The 17th Annual Kansas-IDEA Network of Biomedical Research Excellence Symposium

January 19-20, 2019

Overland Park Sheraton
Overland Park, KS

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LOCATION OF EVENTS

- Registration: Lobby Level/ Leatherwood Foyer
- Friday Night Events: Cottonwood Ballroom and Foyer
- Breakfast: Cottonwood Ballroom and Foyer
- General Session: Leatherwood Ballroom
- Breaks: Leatherwood Foyer
- Lunch: Cottonwood Ballroom and Foyer
- Poster Sessions/Reception: Cottonwood Ballroom
- Dinner: Leatherwood Ballroom
- Boxed Lunches: Leatherwood Foyer
K-INBRE 2019 Symposium
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Poster Presentations

Saturday, January 19, 2019
Poster Session I (3:30-4:30 PM) Abstracts Numbered 1-40
Poster Session II (4:30-5:30 PM) Abstracts Numbered 41-80
Poster Session III (5:30-6:30 PM) Abstracts Numbered 81-103

Sunday, January 20, 2019
Poster Session IV (9:40-10:25 AM) Abstracts Numbered 104-136
Poster Session V (10:25-11:10 AM) Abstracts Numbered 137-173

See Alphabetical list for monitor assignment.
Abstract# = Monitor #

Please be near your monitor during your assigned session until the judges have visited.
Feel free to visit other boards during the alternate sessions.

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 2019 Symposium
Program Schedule
Overland Park Sheraton
Overland Park, KS

Friday, January 18, 2019

3:00 PM  
Early Registration  
Foyer

Open Poster viewing  
Cottonwood Ballroom

4:30 PM  
Early Registration Closes

6:00 PM  
Friday Night Event and Dinner

8:00 PM  
Poster Viewing, Friday Night Event and Dinner Ends

Saturday, January 19, 2019

7:30 AM  
Breakfast Buffet  
Cottonwood Ballroom/Foyer

Registration  
Foyer

8:30 AM  
General Session  
Leatherwood Ballroom

Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center
Opening Remarks

9:15 AM  
Gregory Finnigan, Ph.D. Assistant Professor, Kansas State University
Moderator: Keynote and Regional Scientist Presentations

9:20 AM  
David Scott McVey, D.V.M., Ph.D., D.A.C.V.M., Director, Center for Grain and Animal Health Research, United States Department of Agriculture
Title: Vaccines: There and Back Again

9:55 AM  
C. Ron Yu, Ph.D., Investigator, Stowers Institute for Medical Research
Title: What is critical in the critical period of olfactory system development?

10:25 AM  
Break  
Foyer

University Photos

10:30 AM  
Haskell Indian Nations University Photo

10:35 AM  
Fort Hays State University Photo

10:40 AM  
Kansas State University Photo

10:45 AM  
Pittsburg State University Photo

10:50 AM  
University of Kansas Medical Center Photo

10:55 AM  
General Session  
Leatherwood Ballroom

E. Matthew Morris, Ph.D., Research Assistant Professor, University of Kansas Medical Center
Moderator: Regional Scientist and Trainee Presentations

11:00 AM  
Adam Smith, Ph.D., Assistant Professor, University of Kansas, Lawrence
Title: Wired for social relationships

11:30 AM  
Joshua Miller, Kansas State University, Manhattan, KS
Title: Novel Pilot Study Reveals that Heat Therapy Increases Muscle Mitochondrial Quality Control and Respiratory Efficiency in Healthy Human Subjects
Saturday, January 19, 2019

11:50 AM  Cayla Moore, Langston University, Langston, OK
Title: Hydrocortisone Decreases the Proliferation Rate and Increases Antibiotic Resistance of Streptococcus Pneumoniae

12:10 PM  Lunch  Cottonwood Ballroom/Foyer

1:15 PM  General Session  Leatherwood Ballroom

Jianzhong Yu, Ph.D., Assistant Professor, Kansas State University
Moderator: Regional Scientist and Trainee Presentations

1:20 PM  Prachee Avasthi, Ph.D., Assistant Professor, University of Kansas Medical Center
Title: A tale of two actins: significant functional overlap of divergent actin isoforms in the unicellular green alga Chlamydomonas reinhardtii

1:50 PM  Vaughn Craddock, University of Kansas, Lawrence, KS
Title: Genetic Adaptations Conferring Quorum-Sensing Dependent Antibiotic Resistance in Evolved Strains of Pseudomonas aeruginosa

2:10 PM  Annabelle Dillon, Kansas State University, Manhattan, KS
Title: Impact of reduced liver PGC1a during development on adult diet-induced weight gain

2:30 PM  Erina Kutilek, Wichita State University, Wichita, KS
Title: The Role of Myopalladin in Cardiac Muscle Functions and Disease

2:50 PM  General Session Concludes  Foyer

University Photos

2:55 PM  Langston University Photo
3:00 PM  Emporia State University Photo
3:05 PM  University of Kansas, Lawrence Photo
3:10 PM  Washburn University Photo
3:15 PM  Wichita State University Photo

3:25 PM  Poster Judge Meeting  Maple Room

3:30 PM  Reception/Poster Session I  Cottonwood Ballroom

4:30 PM  Reception/Poster Session II  Cottonwood Ballroom

5:30 PM  Reception/Poster Session III  Cottonwood Ballroom

6:30 PM  Poster Session Ends

Dinner  Leatherwood Ballroom

7:00 PM  Award Presentations  Leatherwood Ballroom

John Stanford, Ph.D., K-INBRE Associate Director, University of Kansas Medical Center
Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center
Closing Remarks
Sunday, January 20, 2019

7:00 AM  Breakfast Buffet  Foyer
8:00 AM  General Session  Leatherwood Ballroom

  John Stanford, Ph.D., Associate Director, University of Kansas Medical Center
  Opening Remarks

8:05 AM  Stephanie Shames, Ph.D., Assistant Professor, Kansas State University
  Moderator: Regional Scientist and Trainee Presentation

8:10 AM  Fawwaz Naeem, Kansas State University, Manhattan, KS
  Title: Maintenance of muscle proteostasis

8:30 AM  Kendall Odle, Langston University, Langston, OK
  Title: Connecting Tumor Suppressor Genes to DNA Damage Response

8:50 AM  Shwetha Ramachandran, University of Kansas Medical Center, Kansas City, KS
  Title: Defective ciliary signaling in initiation of renal cystic disease in Thm1 conditional knock-out mice

9:10 AM  Jeffrey A. Thompson, Ph.D., Assistant Professor, University of Kansas Medical Center
  Title: A statistical approach to determining equivalent functional genomic differences across experiments

9:40 AM  Poster Session IV  Cottonwood Ballroom
10:25 AM  Poster Session V  Cottonwood Ballroom
11:10 AM  Poster Session V ends

11:15 AM  General Session  Leatherwood Ballroom

  John Stanford, Ph.D., Associate Director, University of Kansas Medical Center
  Introduction of Speaker

11:20 AM  James Beck, Ph.D., Assistant Professor, Wichita State University
  Title: RADseq techniques can be applied to DNAs derived from plant museum specimens

11:50 AM  John Stanford, Ph.D., Associate Director, University of Kansas Medical Center
  Oral Presentation Awards

12:00 PM  Doug Wright, Ph.D., K-INBRE Principal Investigator, University of Kansas Medical Center
  Closing Remarks

12:00 PM  Boxed lunches available for pickup  Foyer
12:00 PM  Hotel Checkout
Novel Pilot Study Reveals that Heat Therapy Increases Muscle Mitochondrial Quality Control and Respiratory Efficiency in Healthy Human Subjects

Joshua Miller1, Alex Von Schulze1, Kelly Fuller1, Colin McCain1, Clara Amat Fernandez1, John Thyfault1, and Paige Geiger1
1The University of Kansas City Medical Center – Department of Integrative and Molecular Physiology

We have shown that heat therapy (HT) in rodents protects against high-fat diet induced skeletal muscle insulin resistance and impaired glucose homeostasis. However, it remains unknown whether a similar effect can be translated to humans. The purpose of this pilot study was to determine if HT via hot water immersion was well tolerated in humans and altered mitochondrial metabolism in skeletal muscle. Healthy male and female participants (n=5/sex) received one bout of HT to raise core temperature to 38.5°C for 1h. Muscle biopsies were taken one week prior to HT and 24h post HT to determine whether HT impacted mitochondrial respiratory capacity and mitophagy. HT was well tolerated in all subjects and resulted in increased heart rate and reduced systolic and diastolic blood pressure. HT did not significantly alter maximal mitochondrial respiratory capacity at basal, state3, state3S, or uncoupled states with either palmitoyl-carnitine + palmitoyl-CoA (palmitate) or pyruvate + glutamate + malate (PGM) as substrates. However, HT tended to improve respiratory efficiency as indicated by a reduced coupling control ratio for PGM. This improved efficiency and maintenance of mitochondrial respiratory capacity occurred despite HT-induced reductions in mitochondrial content as inferred by citrate synthase activity. We believe this reduction in muscle mitochondrial content occurs as a result of mitophagy. Our hypothesis is supported by the observed reductions of the mitophagy related protein LC3II. It is possible that HT-induced enhancements in mitophagy increase mitochondrial quality control. Our data indicate that HT increases mitochondrial quality control and efficiency in human skeletal muscle.

