many decades. While urine and blood have been commonly analyzed for drug detection, the interest in hair analysis has been rapidly growing. Hair displays many advantages over urine and blood due to its easy collection and its ability to retain most drugs and their metabolites for longer periods of time[1]. Hair is also relatively easier to collect in comparison to other biological samples. However, there has been minimum research performed on the effects of over-the-counter hair products on hair samples and how these products affect drug detection. This study was an attempt to determine the effects of using over-the-counter hair products on hair samples using a Gas Chromatography tandem Mass Spectrometry (GC/MS). Hair samples were collected randomly from a third-party source and were treated with a variety of over-the-counter products: Heads & Shoulders Shampoo, Olaplex Shampoo, Paul Mitchell Shampoo, Shimmer Lights lightener and developer, and L'Oreal Paris Excellence Cream in Medium Brown. Research from this study determined that certain chemicals could be detected by the GC/MS on treated hair samples, most notably chemicals such as nonamide, squalene, and cholesterol. These chemicals were most prominent in dyeing treatments and consistently did not show in untreated hair samples.

C-27. Interdisciplinary and Comprehensive Evaluations to Increase Service Access for Children Impacted by Autism Spectrum disorder
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Identification and treatment of autism spectrum disorder (ASD) in rural communities continues to lag when compared to opportunities in urban areas (e.g., Antezana et al., 2020), with the average age of diagnosis being delayed in both rural communities and in households with limited income (Mandell et al., 2010). Though many factors may contribute to the deficiency of available services, lack of qualified diagnosticians in rural locations presents as a major barrier to families accessing evaluations. Early intervention services have been found to significantly improve symptomology of ASD in children diagnosed prior to three years of age (e.g., Gabbay-Dizdar et al., 2021), yet a diagnosis is required prior to accessing care. Rural areas may benefit from interdisciplinary ASD evaluation and diagnostic mechanisms that increase access to care. The purpose to the present project is to pilot an interdisciplinary evaluation model using faculty and students at a university located in a rural area of the country. The evaluation model includes multiple disciplines engaging in a multi-department collaboration for both collegiate student training and community support, creating a comprehensive evaluation process to support children suspected of having ASD but who are unable to access timely diagnostics and treatment due to geographic location.

C-28. The Impact of Dry Cupping on Pain Relief and Distal Circulation, Along the Meridian Chain.
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This pilot study investigated the acute effect of a single dry cupping session on distal pain relief and circulation. Five adults, ages 26 to 66 years, suffering from lower back pain for three or more months participated. Participants completed a Brief Pain Inventory (BPI) (Cleeland, C. S., 1991) and Near Infrared Spectroscopy (NIRS) using a Portamon NIRS device (Artinis, Elst, Netherlands) prior to, immediately after, and 7-10 days post cupping treatment. NIRS measurements were taken at the hamstring and an inch to the right of L4 vertebrae, with the use of two Portamon units for simultaneous data collection. Prior to data collection the Portamon units recorded Hemoglobin, Deoxyhemoglobin, and total Hemoglobin to allow for calibration per the procedures in the Portamon manual. The data collection period length was 30 seconds at a frequency of 10 Hz. A single nine-minute dry cupping session was administered by a certified practitioner on each participant's hamstring. Following the cupping treatment, participants avoided all forms of treatment (massage, dry cupping, other) and continued their daily lives for seven to 10 days. The NIRS found no significant changes in Hemoglobin, Deoxyhemoglobin, and total Hemoglobin. A significant decrease in pain was found immediately after dry cupping (2 ± 1.41). At seven to ten days post intervention pain levels returned to baseline. The lack of significant change in hemoglobin levels and immediate pain relief possibly indicates the pain gate theory as a possible mechanism of dry cupping therapy, suggesting that cupping therapy may not have long-term benefits.

C-29. Purification and characterization of stimulators of programed cell death protein 1 (PD-1) and granzyme B expression in T Lymphocytes.
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Lung cancer is the leading cause of cancer related deaths in the United States. Our previous studies have shown that an orally administered (OP) water extract of *Euglena gracilis* (EWE) prevents lung tumor growth in a Lewis lung carcinoma (LLC) allograft mouse model and tobacco smoke carcinogen-induced lung cancer model through the attenuation of myeloid derived suppressor cells, activation of cytotoxic T cells, and alteration of the intestinal microbiota. Recently, our group also discovered that EWE-OP increases the expression of both PD-1 and PD-L1 in both lung tumor cells and $CD45^+$ lymphocytes in the lung tumors. *In vitro*, EWE also stimulates the expression of PD-1 and PD-L1 in LLC cells and T lymphoblasts. Furthermore, EWE directly stimulates the expression of several cytokines such as Granzyme B, tumor necrosis factor alpha (TNFa), and interferon gamma (IFNy) in T lymphoblasts. However, the mechanism of action and specific substances in EWE that accomplish these effects are not known. To find the specific substance(s) within EWE that are responsible for the expressions of PD-1 and granzyme B in T lymphoblasts, we purified these bioactive substances in EWE using fast protein liquid chromatography (FPLC) with a Superdex 75 gel filtration column and HPLC with a C18 reverse phase column, and characterized whether these two bioactivities are caused by a single purified substance or not. The results strongly suggest that a small molecule with relatively weak hydrophobicity stimulates the expression of PD-1 and granzyme B in T lymphoblasts.1H NMR and LC-MS-based structural characterizations are ongoing.

C-30. The Function of Human CLPB in Apoptosis of Breast Cancer Cells
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Human CLPB is a mitochondrial protein disaggregase residing in the mitochondrial intermembrane space. CLPB belongs to the AAA+ family of ATPase and functions to couple ATP hydrolysis with protein disaggregation and unfolding. Mutations in CLPB have been associated with methylglutaconic aciduria type VII and congenital neutropenia. Previous studies indicate that deletion of CLPB by CRISPR/Cas-9 in neutrophil precursors leads to increased apoptosis. However, CLPB's role in apoptosis within cancer cells remains largely unexplored. This study investigates the role of CLPB in triple-negative breast cancer cell lines MDA-MB-231 and Hs578T. It is hypothesized that disabling CLPB in these two cell lines would cause an increased sensitivity to apoptosis. To test this hypothesis, CLPB expression in MDA-MB-231 and Hs578T cells was first confirmed with western blotting using anti-CLPB antibody. Then, transfection conditions and apoptotic conditions were optimized through two different experiments. Once experimental conditions were optimized, cells were first transfected to express a dominant negative CLPB mutant. Then, apoptosis was induced using staurosporine. Apoptotic activity was measured using fluorescent markers Annexin V and a cytotoxicity dye, and analyzed with a live cell imager (Sartorius Incucyte). These studies will help understand CLPB function in apoptosis of triple-negative breast cancer.

C-31. Role of Mitochondrial Protein CLPB in Apoptosis of Breast Cancer Cell Lines
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Human CLPB is a ubiquitously expressed ATPase that resides in the mitochondrial intermembrane space. This protein functions to disaggregate proteins, which is coupled with ATP hydrolysis. Mutations in CLPB have been found to cause congenital neutropenia. This study aims to investigate the role of CLPB in apoptosis in triple negative breast cancer cell lines, BT549 and SUM159. First, endogenous CLPB expression was quantified in these two cell lines using western blotting and anti-CLPB antibody. Transfection efficiency of these cell lines was then optimized using a GFP expressing plasmid and confirmed using live cell imaging (Sartorius Incucyte). In a separate experiment, apoptosis was induced in SUM159 using staurosporine. An optimal concentration of staurosporine was determined. Lastly, these two approaches were combined to observe the role of CLPB in induced apoptosis. SUM159 cells were transfected with GFP plasmid that also contains dominant negative human CLPB mutant to block the function of the endogenous CLPB. Then apoptosis was induced in transfected cells using staurosporine. Apoptotic activity was observed in the Incucyte using Annexin V, an apoptotic marker, and Cytotoxicity dye. These tests will help to better characterize the role of CLPB in apoptotic triple negative breast cancer cells.