Hydrocortisone Decreases the Proliferation Rate and Increases Antibiotic Resistance of Streptococcus pneumoniae

Cayla A. Moore1, Colette Ngo Ndjom, M.S.2 and Harlan P. Jones, Ph.D.2, 3
1Department of Biology, Langston University, Langston, Oklahoma 73050
2Institute of Molecular Medicine, University of North Texas Health Science Center, Fort Worth, Texas 76107

The ecosystems of microbial species that reside on and within humans play an important role in health and disease. Microbial endocrinology is a transdisciplinary field which bridges neurophysiology and microbiology, focused on understanding the relationship between neuroendocrine responses’ influence on microbial physiology. Such interplay is believed to have a significant impact on human health and disease.

Streptococcus pneumoniae (S. pneumoniae) is a common commensal and opportunistic pathogen of the respiratory tract. S. pneumoniae is the cause of significant mortality rates in the United States and worldwide, particularly among the chronically ill and individuals with poor immune function. Previous studies in our laboratory have demonstrated that corticosteroid hormone can directly influence the growth and virulence of S. pneumoniae. Researchers have also shown similar effects to norepinephrine.

The purpose of the current study was to examine the effects of hydrocortisone on the growth and antibiotic resistance. We hypothesized that hydrocortisone exposure would increase the growth and antibiotic resistance of S. pneumoniae. Growth curve analysis was performed to determine the effect of various concentrations by which hydrocortisone would influence the growth phase of S. pneumoniae. In addition, antibiotic resistance in the presence of hydrocortisone was determined by minimal inhibitory concentration (MIC) analysis.

Results demonstrated that hydrocortisone significantly decreased S. pneumoniae growth in a concentration-dependent manner. In two independent experiments, S. pneumoniae demonstrated increased resistance against penicillin/streptomycin in the presence of hydrocortisone. Our findings suggest a dichotomous relationship between hydrocortisone influence on growth and antibiotic resistance that may be influential in mediating its pathogenicity.

Genetic Adaptations Conferring Quorum-Sensing Dependent Antibiotic Resistance in Evolved Strains of Pseudomonas aeruginosa

Vaughn Craddock1, Rhea G. Abisado1, John Henry Kimbrough1, Nicole E. Smalley2, Aji A. Dandekar2, Josephine R. Chandler1
1Department of Molecular Biosciences, University of Kansas, Lawrence, KS, USA; 2University of Washington School of Medicine, Seattle, WA, USA

The opportunistic pathogen Pseudomonas aeruginosa is a major cause of nosocomial infections and mortality in cystic fibrosis (CF) patients. P. aeruginosa is known to rapidly adapt to antibiotics such as tobramycin. To study the evolution of antibiotic resistance in P. aeruginosa, we passaged strain PA14 with sublethal concentrations of tobramycin or another aminoglycoside, gentamicin. The antibiotic-passaged lineages had enhanced aminoglycoside resistance compared with the ancestral parent. We performed whole-genome sequencing to identify the mutations. In the gentamicin-resistant strain we found mutations in a gene called ptsP, which is known to regulate resistance through an efflux pump, MexXY. The tobramycin-resistant isolate had mutations in fusA1, a translation factor known to increase resistance through a poorly understood mechanism. These isolates also had mutations in ptsP, which codes for an enzyme involved in a nitrogen-responsive phosphotransferase system. Deletions in ptsP increase activity of LasR, a transcription factor involved in quorum sensing. Quorum sensing causes cell density-dependent increases in aminoglycoside resistance that are relatively small in laboratory strains. Interestingly, in all the tobramycin-adapted lineages resistance was substantially LasR dependent, while LasR involvement in gentamicin-adapted lineages was similar to the ancestral lab strain. These results suggest mutations in ptsP and fusA1 might increase resistance through mechanisms that rely on LasR. In the case of ptsP this may be through enhanced LasR activity. The results provide new evidence for the involvement of LasR and quorum sensing in antibiotic resistance and are important for understanding how P. aeruginosa adapts under antibiotic selection.

Impact of reduced liver PGC1α during adulthood on adult diet-induced weight gain

Annabelle B. Dillon1, Julie A. Allen, John P. Thyfault, and E. Matthew Morris2
1Department of Microbiology at Kansas State University, 2Molecular & Integrative Physiology, University of Kansas Medical Center

Liver-specific, PGC1α heterozygous (LPGC1α) mice have reduced liver mitochondrial respiratory function and increased high fat, high sucrose (HFHS) diet intake and weight gain compared to wild type. In this study, we examined if the neonatal loss of PGC1α is necessary for increased HFHS-induced weight gain. We utilized two mouse models: LPGC1α and heterozygous floxed PGCG1α mice injected with adeno-associated virus-cre recombinase (AAV-cre). All mice were housed at thermoneutral temperatures (82-84°F) with access to low-fat diet. We began the 7-day HFHS feeding studies at 12 weeks of age, and measured pre- and post- study body weight, and body composition. The AAV-Cre mice did not display a significant increase in body weight following HFHS compared to the AAV-GFP, while LPGC1α mice gained 20% more weight than wild type. However, we continued to see increased feed efficiency in LPGC1α compared to wild type mice. These data demonstrate that while a developmental reduction in liver PGC1α (LPGC1α model) led to increased diet-induced weight gain, the same results were not observed in a model where liver PGC1α was reduced during adulthood (AAV-Cre). Therefore, these data suggest that there may be developmental component influencing diet-induced weight gain in LPGC1α mice.
The Role of Myopalladin in Cardiac Muscle Functions and Disease

Kutleke, Eri A N., Vinay K. Kadarla, Moriah R. Beck
Department of Chemistry, Wichita State University

Myopalladin is a protein that is associated with proper formation of both the actin cytoskeleton and organization of sarcomere structure, specifically in the heart. Mutations in myopalladin have been linked to cardiomyopathy and may be due to the disruption of actin regulation in the sarcomeres. Our goals for this research are to discover the molecular mechanisms behind the assembly of sarcomere thin filaments as well as to learn about the functional consequences of myopalladin on thin filament assembly in cells. Palladin is a close relative to myopalladin that likewise contains multiple immunoglobulin (Ig)-domains and has essential, but still unclear roles in organizing the actin cytoskeleton. The Beck Lab has recently shown that Ig3 domain of palladin binds directly to F-actin and increases the rate of actin polymerization as well as stability of actin filaments. In order to show that myopalladin, which has a similar structure to palladin, also binds to F-actin; we will use purified myopalladin Ig3 wildtype and several mutants to directly measure 1) actin binding and crosslinking by utilization of co-sedimentation assays, 2) assembly kinetics and the functional consequences of mutations in myopalladin. Our aim is to provide new knowledge about the role that myopalladin plays in cardiac disease, function, and structure.

Sunday, January 20, 2019

Maintenance of muscle proteostasis

Fawwaz Naeem, David Brooks, Marta Stetsiv, Erika Geisbrecht
Department of Biochemistry and Molecular Biophysics, Kansas State University

Proteostasis is important for maintenance and homeostasis of the cellular proteome in normal and stressed cells such as muscles and neurons. When this system fails, protein misfolding and/or aggregation can result in neurodegenerative diseases and protein aggregate myopathies. Tension exerted by contractile muscle tissues requires continuous folding and refolding of proteins, like Filamin, eventually damaging the ability of such proteins to sense and transmit mechanical strain. The specialized autophagy pathway called Chaperone Assisted Selective Autophagy (CASA) helps recycle and turn over damaged proteins to maintain cell function. Here we characterize a role for the evolutionarily conserved co-chaperone protein BAG3 in the genetically amenable Drosophila model. Mutations in starvin (stv), which encodes for Drosophila BAG3, show novel protein aggregation phenotypes and muscle degeneration. These results were confirmed using RNAi knockdown approaches. We also developed a sensitized background assay to identify genes that function with stv to regulate muscle proteostasis. Two candidate stv-interacting genes were uncovered using this approach - one encodes for a novel kinase and the other encodes for jaguar (jar), or the plus-end directed motor Myosin VI. Our working hypothesis is that the kinase cooperates with BAG3/Stv to remove damaged proteins and Myosin VI functions to deliver damaged cargo to the site of protein degradation.

Connecting Tumor Suppressor Genes to DNA Damage Response

Kendall Odle, Jessica Rakijas PhD, Berenice Jimenez, Brad Olson PhD
Department of Biology, Langston University, Department of Biology, Kansas State University

DNA damage repair is essential in all domains of life. Accurate sensing and repair are vital to protect the genetic code so the next generation inherits the proper DNA sequences. An organism must combat a variety of DNA damaging agents and perform appropriate repair. Among photosynthetic organisms, sunlight is an essential source of energy for photosynthesis, yet the UV rays are harmful to DNA. DNA damage is a larger problem in aquatic, photosynthetic organisms because they’re sensitive to chemicals in their environment. Little is known about how these organisms sense DNA damage and repair it despite the myriad insults. The algae Chlamydomonas reinhardtii was used as a model organism to study the DNA damage response (DDR). It was chosen because it’s unicellular, genetically tractable, easy to grow, and has a sequenced genome. Based on BLAST comparisons of known animal DDR genes, we hypothesized the DDR in C. reinhardtii is similar to that in animals and that mutants of genes known to have a defective DDR in mammals are similar in C. reinhardtii. Because of this, we tested the genes ataxia telangiectasia-mutated (ATM) and ataxia telangiectasia and Rad3-related (ATR) to the DDR in C. reinhardtii. We obtained knockout mutants of ATM and ATR through an insertional mutagenesis. These mutants were subjected to DNA damage through doses of UV and zeocin to cause single and double stranded breaks respectively. The results suggested that ATM mutants are insensitive to and incapable of detecting DNA damage. ATR mutants were hypersensitive, indicating it’s required for sensing DNA damage. Our results provide support that the DNA damage pathway in Chlamydomonas reinhardtii is similar to that of mammals.

Defective ciliary signaling in initiation of renal cystic disease in Thm1 conditional knock-out mice

Shwetha Ramachandran, Luciane M. Silva, Ramanan Sivakumar, Sumedha S. Gunewardena and Pamela V. Tran
Department of Anatomy and Cell Biology, Kidney Institute, University of Kansas Medical Center, Kansas City, KS
Department of Integrative and Molecular Physiology, University of Kansas Medical Center, Kansas City, KS

Primary cilia mediate signaling pathways, and ciliary dysfunction leads to renal cystic disease. While multiple cellular and signaling aberrations contribute to disease progression, the molecular mechanisms that initiate renal cystogenesis remain unclear. Previously, we performed RNA sequencing on kidney lysates of ciliary mutant Thm1 conditional knock-out (cko) mice. We reasoned that genes misexpressed before cysts have formed and continue to be misexpressed during disease progression may contribute to renal cyst initiation. Ten genes, typically expressed by renal epithelial, endothelial and immune cells, met this criterion. In our validation of these genes, activated STAT3 appeared most prominently upregulated at the protein level in pre-cystic and cystic kidneys. Here we investigated the connection between the Thm1 ciliary defect and STAT3 activation. We treated control and Thm1 knock-down (kd) 293T human kidney cells with interleukin-6 and assessed STAT3 activation at multiple time points from 0 to 24 hours. Thm1 kd cells showed a more robust second wave of STAT3 activation than control cells, which was preceded by intense activation of ERK. These data suggest that THM1 regulates STAT3 and ERK signaling. To prove that STAT3 signaling initiates renal cystogenesis, we are administering a STAT3 inhibitor, C188-9, to Thm1 cko mice to attenuate disease. Together, our data suggest that cilia dysfunction leading to misregulation of gene expression in renal epithelial, endothelial and immune cells, and in particular, to increased STAT3 signaling, may initiate renal cystic disease. FDA-approved STAT3 inhibitors may present as candidate therapies for repurposing for renal cystic disease.
1. Mahreen Ahsan¹, Krishna M. Donavalli¹, and Dennis H. Burns¹  
¹Department of Chemistry, Wichita State University, Wichita, Kansas 67260, United States: Synthesis of Phosphatidylglycerol Receptor: Precursor Preparation

2. Zaid Alashqar¹, Anil Mahapatro¹  
¹Department of Biomedical Engineering, Wichita State University, Wichita, KS: Comparative analysis of Stereolithography (SL) based 3D Printing Technique Versus Digital Light Processing (DLP) 3D Printing.

3. Alcorn, P., M. Bergstrom, R. Kenner, Y. Kobayashi  
Department of Biological Sciences, Fort Hays State University: Characterization of Microbial Population in the Nasal Cavity of Pigs during Post-Weaning in Response to Probiotic Supplementation

4. Kasra Alizadeh, Brian D. Ackley  
Department of Molecular Biosciences, University of Kansas: An RNAi-based Screen to Identify Novel Molecules in the Nidogen Synaptic Pathway in Caenorhabditis elegans

5. Ambrosier, Morgan, Danica Kostner, Erica Nevarez, and Yass Kobayashi  
Department of Biological Sciences, Fort Hays State University: Identification and Characterization of Messenger RNA Encoding the Prion Protein in Channel Catfish

6. Briana Anderson¹, Anjie Anji², Meena Kumari²  
¹Langston University, ²Department of Anatomy and Physiology, College of Veterinary Medicine, Kansas State University: The Characterization of Exosomal Proteins from P19 Cells.

7. Theodore J. Ball and Dr. Stephanie Shames  
Kansas State University Department of Biology: Characterization of the interaction between the Legionella pneumophila effectors Lpg2505 and SidI

8. Jonathan Barnell¹ and Takrima Sadikot¹  
¹Department of Biology, Washburn University, Topeka, KS: Annotation of gene features in Drosophila takahashii using a computational-genomics approach

9. Erianna M. Basgall, Megan E. Goeckel, Yao Yan, and Gregory C. Finnigan  
Department of Biochemistry and Molecular Biophysics, 141 Chalmers Hall, Kansas State University, Manhattan, KS 66506 USA Transcriptional Regulation & Genetic Screening Using dCas9 in Saccharomyces cerevisiae

10. Molly Bassett, Susumu Ishiguro, Mayme Loyd, E.R. Azhagiya Singam, Jeffrey Comer and Masaaki Tamura  
Department of Anatomy and Physiology, Kansas State University College of Veterinary Medicine: A novel PD-L1 inhibitory peptide enhances CD8+ T cell-dependent cell death against lung cancer cells

11. Bender, Savannah, Dr. Tim Burnett  
Department of Biological Sciences, Emporia State University: Studying the role of Treg cells in mucosal tolerance using the DEREG transgenic mouse model

12. Whitney Bergman, Jacob Yonke, and Cheryl P. Jones  
Washburn University, Chemistry Department: Cloning of the Giardia lamblia Acetyl-CoA Synthetase Gene in Preparation for Structure-Function Studies

13. Benjamin Bunnell, Dr. Matthew Cook, Dustin Caldwell  
Washburn University: Effect of luteolin on ATK/GSK3-beta

14. Nicholas A. Burnett, Abrar Alizahrani, Leah Cuthill, and Anuradha Ghosh  
Department of Biology, Pittsburg State University, Pittsburg, KS: Ecology and Prevalence of Ticks and Tick-Borne Bacterial Pathogens in Southeast Kansas

15. Ariana Cecil¹, Mackenzie Thornton¹, Sarah Gillaspie¹, Tamara Potapova², Jennifer Gerton² and Katsura Asano¹  
¹Molecular Cellular Developmental Biology Program, Division of Biology, Kansas State University, Manhattan, KS 66506 ²Stowers Institute for Medical Research, Kansas City, MO 64110: The Oncogene cMYC Suppresses non-AUG Translation

16. Yusuf Ciftci¹, Brintha Parasumanna Girinathan¹,² and Revathi Govind*  
¹Division of Biology, Kansas State University, Manhattan, KS, USA. ² Current address, Department of Pathology, Harvard Medical School, Boston, MA, USA: Clostridioides difficile SinR' regulates toxin, sporulation and motility through protein-protein interaction with SinR

17. Dekevsha L. Cooper, Holly O’Neill  
Department of Chemistry, Washburn University: Preliminary Studies on More Affordable Effective Means of Homogenization and Quantitation of Tetrahydrocannabinol (THC) in Marijuana Case Samples

18. Corajean Cunningham¹, Sharon Lewis, PhD¹  
¹Langston University, Langston, OK, USA: Bioinformatics of Major Depressive Disorder