C-32. Characterization of a Novel Peptide Computationally Designed to Inhibit the CD80/CD86 and CTLA-4 Interaction in T Lymphocytes
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Lung cancer accounts for more deaths than the next two deadliest cancers, colorectal and pancreatic cancer, combined. Current immunotherapy treatment for lung cancer uses antibodies as immune checkpoint inhibitors (ICI), but these treatments often encounter immune response-related adverse effects. We have previously computationally designed a novel peptide to inhibit the CD80/CD86 and CTLA-4 interaction, which is a known T cell immune checkpoint mechanism. This peptide, hereafter noted as CTLA-4 inhibitory peptide (CTLA-4ip), was shown to enhance tumor antigen-primed $CD8^+$ T cell-dependent lysis of lung cancer cells *in vitro* and inhibit lung tumor growth in mice. Recently, we discovered that CTLA-4ip internalizes into the cytoplasm of both murine and human NSCLC cells and induces the expression of PD-L1 which is a ligand for another immune checkpoint protein PD-1. The PD-1 and PD-L1 interaction is also another target for ICI therapy. Since moderate to medium high expression of the PD-L1 in human lung cancer is associated with a better outcome of ICI therapy, this CTLA-4ip may have another benefit in lung cancer ICI therapy in addition to immune checkpoint inhibition. It was also discovered *in vitro* that CTLA-4ip interacts with T Lymphocytes and increases the expression of cytokines such as TNF-a, IFN-y, and Granzyme B, all of which are associated with cytotoxic T cell-dependent tumor cell lysis. Accordingly, it is postulated that CTLA-4ip could be a less expensive and safer ICI therapeutic than the currently available antibody-based therapeutics.

C-33. The structural landscape of pharmaceutically relevant 5-amino-1H-1,2,4-triazole building blocks and co-crystals thereof
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1. O'Sullivan, A., Long, B., Verma, V., Ryan, K. M., & Padrela, L. (2022). Solid-state and particle size control of pharmaceutical co-crystals using atomization-based techniques using atomization-based techniques. International Journal of Pharmaceutics, 621(0378–5173), 121798. https://doi.org/10.1016/j.ijpharm.2022.121798

5-amino-1H-1,2,4-triazoles are common building blocks of many pharmaceutically active compounds. However, to generate new triazole-containing drugs through a targeted 'bottom-up' strategy, we need to better understand the balance between different intermolecular forces in such compounds as this would allow us to control solid–state assembly and thus fine-tune physical properties such as aqueous solubility. Mapping their structural landscape will allow the development of reliable synthesis protocols for co-crystals, which are promising new solid forms in drug formulations.[1] Our previous study focused on the influence of steric effects on the crystal structures of alkyl-decorated triazoles. This project aims to investigate the structural effects on solid-state assembly by synthesizing co-crystals with FDA-approved coformers through liquid-assisted solid-state grinding. We will analyze the bulk products for yield, side-products, and unreacted materials, using single-crystal X-ray diffraction and infrared spectroscopy for structural determination and intermolecular bonding detection. Dissolution studies and thermal analyses will be conducted to correlate structural changes with physical properties. Expected outcomes include insights into molecular interactions, rationalization of successful coformer selection, and understanding the impact of electron-donating and withdrawing groups on triazole bonding interactions.

C-34. Next Generation Sequencing at KU Genome Sequencing Core
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The Genome Sequencing Core (GSC) is one of three research service core labs in the NIH COBRE Center for Molecular Analysis of Disease Pathways (CMADP) at the University of Kansas (KU). The major mission of the GSC is to provide researchers with next-generation sequencing (NGS) technologies. NGS, carried out in a massively parallel fashion, has been revolutionizing bio-medical research and used in a growing list of applications. Projects supported by the GSC include de novo genome assembly, genome re-sequencing for identification of mutations and polymorphisms, transcriptome analysis (RNA-seq), and epigenomic and gene regulation studies such as ChIP-seq, Methyl-seq, and small RNA analysis. The GSC enhances the genomics infrastructure already at KU by providing a range of Illumina sequencing platforms including the NextSeq2000 and NextSeq550 (mid-sized genome re-sequencing or transcriptome projects) and the MiSeq (metagenomic or targeted amplicon sequencing projects) to researchers at KU-Lawrence and across the region. To capture the full power of NGS, we provide a range of project support, including project consultation, sample quality check, sequencing library construction, Illumina sequencing, and FASTQ generation and demultiplexing. For latest pricing, current sequencing queue, or other information, visit the Genome Sequencing Core's website: https://gsc.ku.edu/.

C-35. University of Kansas Nanofabrication Facility: Equipment and Services
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The Kansas University Nanofabrication Facility (KUNF) is a Core Lab supported by the KU Office of Research and the Center for Molecular Analysis of Disease Pathways COBRE. The KUNF primarily caters to researchers who are manufacturing micro- and nanofluidic devices for biomedical research, but has the equipment and resources to accommodate broad research applications with micro- and nanofabrication needs. The core lab consists of about 1,300 ft^2 of ISO class 5, 1,700 ft^2 of ISO class 6 and 1,250 ft^2 of ISO class 7 cleanroom space, housing tools and materials for techniques including photolithography, nano-imprint lithography, plasma (dry) etching (ICP-RIE), wet etching, thin film deposition, scanning electron microscopy (VP-SEM), atomic force microscopy, contact angle goniometry, ellipsometry, profilometry, wafer dicing, wire bonding, laser ablation and engraving, 3D printing, hot embossing, and COMSOL software for device modeling. In addition, the facility has numerous microscopes for general inspection, ovens and furnaces, ultrapure water, and dedicated process fume.
This facility is under the leadership of Dr. Susan Lunte, and the direction of Ryan Grigsby. Services and usage of the facility are available to researchers from all Kansas universities. Training is provided to new investigators and graduate students in the use of micro- and nanofabrication procedures and equipment. In addition, researchers from both non-Kansas academic and private industry institutions may contract with the facility for consultation and services. Hourly and per-use rates apply for facility access, equipment usage, and staff labor. Consultation is free.

C-36. RpoN-dependent phosphotransferase systems in *Enterococcus faecalis*
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Phosphotransferase systems (PTS) play significant roles in the uptake and phosphorylation of sugars for metabolism. *Enterococcus faecalis* is predicted to encode 46 distinct PTS pathways, highlighting its versatility to grow on a variety of carbon sources, but many of the PTS substrates are unknown. In *E. faecalis*, six PTS are predicted to be regulated by the alternative sigma factor, RpoN, but only a small number of PTS substrates are known. To identify potential substrates for the RpoN-dependent PTS, we performed a Biolog carbon source phenotype array comparing commonly used strains of *E. faecalis* along with their isogenic *rpoN* mutants. We also took a bioinformatic approach to identify functionally characterized PTS from other organisms that show relatedness to the *E. faecalis* RpoN-dependent PTS. RpoN-dependent gene regulation in *E. faecalis* is also dependent on five bacterial enhancer binding proteins (bEBPs) of the LevR-family. Biolog results showed that the metabolism of glucose, mannose, cellobiose, gentiobiose, arbutin, salicin, glucosamine, and amygdalin require PTSs that are RpoN-dependent. Through bioinformatics, we were able to identify additional sugar substrates (glucosaminic acid, glucoselysine and fructoselysine) as potential PTS substrates dependent on RpoN. Through mutational analysis of the various bEBPs in *E. faecalis*, we were able to demonstrate a linkage between those sugars and a dedicated PTS responsible for their import. We also demonstrate through luciferase reporter assays that the PTS operons are induced by the sugar substrates in a manner that requires both RpoN and the corresponding bEBP.

D-1. The Computational Chemical Biology Core: A Chemical Biology of Infectious Disease COBRE Core Laboratory
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Part of the Chemical Biology of Infectious Disease COBRE at the University of Kansas, the Computational Chemical Biology Core (CCB) works in collaboration with the Molecular Graphics and Modeling (MGM) Laboratory to provide the computational resources and expertise to enhance the productivity of researchers studying infectious diseases, in addition to other projects. The CCB has the tools and expertise to perform virtual screening, small molecule docking, chemoinformatics analysis of high-throughput screening hits, binding site prediction, protein/peptide/antibody modeling and docking (including Alphafold modeling), protein design, and molecular dynamics simulations.
Recent highlights include the identification inhibitors of ACMS decarboxylase and DNAJA1 via virtual screening, using modeling to identify the functional activity of *Legionella pneumphila* effector protein SidI, using modelling to assess the structural impact of clinically relevant point mutations of TRIM32, modeling the interaction between the Type III secretion system basal body and sorting platform proteins SctK and SctD from *Pseudomonas aeruginosa*, and the optimization of an inhibitor of PTPRD.
With the software and expertise to perform virtual screening, protein-small molecule docking, protein/peptide modeling/docking, and cheminformatic analysis, the CCB is a valuable resource to enhance the productivity of researchers studying infectious diseases, in addition to other projects.
The CBID COBRE is funded by the NIH NIGMS grant 1P20GM113117.