19. Callie Dallimore, Colin Dallimore, Marcia Schulmeister, Qiuyang Zhang  
Department of Physical Sciences, Emporia State University, Emporia, KS: Heavy metals threatening aquatic ecosystems and human health in sediments in the Tri-State Mining District
20. Max Murphy,1 Randy Nudo,2 David Guggenmos,2 Alexis Delgado,3,2  
1 Department of Bioengineering, University of Kansas Medical Center  
2 Department of Rehabilitation Medicine, University of Kansas Medical Center  
3 Avila University: Developing a controlled behavioral assay to test bilateral cortical plasticity in intact rats

21. Delperdang, Cade W., Sharifah Albraiki, Moriah R. Beck  
Department of Chemistry, Wichita State University: Investigating the importance of proline-rich motifs in palladin for VASP binding

22. Hawa Dembele†, Moritz Matting,6 Om Prakash, Yoonseong Park,6 Alvaro I. Herrera†  
†Department of Biochemistry and Molecular Biophysics, Kansas State University, Manhattan KS; 6Department of Entomology, Kansas State University, Manhattan KS: NMR Solution Structure of Ecdysis Triggering Hormone Peptide from the African Malaria Mosquito Anopheles gambiae

23. Rachel Diebold and Stephen Fields  
Department of Biological Sciences, Emporia State University: Motorless myosin V transcripts expressed in mouse brain

24. Riley G Drees†, Andrew Tucker†, Mitchell J. Greer†  
†Department of Biological Sciences, Fort Hays State University; †Department of Agriculture, Fort Hays State University: Restoring Magnesium to the world’s food source through soil nutrient additions

Kansas State University: Analysis of Non-AUG Translation Initiation using Molecular Dynamics Simulations

26. Elliott, Jorga E, Naomi C. Quispe, and Brian R. Maricle  
Department of Biological Sciences, Fort Hays State University: Effects of sulfide, ethanol, and lactic acid on cytochrome c oxidase and citrate synthase activities in plant roots

27. Rebekah Elliott, Bayan Ahmad Dous, Saloni Darji, Tuhina Banerjee, and Santimukul Santra†  
Chemistry, Pittsburg State University: Synthesis of Doxorubicin-Based Prodrug and Activatable MR Nanoprobe For the Imaging and Treatment of Cancer

28. Mariah Emily†, Ryan W. Mentzer†, Elizabeth McCuiston†, Tim G. Burnett†, and Melissa M. Bailey†  
†Department of Biological Sciences, Emporia State University, Emporia, Kansas 66801: Changes in Cyp2b10 Gene Expression During Cyclophosphamide Exposure and Maternal Restraint Stress

29. Hannah Fairchild and Sam Leung  
Washburn University Chemistry Department: Progress Toward the Synthesis of Porphyrins with a β-Azo Linkage to Other π-Conjugated Systems

30. Emily Freeburne, Sutton Stegman, Lisa Timmons  
Department of Molecular Biosciences, University of Kansas: yy6: An Allele Affecting RNA Interference in C. elegans

31. Johnny Fuller†, Ginny Ke†, Catherine Raacke†  
Hays High School: KU Medical School Night @ the Lab†: The Impacts of High Blood Pressure

32. Ian Gambill, Dr. Bridgett Chapin, and Josh Meisel  
Haskell Indian Nations University: Community-based research: Investigation and comparison of dissolved oxygen and water chemistry between Haskell and Baker wetlands

33. Sreenavya Gandikota and Mark A. Schneegurt  
Department of Biological Sciences, Wichita State University: Presence of Halotolerant Bacteria in Oligohaline Environments

34. Christopher Gebhardt†, Lan Lan†, Minli Xing†, Dr. Liang Xu†  
†Department of Molecular Biosciences, †Bio-NMR Core Facility, University of Kansas: Molecular Target Validation of Active Fragments for RNA-Binding Protein Musashi-2

35. Gibson, Mallory, Ashleigh Elbert, and Virginia Rider  
Department of Biology, Pittsburg State University: Spatial distribution and regulation of CCL19 and CCL21 expression in rat uteri in preparation for embryo implantation

36. Robin Goodreau, Kelly Mallatt, and Christine Brodsky  
Department of Biology, Pittsburg State University: Remediation of Tar Creek: Ecological diversity and potential human health impacts.

37. Gu, Xuan‡, Han, Shuang‡, Xu, Liang‡  
‡Department of Molecular Biosciences, University of Kansas: Targeting RNA-Binding Protein HuR Regulates Characteristics of Human Cervical Cancer Cells

38. Ryan Haller and Sam H. Leung  
Department of Chemistry, Washburn University, Topeka, KS 66621: Progress Toward the Synthesis of Expanded Oxophlorins

39. Hanson, Tyler, Aakash Pandey, Thomas G. Platt  
Kansas State University Division of Biology: Virulence evolution of low and high virulence Stenotrophomonas maltophilia strains

40. Katlyn J. Hays, Paul M. Heffren, Shaun E. Schmidt  
Department of Chemistry, Washburn University: Base Hydrolysis of Tosylamide to form Secondary Cyclic Amines Using a Microwave Reactor
41. Paul Heffren, Shaun Schmidt  
Department of Chemistry, Washburn University: Schiff Base Cyclization to Form a Cross Protected Tetraazamacrocycle

42. Abigail K. Hosek, Shaun E. Schmidt  
Department of Chemistry, Washburn University: Alkylation of Nosylamide to form Azamacrocycles

43. Alex Hydock, Dr. Stephanie Shames  
Kansas State University: Investigations of Effector-Mediated Growth Restriction in Legionella pneumophila

44. Vedant Jain[1], Tyler Shelby[1], Elena Mekhedov[2], Joshua Zimmerberg[2], Prasad Dandawate[3], Shrikanth Ananth[4], Ahinsa Ranaweera[4], David Weliky[4], Tuhina Banerjee[1] and Santimukul Santra[1]  

45. MD Joad, Hassan Zbeeb, Mark Schneegurt PhD  
Wichita State University Department of Biological Sciences: Growth of Bacterial Isolates in Iterative Matrix of Salts at High Concentrations Relevant to Mars

46. Caleb B Jones, Jacob D Herford, Bruce D Schultz  
Department of Anatomy and Physiology, Kansas State University, Manhattan, KS 66508: Anti-inflammatory drugs inhibit bradykinin-induced changes in epithelial ion transport

47. Abby Jurgensemeier, Linh Nguyen, MarcArthur Limpiado, Moriah R. Beck  
Chemistry Department, Wichita State University: Using Fluorescent Tags and TIRF Microscopy to Monitor Palladin Interactions with Actin

48. Kanemoto, Sierra, U., Nayan Shrestha, and William J. Hendry  
Department of Biological Sciences, Wichita State University: Continued development/testing of improved methods for immunohistochemistry

49. Benjamin Kelm, Luke Wenger, Everett Hall, Jeremy Goering, Benjamin Parker, Irfan Saadi  
Department of Anatomy and Cell Biology, University of Kansas Medical Center, Kansas City, KS: Investigation of Congenital Hydrocephalus in Specc1l-Deficient Mice

50. Nathan Kerr, Daniel Zurek  
Department of Biology, Pittsburg State University: Cytotoxicity of a Novel Antibiotic Protein

51. Jordyn Koehn and James Walters  
University of Kansas, Department of Ecology and Evolutionary Biology, Lawrence, KS, 66045: Proteo-genomic analysis of butterfly sperm using shotgun mass-spectra data.

52. Kostner, Danica, Megan Dougherty, Yass Kobayashi  
Department of Biological Sciences, Fort Hays State University: The relationship between food exposure frequency and expression of AMP-activated protein kinase (AMPK) mRNA in the brain, liver, and muscle of channel catfish.

53. Kostner, Danica, Megan Dougherty, Abigail Schmidtberger, Oaklee Abernathy, Yass Kobayashi  
Department of Biological Science, Fort Hays State University: Relationship between O-linked N-acetylg glucosamine Transferase (OGT) mRNA Expression and Nutritional Status in the Channel Catfish Muscle.