D-2. Assessing depth and evenness of sequencing coverage across hundreds of archived plastid genomes
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More than 24,000 complete plastid genome records are accessible on public sequence databases such as NCBI GenBank as of December 2024, but multiple studies have reported widespread assembly and annotation errors among these genome records. The depth and evenness of sequencing coverage are considered potential assembly quality indicators that could offer valuable insights into the accuracy of these plastid genome records. However, the typical variation of sequencing depth and evenness among archived plastid genomes is poorly understood, limiting their applicability as quality indicators. Here, we explore this variation across a sample of hundreds of publicly accessible plastid genomes. We especially evaluate the variation of sequencing depth and evenness in relation to plastid genome structure and to methodological factors of the assembly process such as the type of next-generation sequencing platform employed. Our results reveal significant differences in sequencing depth among the structural partitions of most plastid genomes and significant variation in sequencing evenness across different sequencing platforms. Several aspects of this research were conducted by an undergraduate student at Fort Hays State University as part of an undergraduate research experience.

D-3. DNA barcoding of bloom-forming cyanobacteria in western Kansas.
Louisa Acquah, Dr. Michael Gruenstaeudl
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Cyanobacteria are water-dwelling prokaryotes that make up a significant portion of the biomass in freshwater systems and are found in aquatic habitats all over the world. The production of toxic compounds that are detrimental to both plant and animal life is frequently associated with the mass propagation of cyanobacteria. Exposure to cyanobacterial blooms can cause severe health problems in humans and animals. Cyanobacterial blooms are occurring more frequently due to increased fertilizer run-off from agriculture and climate change, particularly in the midwestern regions of the United States.
In regions like western Kansas where water is scarce, cyanobacterial blooms pose a serious but sometimes disregarded threat. The precise identity of the cyanobacteria causing cyanobacterial blooms in western Kansas's rivers and lakes is essentially unknown, despite reports of them occurring frequently. Understanding the genetic identity of cyanobacteria strains driving blooms in western Kansas is crucial for identifying the toxins produced and assessing the associated health risks. The high temperatures in western Kansas create ideal conditions for diverse cyanobacteria strains, each potentially adapted to environmental stresses, suggesting the coexistence of multiple strains in the region.
This research proposal aims to cultivate cyanobacteria collected from Horsethief Reservoir, located in Jetmore, Kansas, followed by molecular identification to characterize the strains. Currently, I have successfully cultivated *Microcystis spp*. and *Geitlerinema spp*. under controlled laboratory conditions using BG-11 medium within a growth chamber. To mitigate diatomic contamination, Germanium oxide was incorporated into the samples, demonstrating effective results.

D-4. Development of PROTAC-based Cellular Probe to Protein N-Terminal Methylation

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Since the concept of Proteolysis-Targeting Chimeras (PROTACs) was first reported in 2001, PROTACs technology has become one of the most powerful tools for drug discovery and development. PROTAC-based therapy has shown a promising future for the treatment of various diseases, such as cancers, neurodegenerative dysfunction, and autoimmune diseases. Protein methylation catalyzed by N-terminal methyltransferase 1 (NTMT1) is a widespread post-translational modification, which has been shown to play important roles in transcription, DNA repair, signal transduction, and regulation of protein-DNA and protein-protein interactions. However, the biology of N-terminal methylation remains enigma. Therefore, it is of great interest to develop PROTAC-based degraders to NTMT1 as a cellular probe to study N-terminal methylation. Here I will report our efforts to explore various strategies to develop and optimize different types of NTMT1 degraders. A series of PROTACs targeting NTMT1 have been synthesized and their degradation efficiencies have been characterized. The results obtained will guide us to develop the first NTMT1-specific cellular probe with nM potency.

D-5. The Autoimmunity-Associated Minor Allele of *PTPN22* Enhances Innate Antiviral Immunity During Coronavirus Infection

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Allelic variation can impact viral clearance and disease severity. Mice expressing the autoimmunity-associated allelic variant of *Ptpn22* (PEP-R619W) clear chronic LCMV-cl13 infection whereas PEP-WT mice do not. However, little is known about *Ptpn22* during other virus infections. This research defines how the loss of *Ptpn22* (PEP-null) and PEP-R619W changes antiviral immunity during coronavirus infection. We addressed the hypothesis that CRISPR/Cas9 generated PEP-null and PEP-R619W mice have enhanced antiviral immunity over PEP-WT mice during coronavirus infection. Following Mouse Hepatitis Virus (MHV) A59 infection, we interrogated pathology, cytokine production, and cellular responses in the spleen, blood, and liver of PEP-WT, PEP-null, and PEP-R619W mice. Key findings show that PEP-R619W mice have 1) reduced viral titer and weight loss, 2) increased survival, and 3) more mature NK cells in the liver and spleen over PEP-WT and PEP-null mice. Further, Rag1$^{-/-}$ PEP-R619W mice had increased survival and reduced viral titer over Rag1$^{-/-}$ PEP-WT mice. PEP-R619W mice also had higher concentrations of IFNγ and enhanced IFNγ production by mature NK cells in the liver at 3 days post-infection. Finally, NK cell depletion elevated PEP-R619W viral titer to similar levels of PEP-WT mice. This is one of the first studies investigating the role of *Ptpn22* within NK cells. We demonstrate that the *Ptpn22* allelic variant augments NK cell function and is beneficial during coronavirus infection.

D-6. Molecular target for the tamoxifen-refractory breast cancers

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Breast cancer remains a leading cause of cancer-related mortality among women. Tamoxifen, an estrogen receptor (ER) antagonist, is the most effective first-line therapy for ER-positive breast cancer in clinical practice. However, the development of tamoxifen resistance significantly reduces its therapeutic efficacy. This study aims to elucidate the molecular mechanisms underlying tamoxifen resistance in human breast cancer cells. Two human breast cancer cell lines, MCF-7 and MDA-MB-468, were utilized. The cells were cultured in DMEM medium containing 5 – 10% fetal bovine serum and subjected to increasing doses of tamoxifen, ranging from 0.5 μm to 25 μm. While MDA-MB-468 cells did not survive tamoxifen treatment at 3 μm dose, some MCF-7 cells endured up to 25 μm over a six-month period. Scratch tests revealed that 5μm tamoxifen did not affect the tumor cell growth after five months of treatment, whereas 25 μm significantly reduced cell proliferation and migration, although the cells remained viable. Real-time PCR analysis showed a substantial loss of estrogen receptor (ESR) mRNA expression in tamoxifen-refractory MCF-7 cells compared to naïve cells, while ABCB1 (multi-drug resistance protein 1) expression was over 40 times higher. Conversely, the progesterone receptor (POR) level remained unchanged. These findings underscore the importance of further research to translate these observations into clinical applications, with a particular focus on targeting ABCB1 as a potential therapeutic strategy.

D-7. Extracellular Vesicles (EVs) as a Prognostic Biomarker for Molecular Therapy Targeting RNA-binding Protein HuR in Cancer Immunotherapy

Alfred Buabeng, Sunghae Kim, Qi Zhang, Xiaoqing Wu, and Liang Xu.
University of Kansas/ Department of Molecular Biosciences

Breast cancer is highly heterogeneous with diverse molecular alterations. The RNA-binding protein HuR (Human antigen R) plays a crucial role in cancer progression and treatment resistance. Molecular therapies targeting HuR are being developed, but predicting patient response remains challenging. Emerging evidence suggests extracellular vesicles (EVs) are involved in intercellular communication and can serve as biomarkers for cancer diagnosis and prognosis. This study investigates EVs as prognostic biomarkers for therapies targeting HuR in cancer immunotherapy. EVs from breast cancer cell lines were isolated and characterized for size distribution and protein markers. HuR expression levels were assessed in cancer cell lines and EVs. In vitro assays evaluated the effect of Immuno Checkpoint Blockage immunotherapy and HuR small molecule inhibitors on cancer cell viability and EV levels. EVs from cancer cells contained HuR, suggesting HuR communication between cells via EVs. Treatment with ICB immunotherapy and HuR inhibitors inhibited cancer cell growth, increased T cell activation, and reduced EV levels and surface markers. These findings indicate the potential prognostic value of EVs in HuR-targeted therapy and immunotherapy in breast Cancer.
Further research is needed to understand HuR mechanisms in EVs and validate the in-vivo efficacy of HuR-targeted therapy. This study highlights the potential of targeting HuR and utilizing EVs in developing personalized cancer treatments.

D-8. FINDING A GUT-PAINCONNECTION IN NEUROGENIC BOWEL PAIN AND DISORDERS

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Neurogenic bowel (NB) is a condition characterized by slow colonic transit, fecal incontinence, and chronic abdominal pain that most often develops following damage to the central nervous system, as in traumatic spinal cord injury (SCI) or non-traumatic diseases such as Parkinson's disease. Since the molecular mechanisms of NB remain unknown, current treatments remain symptom-focused and are largely ineffective. Therefore, the primary goal of the present studies is to uncover the mechanisms underlying NB. Our lab has recently characterized a rodent model of moderate SCI resulting in NB phenotypes comparable in SCI patients. We found that peripheral calcitonin gene-related peptide (CGRP) is released into the colon after SCI. CGRP is a neurogenic inflammatory mediator released by primary sensory neurons that increases neuronal activity and induces pain. We have shown that intrarectal antagonism of CGRP significantly prevents NB phenotypes like colonic dysmotility, and neoplastic lymphoid hyperplasias of colon. Existing literature and studies from our lab link CGRP overexpression and release after SCI with the establishment of a proinflammatory environment within the colon and, potentially, induction of gut microbial dysbiosis. Interestingly, calcium imaging of nodose ganglion neurons *in vitro* revealed that colon-specific vagal afferents become hyperresponsive to fecal supernatants from SCI animals, suggesting a role for CGRP and the gut microbiome in sensitization of sensory afferents that carry nociceptive signals to the brain. Further studies on the impact of gut dysbiosis on pain development are currently underway to identify novel therapeutic targets to restore gut homeostasis and treat NB in SCI patients.

Funding Source: Craig H Neilsen Foundation SCIRTS pilot grant

D-9. Analyzing Common Molecular Targets in Diabetic Brain and Kidneys

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Cognitive impairment is a common complication of chronic kidney disease (CKD), yet there are no therapies that concurrently address both cognitive decline and CKD progression. Proteins such as nephrin and synaptopodin, essential for maintaining the glomerular filtration barrier (GFB) in diabetic nephropathy, are also expressed in brain cells proximal to the blood-brain barrier (BBB). This shared expression suggests conserved molecular mechanisms between the GFB and BBB. We hypothesize that disruptions in these barriers are driven by convergent molecular pathways that are potentially targetable, thereby offering dual therapeutic benefits for CKD and cognitive dysfunction.

To investigate this hypothesis, we utilized the db/db mouse model of diabetes, which manifests both CKD and cognitive decline. Mice were treated with the mineralocorticoid receptor antagonist spironolactone, a known modulator of GFB integrity, and disease progression was evaluated in renal and neural tissues. Spatial transcriptomics (10x Visium) was employed to map the spatial distribution of dysregulated genes in the kidneys and brains of diabetic versus nondiabetic mice, enabling the identification of shared molecular alterations.

Spironolactone treatment improved histopathological features in both kidney and brain tissues. Spatial transcriptomics and immunohistochemical analyses revealed that *Rnaset2* and *Lpn2* were markedly upregulated across several cell clusters in the db/db model, suggesting their involvement in the pathophysiology of both tissues. These findings highlight conserved molecular pathways implicated in diabetic kidney and brain dysfunction, underscoring the potential for shared therapeutic targets in diabetes-associated complications.

No conflicts of interest

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D-10/ Pleiotropic Prioritization: Unraveling Shared Genetic Threads in Insomnia and Chronic Pain Through an Advanced Gene Prioritization Pipeline

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Many chronic pain patients report co-occurring sleep disturbances, like insomnia, which have been linked to chronic pain development and exacerbation. Though these complex conditions frequently co-occur, it is unclear whether these are distinct conditions or whether a common mechanism may underlie development of both. Using a bioinformatics approach, we identified potential pleiotropic genes associated with both phenotypes. First, we developed a pipeline to prioritize genes implicated via single nucleotide variants (SNVs) associated with either insomnia or chronic pain phenotypes in genome-wide association studies (GWAS). Using the Functional Mapping and Annotation database, FUMA v1.5.6, we identified genes associated with our phenotypes of interest and mapped these to their mouse orthologs using the DRSC integrative ortholog prediction tool (DIOPT v9.0). We further searched for phenotypes of interest resulting from gene knock-outs in mice from the International Mouse Phenotype Consortium (IMPC) database. We then mapped prioritized gene product interactions using StringDB (v. 2.14.3). Interaction maps were identified for both human and mouse. Filtering using Pharos drug target database (v 3.19.1) resulted in 32 gene products with known drug targets. Using previously published whole genome-sequencing data generated from multiple inbred mouse strains, we identified 4 genes from our prioritized list that contained genotypic differences between substrains. We selected the gene Grik2, glutamate ionotropic receptor kainite type subunit 2, for further analysis. This pipeline facilitates the generation of novel hypotheses centered around common genetic mechanisms of risk for insomnia and chronic pain. Follow-up studies examining how Grik2 influences both insomnia and chronic pain are warranted.

D-11. *Caenorhabditis elegans* models of neurodevelopmental disorder-associated *AGO1/AGO2* variants

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Gene regulation by microRNAs (miRNAs) is essential for animal development. miRNAs associate with Argonaute (AGO) proteins to form a functional complex that regulates gene expression by silencing target genes. As AGOs are required for miRNA function, impaired AGO activity alters miRNA-dependent gene regulation and disrupts animal development. In humans, several coding variants in the AGO1 and AGO2 genes have been linked to neurodevelopmental disorders (NDD), although it remains unclear how altered miRNA-dependent gene regulation might be linked to NDD pathogenesis. We previously showed that introducing a subset of equivalent NDD-associated variations into a highly conserved *Caenorhabditis elegans* AGO (ALG-1) leads to allele-specific defects in miRNA processing and target repression. As each modeled variant disrupts distinct miRNA populations, this may suggest that multiple miRNAs contribute to NDD phenotypes. However, pathogenesis may also result from the dysregulation of a small, shared set of miRNAs. Given that our previous analysis only characterized a subset of the NDD variations, the prevalence of common molecular defects across variants remains unclear. Here, we used genome editing to engineer six additional ALG-1 variations, further expanding our coverage of NDD-associated human AGO variants. Preliminary characterization revealed varying degrees of developmental defects among ALG-1 NDD strains, consistent with previous findings. Interestingly, introducing wildtype ALG-1 into these mutant strains partially restores normal development, suggesting dose-dependent developmental functions of ALG-1. Our current work is aimed at characterizing the effects of ALG-1 NDD variations on miRNA-dependent gene regulation and assessing neuronal phenotypes to further understand the pathobiology of AGO-associated NDD.

D-12. RNA binding protein HRPK-1 coordinates with miRNAs to regulate *C. elegans* development
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Noncoding RNAs known as microRNAs (miRNAs) post-transcriptionally control the expression of many genes. miRNAs are loaded by Argonaute proteins to form a complex called miRISC, or miRNA-Induced Silencing Complex. The miRISC then targets mRNAs for repression via partial sequence complementarity and negatively regulates the expression of target genes through mRNA degradation and/or translational repression. Additionally, mRNAs are also known to be extensively regulated by RNA-binding proteins (RBPs) through RNA processing, transport, and translational regulation. We found that RNA-binding protein, HRPK-1 (Heterogeneous nuclear ribonucleoprotein K homolog) coordinates with ALG-1 Argonaute to regulate gene expression during *C. elegans* development (LiLi,2014). In *C. elegans,* genetic and molecular characterization of *hrpk-1* revealed its function in development and miRNA-mediated targeted suppression (LiLi,2014). Loss of *hrpk-1* causes a variety of developmental defects and exacerbates the mutant phenotypes associated with reduced miRNA activity, including *let-7* and *mir-35*-family miRNAs (LiLi,2014). To identify which functional domains of HRPK-1 are important for gene-regulatory coordination with the miRNA pathway, we performed functional domain analysis by deleting or mutating the combinations of six different HRPK-1 domains or predicted signaling sequences and found its effects on different developmental phenotypes. We are also examining to check effects of one of the clinical mutations in human HNRNPK gene. Here, I will provide an update on current investigations toward delineating the interacting pathway of HRPK-1 with miRNAs during the development of *C.elegans*.