54. Frank Kutilek, Eric Guo, Dr. Moriah Beck  
Wichita State University Department of Chemistry: Examination of Structure Function Patterns in Non-Conserved Regions of Lactate Dehydrogenase (LDH)

55. Jacob Lutgen[1], Jared Ridder[1], James R. Balthazor[2]  
[1] Fort Hays State University Department of Biological Sciences; [2] Fort Hays State University Department of Chemistry: RNA Interference of X-Box Binding Protein 1 in Acyrthosiphon pisum

56. Skyler Markham, Zachary Araki, Benjamin Wicker, Bruce Atwater  
Fort Hays State University Department of Chemistry; Southeastern Louisiana, Department of Chemistry and Physics: Synthesis of Unsymmetrical 2,2′-bipyridine Derivatives Via a Phosphorus Extrusion

57. Mathias, Betty, George Bousfield, and Bin Shuai  
Department of Biological Sciences, Wichita State University: Amplification and Analysis of Genes for hFSH Glycoform-Specific Antibodies in Hybridoma Cell Lines

58. Elizabeth McCuistion, Mariah Emily, Sam Pimpl, and Melissa M. Bailey  
Emporia State University, Department of Biological Sciences: Cannabidiol Oil Does Not Have Adverse Effects on Fetal Development in ICR Mice

59. Alec McDaniel, Qiyang Zhang  
Emporia State University: Optimization of Custom-built Capillary Electrophoresis Coupled with Laser-induced Fluorescence Detection
60. Reegan Miller and Chad Slawson
Department of Biochemistry and Molecular Biology, University of Kansas Medical Center: The Effects of OGT Knockdowns on Mitochondrial Functions in SY5Y Cells

61. Mary K Mitchell, Stuart J Macdonald
Department of Molecular Biosciences, University of Kansas, Lawrence, KS: The Effects of Sleep Deprivation on Gene Expression in Drosophila melanogaster

62. Tucker Morey 2, Kinsey Morey 2, Khamis S Siam 2, Pawan Kumar Kahol 1, Ram Gupta 2
112 Russ Hall, Pittsburg State Univ, Pittsburg, Kansas, United States; 2 Chemistry, Pittsburg State University, Pittsburg, Kansas, United States: Bioinspired synthesis of molybdenum carbide for hydrogen evolution reaction

63. Sierra Mortimer1, Matthew E. Ochs1, Rebecca McWhirter2, David M. Miller2, and Erik A. Lundquist1
1Department of Molecular Biosciences, University of Kansas; 2Department of Cell and Developmental Biology, Vanderbilt University: The Q neuroblast transcriptome reveals novel transcription factors involved in Q descendant migration.

64. Denise Muchangi, Sneha Ramanujam, Tuhina Banerjee, Santimukul Santra*
Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762: Magneto-Plasmonic nanosensor for the Detection of Ebola Virus

65. Alireza J. Nasrazadani1, Myranda Q. Sofuoa1, Emily L. Torrey1, Hernan A. Hernandez1, Matthew P. Thompson2, Nathan C. Gianneschi2, and Andrea J. Luthi1
1Department of Chemistry, Emporia State University; 2Department of Chemistry, Northwestern University: Peptide-polymer Amphiphiles with Different Properties for Studying Biological Interactions

66. Nieves, N.1, A. Amer1,2, N. Barts1, and M. Tobler1
1Division of Biology, Kansas State University, Manhattan, KS 2Department of Biology, The Pennsylvania State University, PA: Cellular responses to chronic oxidative stress in Poecilia mexicana

67. Palmer, Casey, Frank J. Kutilek, Shreeya Dalla, Moriah R. Beck
Department of Chemistry, Wichita State University: Examining LDH function and stability in extremophile Hypsibius dujardini through protein engineering

68. Peery, Seth T.1, and Kathrin Schrick1,2.
1Department of Biochemistry and Molecular Biophysics, 2Division of Biology, Kansas State University, Manhattan, KS: HD-Zip transcription factors: an evolutionary engine

69. Ana Perez-Lebron and Stephen Fields
Department of Biological Sciences, Emporia State University: Microbiome of a kleptoplastic dinoflagellate

70. Gabrielle Phillips, Mary Roth, Madeline Colter, Pamela Tamura, Kathrin Schrick, Ruth Welti
Division of Biology, Kansas State University: Characterization of the in-vivo functions of putative lysoglycerophospholipid acyltransferase in Arabidopsis thaliana

71. Picard, Hunter1, Angi D. Rathnayake1, Yunjeong Kim2, Chamandi S. Dampalla1, Nhat Nguyen1, Anusha C. Galasiti Kankamalage1, Nurjahan Mehzeb2em; Kevin P. Battaile2, Scott Lovell1, Kyeong-Ok Chang1, and William C. Groutas1
1Department of Chemistry, Wichita State University, Wichita, KS; 2College of Veterinary Medicine, Kansas State University, Manhattan, KS; 3 IMCA-CAT, APS Argonne National Laboratory, Argonne, IL: Protein Structure Laboratory, The University of Kansas, KS: Structure-Guided Design and Optimization of a Novel Series of Dipeptidyl Inhibitors of Norovirus 3CL Protease

72. Adrienne Pohl and Dr. Stephanie Shames
Kansas State University, Department of Biology: Defining the mechanism of eEF1A regulation by the Legionella pneumophila effector Sid1

73. Peter Powell and Takrima Sadikot
Biology Department, Washburn University: Annotation of Transcription Start Sites for Motif Analysis

74. Quispe, Naomi C., Jorja E. Elliott, Yasuhiro Kobayashi, and Brian R. Maricle
Department of Biological Sciences, Fort Hays State University: Effect of sulfide, lactic acid, and ethanol on respiration enzyme activities in catfish tissues

75. Priyanka Radadiya1, Jessica Idowu1, Francis Franco1, Brenda Magenheimer2, Darren P Wallace3, James P Calvet2, Madhulika Sharma1
1Departments of Internal Medicine and Biochemistry and Molecular Biology, The Jared Grantham Kidney Institute, University of Kansas Medical Center, Kansas City, KS: Analyzing the effect of repurposed drugs on the progression of Polycystic Kidney Disease

76. Maluki Radford 2, Elizabeth Thoenen 1, Tomoo Iwakuma 1
1 KUMC Cancer Biology, 2 Washburn University Biology: Mutant p53 sensitizes cancer cells to stress inducers by inhibiting stress granule formation

77. Renwick, Emma D1, Kimberly G. Stanford1, Emily Fey1, John A. Stanford1
1Department of Molecular & Integrative Physiology, University of Kansas Medical Center: Effects of Intrinsic Aerobic Capacity on Resistance Exercise Performance in Middle-Aged Rats
79. **Romero Elmer, Wimalasena Kandatege, Murphy David, Lickteig Bryan**  
Department of Chemistry at Wichita State University: Measurement of potential Parkinsonian Toxin uptake in C. elegans using HPLC and microscopy techniques.

80. **Grant Ryals and Dr. Aj Mellott**  
University of Kansas Medical Center, R3 Lab, K-INBRE: To evaluate whether composite extracellular matrix scaffolds enhance the proliferation, migration, and differentiation of human Wharton jelly cells over single material scaffolds used in wound healing.

81. **Richard Sandefur, Ashley DeBrot, Li Yao**  
Department of Biological Sciences, Wichita State University: The interaction of nanofibers and oligodendrocyte progenitor cells derived from induced pluripotent stem cells.

82. **Sharp, Courtney¹, Mike Blazanin², Spencer Parish², Teresa Shippy¹, Sherry Miller¹, Joe Sevigny³, Kelley Thomas³, Susan Brown¹, and Thomas G. Platt¹**  
¹Division of Biology, Kansas State University; ²Department of Ecology and Evolutionary Biology, Yale University; ³Department of Molecular, Cellular, and Biomedical Science and Hubbard Center for Genome Studies, University of New Hampshire: The evolution of Agrobacterium tumefaciens in opine and plant cue containing environments.

83. **Sheth, Margi, Sarah Shapiro¹, Colin Curwen-MacAdams², Stephen Jones², and Matt Arterburn¹**  
¹Department of Biology, Washburn University; ²The Bread Lab, Washington State University, Mt. Vernon: Microsatellite Analyses of Specific Linkage Groups in Perennial Wheat Hybrid Lines.

84. **Dania Shoaib; Rob Ward**  
University of Kansas Department of Molecular Biosciences: Genetic and cell biological analysis of eight possible alleles of Mcr in Drosophila.

85. **Haley Smalley, Fernando Nieto, Jazmin Zeledon, Emily Archer Stone, and Sherry D. Fleming**  
Division of Biology, Kansas State University: Small Therapeutic Peptides Reduce Angiogenesis and Melanoma Growth.

86. **Emily Smith, Vaishnavi Nagarajan, and Lisa Timmons**  
Department of Molecular Biosciences, University of Kansas: Dimerization Partners of HAF ABC Transporters.

87. **Colby Spiess¹, Vikalp Vishwakarma¹, Ranjan Preet¹ and Dan A. Dixon¹**  
¹Department of Molecular Biology, University of Kansas, Lawrence, KS and The University of Kansas Cancer Center, Kansas City, KS: Overexpression of the RNA Binding Protein HuR Promotes Chemoresistance to mTOR Pathway Inhibitors in Colorectal Cancer Cells.