D-13. Evolutionary history of two X chromosome meiotic drivers in *Drosophila affinis*
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Meiotic drivers are selfish genetic elements that bias gametogenesis to enhance their own transmission, cheating Mendelian segregation. When located on a sex chromosome, such drivers can skew progeny sex ratios, creating a female-biased population and diminishing average population fitness. In *Drosophila affinis*, two distinct X-linked meiotic drivers, with characteristic karyotypes distinguishable using unique inversions, segregate in wild populations (~10% frequency). The X chromosome in *D. affinis* is a neo-sex chromosome formed by the fusion of the ancestral X with a former autosome resulting in 40% of the genome being X-linked. We hypothesize that these inversions prevent recombination across the entire X chromosome and create distinct X chromosome haplotypes in *D. affinis*. Using whole genome sequencing of wild-caught male flies (with and without meiotic drive), we analyzed (i) the origin of the meiotic driving and non-driving X chromosomes, (ii) the divergence between the meiotic driving and non-driving X chromosomes, and (iii) whether driving X chromosomes accumulate greater genetic load, to assess the impact of meiotic drive on the genome evolution in *D. affinis*.

D-14. Nuclear APC Maintains Colon Homeostasis and Mitigates Inflammation
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Adenomatous polyposis coli (APC) is critical for maintaining intestinal homeostasis. Although widely recognized for its cytoplasmic tumor suppressor functions, the roles of APC in other subcellular compartments and inflammation are less defined. To investigate nuclear APC functions, we developed a mouse model with compromised nuclear Apc import ($Apc^{mNLS/mNLS}$ mice). $Apc^{mNLS/mNLS}$ mice displayed lower levels of mucin-2 (*MUC2*) RNA, the primary component of the intestinal mucus barrier, and had significantly thinner colonic mucus layers. These mice were more susceptible to experimentally induced colitis than their wild-type littermates. Importantly, male $Apc^{mNLS/mNLS}$ mice exhibited significantly increased weight loss and higher colitis scores compared to female mice. Therefore, we hypothesize that nuclear APC promotes gut barrier integrity by upregulating *MUC2* expression. In cultured human colon cells, APC positively regulates *MUC2* RNA levels and inhibits NF-κB signaling. Overall, this study provides evidence that nuclear APC upregulates colonic *MUC2* expression, increases the mucus barrier, and inhibits colonic inflammation. Furthermore, our results reveal notable sex-specific differences in colitis susceptibility.

D-15. Advanced Insights into Non-Adiabatic Dynamics and Ring-Opening Mechanisms of Oxazole and Isoxazole
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This study aims to investigate the photoinduced ring-opening dynamics in oxazole and isoxazole using ab initio non-adiabatic molecular dynamics simulations. Employing the State-Averaged CASSCF/aug-cc-pVDZ level of theory, we explored the excited-state dynamics and ring-opening mechanisms of these heterocyclic compounds. Initial conditions were generated using Wigner sampling, and trajectories were propagated for up to 1,000 fs. Our results reveal distinct photochemical behaviors for oxazole and isoxazole. In isoxazole, we observed O–N bond breaking within an average time of 41.46 fs, predominantly through the S2 → S1 → S0 relaxation pathway. For oxazole, although complete bond breaking was not observed within the simulation timeframe, significant C–O bond elongation with substantial geometric changes and ring puckering were noted, particularly at S1/S0 crossing points. We analyzed vertical excitation energies, simulated IR spectra, and tracked internal coordinates throughout the trajectory. We compared the results from our simulations with experimental ultrafast electron diffraction (UED) patterns and found good agreement between the two. These findings provide detailed insights into population dynamics, quantum amplitudes, and branching ratios for various photochemical pathways in both molecules. This work enhances our understanding of the fundamental photochemistry of five-membered heterocycles, which has significant implications for the design of photoactive materials and photochemical synthesis strategies, with potential applications in the development of advanced organic photovoltaics. By combining ab initio simulations with machine learning, this research opens new avenues for innovation in materials science and photochemistry.

D-16. Platform generation for precisely monitoring the targeted degradation of endogenous NTMT1

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Targeted protein degradation(TPD) is a promising therapeutic strategy for modulating disease-associated proteins, but achieving precise control over degradation processes remains a major challenge. This study aims to establish a robust platform for the efficient generation and evaluation of PROTACs targeting *N-terminal methyltransferase 1* (NTMT1). In this study, we constructed an *in vivo* platform for monitoring the degradation of endogenous NTMT1 using HiBit based NanoBiT technology. The NanoBiT system developed enables real-time, continuous monitoring of endogenous NTMT1 levels in live cells, thereby saving time, and providing a more accurate kinetic profile of NTMT1 degradation than using the Western blot. To enable real-time monitoring of NTMT1 degradation, CRISPR-Cas9 gene editing was employed to introduce a NanoBiT tag into the endogenous NTMT1 locus. This genetic modification allows for the visualization and quantification of NTMT1 protein levels in live cells. As a result, this NanoBiT technology enables the rapid identification and optimization of PROTACs, accelerating the drug discovery and development for NTMT1-related diseases.

D-17. SPECC1L C-terminal truncation results in behavioral differences and cerebellar Purkinje cell loss

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Autosomal missense variants in *SPECC1L*, encoding a cytoskeletal protein, lead to syndromic manifestation of mainly craniofacial anomalies. However, these same patients often express symptoms of intellectual and developmental disabilities. It remains unclear the role *SPECC1L* plays in neurodevelopment and behavior. We have identified several patients with autosomal dominant variants that lead to truncation of SPECC1L protein, where the majority present with neurodivergent behaviors (e.g. anxiety, aggression, learning disability). To model SPECC1L truncation variants in mice, we have generated a line that removes the C-terminal 510 amino acids (*DC510*). Behavioral experiments with adult heterozygous *DC510/+* mice showed reduced fear response and anxiety (Elevated Plus/Open Field) and differences in social preferences (3-Chamber: Social-Empty). Some *DC510/+* female mice also showed hyperactivity phenotype. Immunofluorescence of wildtype (WT) mouse brains showed high expression of SPECC1L in the cerebellum. Consistently, we observed a loss of cerebellar Purkinje cells in *DC510/+* mice. Proteomic analysis of 10-week-old WT and *DC510/+* cerebellums from both males and females showed differential expression of several proteins involved in intracellular transport ($n_{♀}$=26, $n_{♂}$=52). At the molecular level, we have shown that SPECC1L associates with microtubules for cellular trafficking and with filamentous actin (F-actin) to cause turnover. In SPECC1L-DC510, the C-terminal actin-binding domain is lost, thus untethering the truncated protein. In *DC510/+* cerebellar tissue, we observed misexpression of neurofilament proteins as well as SPECC1L-DC510 in the molecular layer. We posit that loss of SPECC1L association with F-actin results in cytoskeletal changes affecting intracellular protein transport and loss of neuronal maintenance, resulting in atypical neurodevelopment.

D-18. Integrating Machine Learning-Assisted Protein Design and Analysis into Rosetta

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With the growing usage of protein language models (PLMs), it is increasingly vital for biomolecular software applications to adapt machine learning (ML) predictions for new and existing workflows. In this study, we use the protein design software Rosetta as a prototypical case to examine current Rosetta-integrated PLMs and expand its implementation coverage for ML models.
We build upon recent changes to the Rosetta codebase and software licensing agreements to implement combined interfaces for ProteinMPNN (Dauparas, et al., 2022) and LigandMPNN (Dauparas, et al., 2023). Our tests against current ML workflows in Rosetta show that our integrations are more accessible to users and more performant compared to their manually implemented counterparts.
Initial benchmarks with LigandMPNN and AlphaFold2 reveal bottlenecks in round-trip design pipelining. To address this, we introduce a templating framework that automatically generates software integrations with minimal additional code. This framework allows for the import of state-of-the-art ML models into existing biomolecular software.
Finally, our work enables the export of these integrations into various interfaces, including scripting and programming languages like Python, R, or C++, and graphical interfaces for direct usage in biomolecular design. This advancement significantly enhances the usability and performance of biomolecular software applications.

D-19. Dopamine signaling modulates partner-seeking behavior in the socially monogamous prairie vole (*Microtus ochrogaster)* during social loss.

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Grief often involves a sense of “yearning” for a lost loved one, which can disrupt daily activities. Recent research looking at receptor expression and behavioral differences in the loss state suggests this preoccupation may be driven by hyperactivity of dopamine signaling in the anterior cingulate cortex (ACC) and insular cortex (IC). This hypothesis was explored here using the socially monogamous prairie vole (*Microtus ochrogaster*) and pharmacological manipulation. Male voles were paired with an opposite-sex partner for one week, and bond status was assessed via a partner preference test (PPT), resulting in three groups: non-bonded loss (NBL), pair-bonded intact (PBI), and pair-bonded loss (PBL). One week later, subjects underwent an odor preference test (OPT) for partner vs. food scented bedding. Prior to OPT, voles were bilaterally and site-specifically infused with vehicle (aCSF), dopamine receptor antagonist (haloperidol), or dopamine receptor agonist (apomorphine), targeting the ACC or IC. Time spent in proximity and actively investigating scented bedding was analyzed. Results showed that PBL animals spent significantly more time investigating partner odor compared to NBL/PBI controls. Haloperidol administration eliminated this difference, reducing partner odor investigation in PBL males to control levels. Apomorphine administration to NBL/PBI groups is trending towards non-significant results when compared to vehicle controls. This is the first study to demonstrate a causal link between dopamine signaling in the ACC and IC and increased partner-associated cue seeking during partner loss. Future research is needed to explore the molecular mechanisms behind this apparent dopamine signaling dysfunction in the loss state.

D-20. FORCE: Feature-Oriented Representation with Clustering and Explanation

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To improve deep learning model performance, researchers have explored capturing latent structures in data to identify patterns that are not directly observable. Traditional cluster analysis, often based on raw feature values, may provide insights already derivable by complex models and fail to capture unobservable data structures effectively. To address this, we propose a SHAP-based supervised deep learning framework, FORCE, which leverages SHAP (SHapley Additive exPlanations) values to enhance training by capturing latent structures.
FORCE integrates SHAP values into a neural network architecture in two ways: (i) clustering SHAP values and utilizing the cluster labels as an additional feature to guide training, and (ii) to initiate an attention mechanism. By integrating these elements, FORCE provides the network with information about unobserved values that modify feature importance for individual observations, improving predictive performance.
The framework was evaluated on three real-life datasets, demonstrating superior results compared to networks without the clustering and SHAP-based attention mechanism. For instance, FORCE achieved an F1 score of 0.80 for predicting heart disease presence or absence, compared to 0.72 without these enhancements. Using SHAP value clusters instead of raw feature clustering offers a more insightful approach, enabling models to better capture latent patterns. Incorporating cluster assignments and SHAP-initiated attention enhances the model's discriminative capabilities, improving predictive accuracy and overall performance in complex tasks.

D-21. The three-component signal transduction system YesLMN of *Enterococcus faecalis* senses host glycans to activate expression of an ABC transporter required for host glycan import

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Enterococcus faecalis, an opportunistic pathogen that normally inhabits the gut in humans has the capacity to utilize a wide range of carbohydrate sources. In *E. faecalis*, σ^{54} (RpoN) controls the expression of multiple phosphotransferase systems (PTS) responsible for metabolism of carbon. Through previous work in our lab, it was shown that in the absence of RpoN, alternative carbon sources are required to maintain bacterial growth when grown on glucose as the principal carbon source. Prior transcriptional analysis comparing parental strain V583 with its isogenic rpoN deletion identified the most differentially expressed genes in the *rpoN* mutant comprising an operon that includes a predicted ABC transporter, EF2223-21 and a three-component signal transduction system (YesLMN). Since YesN is a predicted response regulator, we constructed a *yesN* mutant and assessed its contribution to the regulation of the operon, as well as potentially other genes regulated by YesN by RNA-seq analysis and confirmed the transcriptomic data by qRT-PCR and luciferase promoter fusions. To assess the contribution of YesL and YesM, a predicted ancillary membrane protein and a membrane bound sensor histidine kinase, we constructed in-frame deletion mutants of both genes and complemented those defects by use of an ectopic integration system to address potential polar effects on YesN regulation and activity. A luciferase reporter transcriptionally fused to *ef2223* (the first gene in the operon) allowed us to also address the host glycans that are sensed in a YesLMN-dependent manner and data show that high-mannose type N-linked glycans are sensed by YesLMN.

D-22. A Novel Cancer Therapeutic Treatment: Up-Regulating the cGAS-STING Pathway via HuR Inhibition in Prostate Cancer

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Prostate cancer is the most common cancer type in men and the second leading cause of cancer deaths in the United States. Cancer immunotherapies have been applied in prostate cancer treatment and achieved significant success compared to traditional methods; however, they still face remaining challenges such as low response rate in patients. Therefore, discovering new approaches is crucial to enhance the treatment efficacy and patients' survival. The cGAS-STING pathway is a cytosolic dsDNA sensor that is part of the innate immune system and respond to cancer cells. Human antigen R (HuR), also known as HuA or ELAVL1, is an RNA-binding protein that has been studied as a potential therapeutic target in cancer treatment. HuR is involved in various post-transcriptional regulatory processes, including mRNA splicing, maturation, nuclear export, stability, and translation. Consequently, HuR dysfunction contributes to various diseases, including cancers; and ubiquitous cytoplasmic HuR levels are found in various cancer types. Our lab has developed HuR small molecule inhibitors (KHs) that inhibit the HuR-mRNA interaction. We hypothesize that HuR plays a role in cancer immune resistance by down-regulating the cGAS-STING pathway, HuR inhibition can overcome the immune-resistance and improve the response of cancer immunotherapies. This study will offer a promising strategy to improve cancer immunotherapy and further research is required to understand the mechanisms of actions on how HuR regulates the cGAS-STING pathway, hence lead to a novel therapeutic cancer treatment.

D-23. Unlocking the Gut-Brain Connection: Targeting the Microbiome to Relieve Visceral Hypersensitivity and Restore Function in Irritable Bowel Syndrome

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Disorders of gut-brain interactions (DGBIs), including irritable bowel syndrome (IBS), rank among the most common gastrointestinal disorders, marked by recurrent abdominal pain and altered bowel habits in the absence of structural disease. Current IBS treatments provide limited pain relief, primarily addressing symptoms rather than targeting the underlying pathophysiology. Visceral hypersensitivity (VH), heightened sensitivity to bowel distention, is a key peripheral factor driving pain in DGBIs, yet remains poorly understood. Recent studies suggest a link between gut microbiome dysbiosis, intestinal permeability, and visceral hypersensitivity (VH), but it remains unclear if these changes drive or result from IBS. We have previously identified specific differences in gut microbial colonization between VH-susceptible C57BL/6NTac (BL/6NTac) and VH-resistant (C57BL/6J) mice in a zymosan (ZYM)-induced IBS model, with VH-susceptible mice exhibiting *Firmicutes (Bacillota)* enrichment, specifically *Lachnospiracaea Dorea*, similar to findings from IBS patients. To further understand how the microbiome plays a role in ZYM-VH, we developed and validated an antibiotic (ABX) treatment protocol followed by a fecal microbiota transplant (FMT). Transplanting fecal supernatant from ZYM-VH mice into naive mice caused visceral hypersensitivity, increased colonic *Tlr2* expression indicative of elevated immune response, and increased intestinal permeability. We are currently investigating whether selective reduction of bacteria associated with IBS phenotype can restore normal gut function and promote recovery. Using the ZYM-IBS mouse model, we are using targeted antibiotic treatment to see if we can reverse key symptoms like pain, intestinal dysfunction, and increased intestinal permeability. These findings support microbiome-targeted interventions as a promising approach for managing VH in IBS.

D-24. THE ROLE OF TUMOUR MICROENVIRONMENT(TME) IN GLIOBLASTOMA THERAPEUTIC RESISTANCE

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Glioblastoma (GBM) is the most aggressive primary brain tumour in adults, with a median survival of less than 15 months despite multimodal therapies, including surgery, radiotherapy, and chemotherapy. Therapeutic resistance remains a formidable challenge driven by both the intrinsic properties of cancer cells and the complex tumour microenvironment (TME). The TME, consisting of non-cancerous cells, blood vessels, and signalling molecules, promotes tumour progression and shields cancer cells from therapeutic interventions.
This study investigates the TME's role in GBM therapeutic resistance, focusing on three key aspects: (1) TME contribution to therapy resistance; (2) the cellular and molecular components that enhance tumour survival and inhibit treatment efficacy; and (3) the mechanisms by which factors such as tumour-associated macrophages (TAMs), hypoxia, and the blood-brain barrier (BBB) amplify resistance. To address these objectives, this research employs bioinformatics analysis of publicly available transcriptomic and proteomic datasets to identify key molecular pathways and interactions within the TME. Spatial transcriptomics will map cell-specific gene expression patterns, and network analysis will identify therapy resistance signaling pathways. Experimental validation through in silico simulations and data integration from single-cell RNA sequencing (scRNA-seq) studies will further elucidate the functional roles of TME components.
The study seeks to identify new therapeutic targets in the TME to improve treatment effectiveness and provide critical insights into the interplay between the TME and GBM thus, paving the way for innovative and more effective therapeutic strategies to combat this highly resistant malignancy.