88. **Aubrie Stricker, Maheka Gujar, Lakshmi Sundararajan, Erik Lundquist**  
University of Kansas Molecular Biosciences: UNC-6/Netrin and its Receptors Regulate Growth Cone Polarity, Microtubule Accumulation, and Protrusion in C. elegans.

89. **Mackenzie Thornton, Micah Meyer, Eric Aube, Sarah Gillaspie, Chingakham Ranjit Singh, Katsura Asano**  
Molecular Cellular and Developmental Biology Program, Division of Biology, Kansas State Program University, Manhattan, KS 66506: The Study on Nucleotide Motifs Enriched in 5' UTR of Genes Controlled by Gcn2.

90. **Tran, Daniel H¹, Anna M. Brokesh¹, Cameron C. Hunter¹, Joel T. Steyer¹, Damien J. Downes¹, Meryl A. Davis² and Richard B. Todd¹**  
¹Department of Plant Pathology, Kansas State University; ²Department of Genetics, The University of Melbourne: The mechanism of nitrogen metabolite repression by the NmrA corepressor.

91. **Minh Tran, Weslin Camden, Mark A. Schneegurt, PhD**  
Wichita State University Biology Department: Bacterial Keratinase Enzyme for the Treatment of Nail and Skin Disease.

92. **Villarreal Acha, Daniel; Bodugam, Mahipal; Jayasinghe, Susanthi; McParland, J. P.; Whitehead, Alan; Ganguly, Arghya; Hanson, Paul R.**  
Department of Chemistry, University of Kansas: Phosphate-Tether Mediated Studies Towards the Synthesis of Leustroducsin B and Its Simplified Analogs.

93. **Vonary, Derek, David Eichhorn, John Kromer, and Alan Oberley**  
Wichita State University Department of Chemistry: Syntheses and Properties of Cyanopyrazole Metal Complexes of Interest in Modeling Enzymatic Active Sites.

94. **Alexander Vontz¹, Anjana Suppahia¹, Pushpa Itagi²,³, Jeroen Roelofs¹**  
¹Molecular, Cellular, and Developmental Biology Program, Division of Biology, Kansas State University, 338 Ackert Hall, Manhattan, Kansas 66506 USA; ²Center for Bioinformatics, University of Kansas, 2030 Becker Drive, Lawrence, KS 66047 USA; ³Department of Molecular Biosciences, University of Kansas, Lawrence, KS 66047 USA: Role of 3-Propeptides in Proteasome Assembly Process.

95. **Ryan Walker, Megan Peters, Tayita Abudu, Mandy M. Peak**  
Department of Biology, Pittsburg State University: Analyzing the Gut Microbiome of Human Populations in Fort Scott, KS and Shoal Creek, MO.

96. **Zoey Wallis¹, Rashmi Acharya¹, Dr. Eric Gillock¹**  
¹Department of Biological Sciences, Fort Hays State University, Hays, Kansas: PCR Assay to detect Porcine Endogenous Retroviruses (PERV) A, B, and C.

97. **Kayden Webb, Andrew Herbig**  
Washburn University Department of Biology: Morphology and Replication of Notiopedio, a Bacteriophage Infecting Bacillus subtilis.
98. Wessel, Emily,1 John Tomich,1 Richard Todd 2
1Department of Biochemistry and Molecular Biophysics, Kansas State University, 2Department of Plant Pathology, Kansas State University: Degradation of BAPCs by fungi

99. Emily White, Washburn University Biology Undergraduate
Takrima Sadikot, Department of Biology Associate Professor: Genomic Annotation of 61,000 bp region of 3L chromosome in D. takahashii

100. C. Williams, A.M.M. Stoian, R.R.R. Rowland
Department of Diagnostic Medicine/Pathobiology, College of Veterinary Medicine; Kansas State University, Manhattan, KS: Porcine reproductive and respiratory syndrome virus (PRRSV-1) recognition of peptide sequences in CD163 SRCR5

101. Thomas Williams, Brintha Parasammanna Girinathan, Babita Adhikari Dhungel, Revathi Govind
Division of Biology, Kansas State University: The role of thiolase in Clostridium difficile biology

102. Wilson, Cole1. Durrett, Timothy1
1Kansas State University Department of Biochemistry and Molecular Biophysics: Subdomain shuffling in two similar acyltransferases to investigate acyl specificity

103. Joseph Zupan and Bridgett Chapin
Haskell Indian Nations University: Creating Research Infrastructure in the Haskell Wetlands: Installing a Permanent Sampling Grid and Long-term Water Monitoring Sites

104. Rashmi Acharya and Eric T. Gillock
Department of Biological Sciences, Fort Hays State University: Distribution of Porcine Endogenous Retrovirus (PERV) variants in Domestic and Feral Pigs

105. Jessica Aldrich1 and David Long1
1Wichita State University, Biomedical Engineering: Effects of Mechanotransduction on Nuclear Morphology and Chromatin Organization

106. Haifa Alhadyian and Robert Ward
Department of Molecular Biosciences, University of Kansas: Macroglobulin complement-related is required for Drosophila egg elongation

107. Momin Ansare, Saloni Darji, Sneha Ramanujam, Tanuja Tummala, Tuhina Banerjee, Santimukul Santra*
1Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762: Combination Therapy of Prostate Cancer: PARP Inhibitor Synergizes the Therapeutic Efficacy of Doxorubicin

108. Molly E. Birrer, T. Chris Gamblin, and Brian D. Ackley
Department of Molecular Biosciences, University of Kansas: Expression of mutant human tau protein drives synaptic loss in Caenorhabditis elegans

Department of Molecular Biosciences, University of Kansas, Lawrence, KS: Constitutive Musashi-1 (Msi-1) expression alters growth and intestinal epithelial cell characteristics in mice

110. Ashley Cloud1, Aditya Vargheese1, Raeann Shimak2, Sumedha Gunawardena1, Roy Jensen2, Vargheese Chennathukuzhi1
1Department of Molecular and Integrative Physiology, University of Kansas Medical Center, Kansas City, KS; 2Department of Pathology and Laboratory Medicine, University of Kansas Medical Center, Kansas City, KS: Loss of REST in Breast Cancer

111. Saloni Darji,1 Tuhina Banerjee,1 and Santimukul Santra1,*
1Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762: Development of Functional Magnetic Relaxation Nanosensors for the Investigation of Zika Binding and Fusion Mechanism

112. Oindrila De1 and Robert E. Ward1
1Department of Molecular Biosciences, University of Kansas, Lawrence, KS: Investigating a role for septate junction proteins in cell polarity during Drosophila dorsal closure

113. Ashley DeBrot, Li Yao
Department of Biology, Fairmount College of Liberal Arts and Sciences: Migration of induced pluripotent stem cells and their derivatives in 3D matrices

114. Ruochen Dong1, Kishore Polireddy2, Xiaqing Wu2, Ping Chen1, Tao Wang1, Dan A Dixon2, Liang Xu2, Qi Chen1
1Department of Pharmacology, Toxicology and Therapeutics, The University of Kansas Medical Center; 2Department of Molecular Biosciences, The University of Kansas: An RNA binding protein, HuR, in pancreatic cancer EMT, metastasis, and CSC

115. Bayan Ahmad Dous,1 Saloni Darji,1 Annette Khaled,2 Shrikant Anant,3 Tuhina Banerjee1 and Santimukul Santra1,*
1Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762; 2School of Medicine, University of Central Florida, Orlando, FL 32826; 3Department of Cancer Biology, KUMC: Combination Therapy of TNBC: The Hsp90 Inhibitor Synergizes the Therapeutic Efficacy of CT20p Peptide

Molecular Biosciences, University of Kansas, Lawrence, KS: Ulcerative Colitis Patients Display Increased Levels of APC in Goblet Cells
117. Stefan Graw1, Rosalyn Henn2, Jeffrey A. Thompson1, Devin C. Koestler1,7
1Department of Biostatistics, University of Kansas Medical Center, Kansas City, KS; 2Department of Cancer Biology, University of Kansas Medical Center, Kansas City, KS; *To whom correspondence should be addressed: pwrEWAS: A user-friendly tool for comprehensive power estimation for epigenome wide association studies (EWAS)

118. Everett G. Hall,1 Nathan R. Wilson,1 Luke Wenger,1 Jeremy Goering,1 Youssef Kousa,2 Diana S. Acevedo,1 Lenore Pitstick,3 Martine Dunnwald,4 Bryan C. Bjork,5 Brian C. Schutte,2 Irfan Saadi1
1Dept. of Anatomy and Cell Biology, University of Kansas Medical Center, Kansas City, KS; 2Dept. of Microbiology and Molecular Genetics, Michigan State University, East Lansing, MI; 3Dept. of Biochemistry, Midwestern University, Downers Grove, IL; 4Dept. Anatomy and Cell Biology, University of Iowa, Iowa City, IA: SPECC1L regulates oral epithelial adhesion downstream of IRF6 in palatogenesis