D-25. Loss of *Specc1l* Disrupts the Development of the Blood-CSF Barrier resulting in Embryonic Edema and Ventriculomegaly

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Ventriculomegaly affects 1 in 500 live births. It occurs when cerebrospinal fluid (CSF) accumulates in brain ventricles, increasing ventricular size, which can result in brain compression and impaired brain function. CSF production occurs in the choroid plexus (CP). The main constituent of CSF is blood plasma, and CSF production dynamics are controlled by the blood-CSF barrier. CP development begins at embryonic day (E) 8.5 with invagination of the roof plate of the neural tube. At E11.5 increased WNT/BMP signaling induces a significant period of growth and development resulting in a functional barrier that resembles that of an adult by E14.5. SPECC1L is a cytoskeletal scaffolding protein that interacts with actin, microtubules, and junctional proteins. SPECC1L is expressed broadly, including in the ependyma of ventricle walls and CP. Individuals with autosomal dominant *SPECC1L*-related syndrome frequently manifest ventriculomegaly. To understand the pathogenetic mechanism, we studied the *Specc1l* null mice that show perinatal lethality. At E13.5, *Specc1l* null mutant embryos developed edema along the cranium and spine. Lateral ventricles were enlarged two-fold in mutants at E16.5, with abnormally branched and structurally disorganized CP in the lateral ventricles. Staining for junctional markers showed abnormal patterning of membrane-associated β-catenin (adherens junctions), Occludin (Tight Junctions) and ZO-1 (multiple junctions). Both primary and motile cilia were shorter in the *Specc1l* mutant ependymal cells, which could also contribute to ventriculomegaly as seen in some ciliopathies. Together, we describe a novel role for SPECC1L in the blood-CSF barrier that underlies the ventriculomegaly observed in *SPECC1L*-related syndrome.

D-26. Nrf2 regulates the activation-driven expansion of CD4$^+$ T-cells by differentially modulating glucose and glutamine metabolism

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CD4$^+$ T-lymphocytes (T-cells), a subset of adaptive immune cells, orchestrates immune responses and maintains host immunity. Reactive oxygen species (ROS), the metabolic byproducts of cellular metabolism are also known to serve as second messengers of immune responses. In mammalian cells, ROS levels are tightly regulated by the antioxidation complex, composed of Kelch-like ECH-associated protein 1 (Keap1), nuclear factor erythroid 2–related factor 2 (Nrf2) and cullin 3 (Cul3) proteins. Keap1 regulates the levels of Nrf2, a transcription factor that activates anti-oxidation response element. Nrf2 is a well-known oxidative stress regulator. However, the role of Nrf2 in conventional CD4$^+$ T-cell homeostasis or activation-driven effector responses remains unclear. Here, we elucidate the role of Nrf2 beyond the traditional antioxidation, in modulating the activation-driven expansion of CD4$^+$ T-cells by influencing their nutrient metabolism. T-cell-specific activation of Nrf2 enhances early activation and IL-2 secretion, upregulates TCR-signaling, and increases activation-driven proliferation of activated CD4$^+$ T-cells. Mechanistically, high Nrf2 alters glucose metabolism but promotes glutamine metabolism via glutaminolysis to support increased T-cell proliferation. Further, high Nrf2 in activated CD4$^+$T-cells lead to increased chromatin accessibility and proliferation-associated gene expression. Overall, our findings uncover a novel role of Nrf2 as a metabolic modulator of CD4$^+$ T-cell expansion, thus providing a framework for improving Nrf2-targeting therapies.

D-27. SUSTAINED EXPRESSION OF DCLK1-S PROMOTES INFLAMMATION AND TUMORIGENESIS IN THE COLON

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Doublecortin-like kinase-1 (DCLK1) plays a chemosensory role in the gut. Its role in inflammatory diseases, including inflammatory bowel disease (IBD), has not been thoroughly investigated. Alternative promoter usage generates long and short isoforms (DCLK1-L and DCLK1-S) of DCLK1. This study explored the role of the DCLK1-S isoform in promoting colitis and colon cancer. Transgenic mice (*Dclk1*$^{\Delta IEC}$, *Dclk1-L*$^{\Delta Neu}$) were either infected with *Citrobacter rodentium* (CR) or given g-secretase inhibitor DBZ, and tissues/cells were processed via standard techniques. Following CR-induced infectious colitis in mice, we observed an accumulation of DCLK1-S in the colons of infected mice that inversely correlated with DCLK1-S repressor FoxD3 (Forkhead Box D3). Heightened DCLK1-S levels also corresponded with higher MMP13 staining/activity at peak crypt hyperplasia, while a decline in MMP13 in the CR+DBZ group correlated with collagen accumulation and fibrosis. To further elucidate the role of DCLK1-S in driving the progression of colitis to colon cancer, we bred *Dclk1*$^{fl/fl}$ mice with *MRP8-Cre-ires/GFP* mice to generate *Dclk1*$^{fl/fl}$*;MRP8-Cre*$^{+/-}$ that eliminates DCLK1-L (*Dclk1-L*$^{\Delta Neu}$) upon tamoxifen injection resulting in sustained expression of DCLK1-S in the granulocytes especially neutrophils. When infected with CR or given AOM/DSS, *Dclk1-L*$^{\Delta Neu}$ mice, compared to WT, developed severe colitis and exaggerated tumorigenesis throughout the colon. When *Dclk1-L*$^{\Delta Neu}$ mice were treated with an NE inhibitor, we observed a phenotypic difference in the colons of mice treated with the inhibitor. While more studies are needed, our findings suggest that DCLK1-S overexpression in neutrophils causes inflammation and DNA double-strand breaks in the colonic crypts that are likely to precede tumorigenesis in the colon.

D-28. Oxidative stress regulator NRF2 inhibits inflammatory CD4 T-cell differentiation and protects against inflammatory bowel disease progression.

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Inflammatory bowel disease (IBD) is an incurable condition characterized by chronic inflammation in the gut. A disbalance in inflammatory and regulatory T-cell subsets is a major contributor. In this study, we aimed to identify if and how NRF2 (nuclear factor erythroid 2–related factor 2), an oxidative stress regulator regulated by Keap1 (Kelch-like ECH-associated protein1), impacts the differentiation of inflammatory (Th1/Th17) and regulatory (Treg) CD4 T-cell subsets and in turn, the disease outcome of IBD. To answer this, we tested the effects of an FDA-approved NRF2 activator RTA-408 on T-cells *in vitro*, followed by *in vivo* mouse models of IBD. Using mice with T-cell specific knockout (KO) of NRF2 (NKO, low NRF2) and Keap1 (K-KO, high NRF2) we further dissected out NRF2-driven immune mechanisms. CD4+ T-cells with high NRF2 activity (K-KO) depicted decreased differentiation into the inflammatory T helper 1 (Th1) subset and increased differentiation into the Foxp3+ regulatory T-cells (Treg). To explore the *in vivo* significance of T-cell intrinsic NRF2 in IBD progression, we performed adoptive transfers of T-cells from WT and KKO mice into immunodeficient RAG1 KO mice to develop T-cell-driven IBD. T-cells from KKO (high NRF2) elicited a significantly lower disease severity compared to those receiving T-cells from WT. Overall, these results highlight NRF2 as a novel therapeutic target for treating IBD and other inflammatory disorders.
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D-29. How do new cell types arise?

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How new cell types arise is a central question in cellular biology. While genetic changes can result in the evolution of new cell types in multicellular organisms, it remains unclear whether the development of cellular differentiation can be ancestrally plastic before coming under developmental-genetic control via genetic assimilation. Using the volvocine green algae as a model system, we utilize experimental evolution to address the question of whether the plastic development of a new cell type preceded its fixation via genetic assimilation. We have previously shown that a *Eudorina* species historically characterized as undifferentiated develops a small proportion of plastic somatic cells following exposure to cold shock. Here, we exposed a clonal *Eudorina* culture to multiple rounds of repeated cold shock and characterized the differentiation status of our lines more than 30 generations after the cessation of the cold treatment. We found that the repeatedly cold-shocked lines had an increased proportion of colonies with somatic-like cells. We also found that cellular differentiation was obligate in one cold shocked lineage, which always had somatic cells and had a higher proportion of somatic cells per colony than other lineages. This shows that genetic assimilation can lead to the rapid evolution of cellular differentiation. We conclude that new cell types can evolve through genetic assimilation and that repeated stressors can potentially both generate genetic variation in differentiation and selectively favor differentiation, resulting in the rapid evolution of cellular differentiation.