119. Mikala Heon, Daniel Aks, Mei He
Department of Chemical and Petroleum Engineering, Bioengineering program, University of Kansas, Lawrence, Kansas, USA: In-vivo like Fibroblastic Reticular Cell Growth Determined by 3D Scaffold Material

120. Berenice Jiménez-Marín1, Katherine Johnson1, Bradley JSC Olson1
1Department of Biology, Kansas State University, USA (Jimenezb@ksu.edu): The Extracellular Matrix of Gonium is Important for Multicellularity

121. Ashley Joseph1, Brian Geisbrecht2, and Stephanie Shames1
1Division of Biology, Kansas State University; 2Department of Biochemistry and Molecular Biophysics, Kansas State University: Identification and Characterization of chemical inhibitors of the Legionella pneumophila metaeffector Lpg2505

122. Meagan E Kurland, Brian D Ackley
University of Kansas - Lawrence, Molecular Biosciences: Neuron “GPS”: The Canonical Wnt Pathway in Motor Axon Guidance

123. Naveen Maddukuri and Maojun Gong
Department of Chemistry, Wichita State University, Wichita, Kansas 67260: Online Preconcentration of High-Salt Samples Using Pressure-Assisted Field-Amplified Sample Injection in Flow-Gated Capillary Electrophoresis

124. Richard Meier1, Dominique Michaud2 and Devin Koestler1
1Department of Biostatistics, University of Kansas Medical Center, Kansas City, KS; 2Department of Public Health & Community Medicine, Tufts University School of Medicine, Boston, MA: A Bayesian Framework for Assessing Concordance in Microbial Abundance

125. Abigail Morgan, Phillip Harries
Pittsburg State University: Optimization of a lead biosensor to test environmental samples in an in vitro and in vivo system

126. Hansel, Sara1; Stricker, Michael2; Balthazor, James3; and Carvalho, Claudia4.
Fort Hays State University, Department of Biological Sciences1,3, Department of Chemistry2,4: Natural Products Against E.S.K.A.P.E. Pathogens Isolated from Soil microbes

127. Lisa Neums, Jeffrey A. Thompson
Department of Biostatistics, University of Kansas Medical Center: Detection of inversely enriched pathways in PBMC cells in Alzheimer’s disease and cancer

128. Elena Olson, Andrew Micciche, Steven Ricke and Anuradha Ghosh
Department of Biology Pittsburgh State University: Understanding the characteristics of bacterial isolates obtained from commercial poultry feed using whole genome sequencing approach

129. Taybor W. Parker, Kristi L. Neufeld
Department of Molecular Biosciences, University of Kansas, Lawrence, KS: Wnt-dependent asymmetric redistribution of the β-catenin-destruction complex

130. Picking, Taffra and Brian Maricle
Department of Biological Sciences, Fort Hays State University: Antimicrobial properties of secondary metabolites from plants in Kansas wetlands

Fort Hays State University Department of Chemistry; Fort Hays State University Department of Biological Sciences: RNA Interference of the Unfolded Protein Response in Acyrthosiphon pisum

132. Duncan Rotich, Jeffrey Thompson, PhD1
1Department of Biostatistics, University of Kansas Medical Center: Integrating Multiplatform Data for Improved Prognosis in Cancer Patients.

133. Aaron J. Rudeen1, Minli Xing2, Justin T. Douglas2, and Kristi L. Neufeld1
1Department of Molecular Biosciences, University of Kansas, Lawrence, KS; 2Nuclear Magnetic Resonance Core Lab, University of Kansas, Lawrence, KS: Biomolecular Structure-Function studies of the central binding region of tumor suppressor Adenomatous Polyposis Coli

134. Tao Wang1, Ruochen Dong1, Ping Chen1, Michael J Baltezor2, Scott Weir1, Qi Chen1.
1Department of Pharmacology, Toxicology and Therapeutics, University of Kansas Medical Center, Kansas City, KS, USA; 2Biotechnology Innovation and Optimization Center, University of Kansas, Lawrence, USA: A novel compound inhibits pancreatic cancer cell invasion, tumor sphere formation and in vivo tumor growth in mice by suppressing EMT
135. Camila Zequine1, Fangzhou Wang2, Xianglin Li2, Pawan K. Kahol3, Ram K. Gupta1
1Department of Chemistry, Pittsburg State University, Pittsburg, KS 66762, USA; 2Department of Mechanical Engineering, University of Kansas, Lawrence, KS 66046, USA; 3Department of Physics, Pittsburg State University, Pittsburg, KS 66762, USA: CuCo$_2$S$_4$ Nanoballs: A solution to clean hydrogen generation and urea elimination from wastewater

136. Xin Zhang1, Long Wu1 and Liqin Zhao1, 2
1Department of Pharmacology and Toxicology, School of Pharmacy, 2Neuroscience Graduate Program, University of Kansas, Lawrence, KS 66045, USA: ApoE2 promotes neuronal health and wellbeing via modulation of glycolytic pathways

137. Yomna Badawi1, Kazuhiro Shigemoto, 2 and Hiroshi Nishimune1
1Department of Anatomy and Cell Biology, University of Kansas School of Medicine, Kansas City, 66160, 2Department of Geriatric Medicine, Tokyo Metropolitan Institute of Gerontology, Itabashi, Japan: Super-resolution microscopy analysis of neuromuscular junction reveals degeneration of active zones in ALS model mice

138. Suman Chaudhary, Deepak Kumar, Nanyan Lu, Michael Duff, Mathew Heffel, Teresa Ortega-Villicana, Douglas Marthaler Kansas State Veterinary Diagnostic Lab, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66502, US: Next Generation Sequencing Pipeline for Metagenomic Data Analysis of Clinical Samples

139. Yujun Chen1, George Aranjuez2, Ashley Burtscher1, Ketki Sawant1, Xiaobo Wang1, Damien Ramei1, Jocelyn A. McDonald1
1) Division of Biology, Kansas State University, Manhattan, KS; 2) Lerner Research Institute, Cleveland Clinic, Cleveland, OH; 3) LBCMCP, Centre de Biologie Intégrative (CBI), Université de Toulouse, CNRS, UPS, France: Protein Phosphatase 1 promotes a collective rather than single cell mode of migration

140. Elizabeth R. Everman, Kristen M. Cloud-Richardson, Stuart J. Macdonald University of Kansas: Genomic dissection of natural variation resistance to copper poisoning in Drosophila melanogaster

141. Gulhumay Gardashova1, Xiaqing Wu1, Liang Xu1
1Department of Molecular Biosciences, University of Kansas, Lawrence, KS, USA: The Role of RNA Binding Protein, HuR, in Pancreatic and Breast Cancer Malignancies

142. Jeremy Goering1, 5, Dona Greta Isa1,5, Everett Hall1, Nathan Wilson1, Edina Kosa1, Luke Wenger1, Zaid Umar1, Abdul Yousaf1, Andras Czirok1, Irfan Saadi1
1Department of Anatomy and Cell Biology, University of Kansas Medical Center, Kansas City, KS; 5These authors contributed equally.: SPECC1L-Deficient Cells Show Impaired Collective Cell Migration Attributes that are Rescued by Upregulation of PI3K-AKT Pathway

143. Ryan Grigsby1 and Susan M. Lunte1,2,4,3
1The Center for Molecular Analysis of Disease Pathways, 2The Ralph N. Adams Institute for Bioanalytical Chemistry, 3Department of Pharmaceutical Chemistry, 4Department of Chemistry, University of Kansas: Equipment and Services of the Ralph N. Adams COBRE Core Nanofabrication Facility

144. Jennifer L. Hackett1,2,3,4, Britny R. Smith1,2,3,4, Erik A. Lundquist1,2,4, Susan M. Lunte1,5,6
1Center for Molecular Analysis of Disease Pathways, 2Genome Sequencing Core, 3Higuchi Biosciences Center, 4Department of Molecular Biosciences, 5Department of Pharmaceutical Chemistry, University of Kansas, Lawrence, KS, USA: Next Generation Sequencing at KU Genome Sequencing Core

145. Haley Haines, Yan Hong, Hanan S. Elsarraj, Ruoman Zhao, Aria Sabbagh, Chad Slawson, Antonio Artigues, Jeffery Thompson, Fariba Behbod. University of Kansas Medical Center, Kansas City, KS; University of Kansas School of Medicine-Wichita, Wichita, KS: BCL9 protein-protein interactions that drive breast cancer progression