D-30. *Thm1* heterozygous female mice protect against cleft palate in offspring due to uterine cytoskeletal changes and increased expression of nutrient receptors

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Craniofacial anomalies accompany a third of all birth defects, with cleft palate (CP) alone affecting 1/1200 newborns. Autosomal dominant mutations in the cytoskeletal gene, *SPECC1L*, result in syndromic CP. Mirroring this in mice, *Specc1l*$^{DCCD2/+}$ heterozygotes manifested 16% CP. Further, when combined with loss of one allele of a primary cilia gene, *Thm1*$^{+/-}$, CP incidence in double heterozygous embryos increased to 34%, indicating a genetic interaction. Surprisingly, this CP occurrence was observed only when *Specc1l*$^{DCCD2/+}$ females were crossed with *Thm1*$^{+/-}$ males. When parental genotypes were reversed, CP was not observed in offspring of *Thm1*$^{+/-}$ mothers. Using control crosses, we determined that maternal *Thm1*$^{+/-}$ heterozygosity was protective against CP. We proposed that *Thm1* heterozygosity leads to changes in maternal uterine tissue that positively influence fetal development. Consistent with this, global proteomics analysis revealed that uterine tissue of *Thm1*$^{+/-}$ mothers had increased abundance of proteins in Gene Ontology categories of Response to Nutrients and Placenta Development. Validation of these results showed increased expression of folate receptor (FOLR1) and one component of Cubam (B12) receptor, CUBN. Phospho-proteomic analysis revealed a decrease in Rho signaling via ARHGAP17. This was validated through filamentous actin staining which was decreased in *Thm1*$^{+/-}$ uterine tissue, suggesting cytoskeletal changes may affect membrane trafficking or turnover of nutrient receptors. To our knowledge, maternal *Thm1* heterozygosity is the first mouse model of a protective genetic effect against a birth defect. These studies are generating novel insights into the role of maternal environment in the etiology of isolated CP complex disease.

D-31. 3' Nucleotide Asymmetry Directs miRNA Strand Selection

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microRNAs (miRNAs) are central regulators of gene expression and are essential for animal development. During miRNA biogenesis, stem-loop miRNA precursors are processed into a double-stranded duplex that is loaded into Argonaute (Ago). The guide strand is retained within Ago to assemble the miRNA-induced silencing complex (miRISC) whereas the passenger strand is discarded. As each miRNA strand is expected to have different targets, strand selection effectively determines the target repertoire of miRISC. Previous studies have suggested that 5' nucleotide identity and thermodynamic asymmetry of duplex ends are major drivers of strand selection. However, strand choice of many miRNAs cannot be explained by these mechanisms, suggesting that additional factors promote strand selection. Here, we generated a series of mutations within endogenous *C. elegans* miRNAs to identify duplex features important for strand selection *in vivo*. Our data suggests that while a 5' uracil is favorable, it is not sufficient to specify strand choice. Surprisingly, the thermodynamic stability of each duplex end poorly predicted strand choice, suggesting thermodynamic asymmetry is dispensable for strand selection. We identified a bipartite motif comprising the 5' nucleotide of the guide strand and the 3' nucleotide of the passenger strand that strongly predicted strand selection of both endogenous miRNAs and described the behavior of genome-edited variants. Mutating the 3' nucleotides of the miR-58 duplex was sufficient to reverse strand selection in *C. elegans* and human cells, supporting that 3' nucleotide identity directly influences strand selection. We propose that discrete nucleotide preferences within miRNA duplex ends determines asymmetric strand choice.

D-32. *De novo* Generation of Outer Membrane β-Barrel Sequences with Bidirectional LSTM

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Protein sequence space is vast. With twenty options, computers are required for effective sequence space exploration. We have investigated *de novo* design of full-length sequences of transmembrane β-barrel proteins (TMBB) using long short-term memory (LSTM) models. We assess the ability of a small generative model to explore uncharted protein sequence space within this structural fold. Our training procedure is efficient because of LSTM's low complexity. Comparatively, the number of parameters (161,302 total trainable parameters for 128 nodes per LSTM cell) is ~2% of those used in transformers. We found that the generated proteins computationally resemble the desired barrel fold by correctly capturing statistics of the primary structure and reproducing the β-signal, a C-terminal sequence motif of true TMBBs necessary for insertion in the outer membrane. We also find that synthetic sequences generated reproduce characteristics of native TMBBs well enough that the designs are readily predicted as TMBBs by three independent predictors that do not directly rely on homology. The sequences have low similarity to known TMBBs. Our results indicate that for the TMBB family it is possible to successfully train low complexity generative models that efficiently explore uncharted sequence space.

D-33. Optimizing Tissue Clearing of Embryonic Palatal Shelves to Visualize the Actin Cytoskeleton in 3D

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The process of tissue clearing has revolutionized the field of developmental biology, enabling researchers to visualize intricate three-dimensional structures within biological tissues. We aimed to use tissue clearing to study embryonic palate closure, failure of which leads to cleft palate – a common craniofacial birth anomaly. The embryonic palatal shelves (PS) present unique challenges for tissue clearing due to their dense cellular composition and complex morphology. Traditionally, PS have been analyzed using 2D coronal sections, with most studies focusing on select sections from the anterior, middle, and posterior regions. However, our magnetic resonance imaging-based 3D structural analysis of intermediate stages of palate closure revealed dynamic anteroposterior movements. We propose that these movements rely on supracellular actomyosin organization, which is not easily captured in 2D sections. We systematically evaluated E13.5 embryos using various tissue clearing protocols, including organic solvent-based (iDISCO), aqueous-based (SeeDB, CUBIC), and hybrid (FRUIT, combines SeeDB and urea) methods, to identify the most effective approach (modified SeeDB at 37°C) for preserving tissue integrity and enhancing transparency while visualizing actomyosin cytoskeleton. We integrated this optimized tissue clearing method with spinning-disc confocal imaging to generate 3D images to assess cell shape, nuclear orientation, and filamentous actin organization across the cleared PS. We are now extending this analysis to PS from *Specc1l* mutant embryos that show abnormal actin cytoskeletal organization and PS closure delay. Our optimized clearing and imaging technique offers a powerful tool for investigating craniofacial development and will provide new insights into the spatial and temporal cytoskeletal dynamics underlying PS closure.

D-34. Infectious Disease Assay Development Core: High Throughput Screening Laboratory at the University of Kansas

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The overall goal of the IDAD Core is to provide expertise, facilities, services, and training in the area of HTS assay design, development, validation, small and large-scale screening for whole cell based or biochemical infectious disease targets. The IDAD core is an extension of the University of Kansas High Throughput Screening Laboratory which is a fee-for-service, state-of-the-art facility dedicated to providing academia, not-for-profit institutions, biotech, and pharmaceutical industries with exceptional assay development, high throughput screening and data mining services at economical rates. The staff has experience in executing cell-based, biochemical, siRNA as well as high content screening campaigns against a plethora of target classes. The laboratories are equipped with cutting-edge liquid handling and signal detection instrumentation for increasing throughput and precision of screening campaigns. Clients have the option of using our collection of 395,000 compounds and/or a client's own chemical library. KU-IDAD/HTS lab further leverages the strengths of the medicinal chemistry/ computational modeling cores under CoBRE Chemical Biology of Infectious diseases (CBID) program to support your tool/lead discovery research.

D-35. Using Camera Trapping to examine the Effects of Wetland dissection on Vertebrate Activity and Community Structure.
Benally, Aysa,1 Bridgett Chapin1, Courtney King1, Department of Environmental Science

At the Haskell wetlands (Lawrence, KS), camera trapping has been used to identify terrestrial and aquatic animals and their usage of the wetlands as well as migration corridors through South Lawrence Trafficway infrastructure in the surrounding area. During summer 2024, I continued this Haskell research of characterizing the vertebrate populations of the wetlands while increasing emphasis on monitoring wildlife use of the highway underpass structures on the east and the west sides of the wetlands. Additional cameras and site locations were added on the east side to capture sensitive species as previous monitoring efforts there were unsuccessful. Results show that the west underpass had more vertebrate traffic compared to the east underpass. The west underpass had 40% more species sightings compared to the east. We hypothesize that the presence of more natural habitat along the corridor (e.g., higher proportion of vegetation cover, etc…) make the west underpass a more protected pathway for animals. In the east underpass, there was less vertebrate traffic, and the corridor is more disturbed with large areas of riprap and less vegetation cover. This summer season data sheds light on how the dissection of the Haskell wetlands by the trafficway has affected animal migration and distribution. Research demonstrating wildlife corridor benefits in highway projects has implications for engineers during the consultative design/build decision-making process and could potentially inform the KDOT expansion of the South Lawrence Trafficway to decrease wildlife accidents.

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