146. Tom Hill, Robert L. Unckless University of Kansas: Antiviral evolution and population history of Drosophila innubila

147. Shengping Huàng2, Prachee Avasthi1,2
1Anatomy and Cell Biology, University of Kansas Medical Center, Kansas City, KS; 2Ophthalmology, University of Kansas Medical Center, Kansas City, KS: Regulation of heterotrimeric kinesin-II motor complex trafficking by RanGTP and canonical nuclear import/export signals

148. Lan Lan1, Minli Xing2, Justin T. Douglas2, Amber Smith1, Philip Gao3, Amber D. Somoza4, Clay C. C. Wang5,6, Beri Oakley1, Roberto De Guzman1, Robert P. Hanzlik6, and Liang Xu1, 7
1Departments of Molecular Biosciences, 2Bio-NMR Core Facility, 3Protein Production Group, NIH COBRE in Protein Structure and Function, The University of Kansas, Lawrence, Kansas; 4Department of Chemistry, 5Department of Pharmacology and Pharmaceutical Sciences, School of Pharmacy, University of Southern California, Los Angeles, CA; 6Department of Medicinal Chemistry, The University of Kansas, Lawrence, Kansas; 7Department of Radiation Oncology, The University of Kansas Cancer Center, Kansas City, Kansas: Dissecting the structural basis for inhibitors of RNA-binding proteins

149. Patrick Lansdon, Mariaelena Naboros, Robert L. Unckless, and Brian D. Ackley, Department of Molecular Biosciences, University of Kansas, Lawrence, KS: An Evolutionary Approach to Understanding Host Interactions with Microbial Pathogen

150. Minwoo Joseph Lee, Lee892@student.usd250.org, James Mcafee and Irene Zegar, izegar@pittstate.edu Pittsburg High School, Pittsburg, Kansas & Pittsburg State University, Pittsburg, Kansas: Using Molecular Docking to Find Potential Anti-Cancer Drugs that Bind a Mutant form of Spekle-Type POZ, SPOP, a Protein Necessary for the Maintenance of a Number of Cancer Cells

151. Takashi Matsuda1, Yomna Badawi,1 Sudheer Tungtur,1 Rupal Soder,1 Jim Mitchell,2 Buddhadeb Dawn,2 Richard Barohn,3 and Hiroshi Nishimune1
1Department of Anatomy and Cell Biology, 2Midwest Stem Cell Therapy Center, 3Department of Neurology, University of Kansas Medical Center, Kansas City, KS, USA: Applying Human Mesenchymal Stem Cells for the Treatment of Amyotrophic Lateral Sclerosis
152. Devin McAfee, James McAfee, and Irene Zegar, izegar@pittstate.edu
Pittsburg High School, Pittsburg, Kansas & Pittsburg State University, Pittsburg, Kansas: Fighting Cancer By Targeting Cancer Stem Cells (CSCs) Using Virtual Docking Techniques

153. Aaron Meers, Sara Veesart, Irene Zegar, and James McAfee
Department of Chemistry, Pittsburg State University, Pittsburg, Kansas: Analysis of Leucine Zipper Mediated Interactions in the HNRNP C Family of Proteins

154. Thiya Mukherjee1, Ruben-Lerma Reyes1, Kyle Thompson1, and Kathrin Schrick1
1Division of Biology, Kansas State University, Manhattan, KS 66506: Homeodomain proteins linking lipid metabolism to gene expression in plants

155. Tshogfotso Ngwaga, Alex J. Hydock and Stephanie R. Shames
Division of Biology, Kansas State University, Manhattan, KS; tngwaga@ksu.edu: Overexpression of the Legionella pneumophila Effector LegC4 Attenuates Intracellular Bacterial Replication

156. Kazushi Okada1, Robert S. Rogers2, Sumedha Gunewardena2, Jeffrey H. Miner3, and Hiroshi Nishimune1
1Department of Anatomy and Cell Biology, University of Kansas School of Medicine, 2Department of Molecular & Integrative Physiology, University of Kansas School of Medicine, 3Division of Nephrology, Department of Medicine, Washington University School of Medicine: Elucidation of molecular mechanisms causing aging-related synapse degeneration using RNA-seq transcriptome data

157. Dong Pei1, Ina Zaimi2, Devin C. Koester1, Carmen J. Marsit3, Immaculata De Vivo4, Shelley S. Tworoger5,6, Alexandra E. Shields7, Karl T. Kelsey7,8,9 and Dominique S. Michaud2,8
1Department of Biostatistics and University of Kansas Cancer Center, The University of Kansas Medical Center; 2Department of Public Health & Community Medicine, Tufts University School of Medicine, Tufts University, Boston; 3Department of Environmental Health and Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta; 4Channing Division of Network Medicine, Department of Medicine, Brigham and Women’s Hospital and Harvard Medical School, Boston; 5Department of Cancer Epidemiology, Moffitt Cancer Center, Tampa; 6Department of Epidemiology, 7Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston; 8Department of Epidemiology, 9Department of Pathology and Laboratory Medicine, Brown University, Providence: Variation in DNA methylation of human blood over a 1-year period using the Illumina MethylationEPIC array

158. Chamani T. Perera1, Blake R. Peterson1,2, Thomas E. Prisinzano1,2
1Department of Medicinal Chemistry, University of Kansas, Lawrence, KS, USA; 2School of Pharmacy, University of Kansas, Lawrence, KS, USA: The Synthetic Chemical Biology Core (SCB): A Resource for Research in Chemical Biology

159. Ranjan Preet1, Vikalp Vishwakarma1, Wei-Ting Hung2, Lane K. Christenson2, and Dan A. Dixon1
1Departments of Molecular Biology, University of Kansas, Lawrence, KS 66045 and 2Molecular and Integrative Physiology, University of Kansas Medical Center, Kansas City, KS 66160: The RNA Binding Protein HuR Regulates Exosome Secretion in Colorectal Cancer via Rab 27B

160. Jessica B. Rakijas and Bradley JSC Olson
Division of Biology, Kansas State University, Manhattan, KS: Mechanism of Genome Instability Driven by RB Loss

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1Department of Molecular Biosciences, University of Kansas, Lawrence 66045; 2School of Medicine, University of Kansas Medical Center, Kansas City, KS 66160; 3University of Kansas Cancer Center, Cancer Prevention & Survivorship Program, University of Kansas Medical Center, Kansas City, KS 66160: Role of RNA Binding Protein HuR in Non-alcoholic Fatty Liver Disease (NAFLD) Progression

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   1Department of Anatomy & Physiology, Kansas State University College of Veterinary Medicine, Manhattan, KS 66506, USA: **Headcase regulates tissue growth and cell cycle progression in response to nutrient restriction**

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   1Dept. of Pathology & Laboratory Medicine; 2Dept. of Anatomy & Cell Biology; and 3Department of Molecular & Integrative Physiology, University of Kansas Medical Center, Kansas City, KS 66160: **ESR2-regulated ovarian kisspeptins in oocyte maturation**

173. **M.T. Alsmady and E.T. Gillock**
   Department of Biological Sciences. Fort Hays State University: **Prion Gene Polymorphisms in Feral Pigs**
1. Synthesis of Phosphatidylglycerol Receptor: Precursor Preparation

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With the emergence of an alarming amount of multidrug resistant bacteria, there has been a growing interest in antimicrobial peptides as potential antibiotics to combat this issue in the medical community. Antimicrobial peptides target the bacterial plasma membrane organisms by first binding to phosphatidylglycerol (PG) via Coulombic interaction, followed by insertion into the membrane and killing the bacterial cell. However, antimicrobial peptides can be toxic, difficult and expensive to make, and in general exhibit low bioavailability. Our focus will be directed toward the development of small molecules that specifically bind to (PG), the major anionic phospholipid found in bacterial membranes. In doing so, the membrane is disrupted. So far, precursors of a family of cyclophanes whose structure is privileged are being developed. Previously prepared small molecules that bind to the PG head groups have displayed high bacteriostatic properties at low concentrations (1-4 μM). Moreover, this privileged structure is expected to similarly bind to PG that will cause the antimicrobial effect due to its commonality in binding pocket. The antimicrobial effect makes plasma membrane more permeable which depolarizes the membrane with the aim to stop replication. The precursors developed so far consist of the synthesis of bis-phenol from bis-anisole from demethylation and the synthesis of allylic mesylate from the transformation of pentane-1,5-diol into 5-(methoxymethoxy)pentyl-4-methylbenzene sulfonate.

2. Comparative analysis of Stereolithography (SL) based 3D Printing Technique Versus Digital Light Processing (DLP) 3D Printing.

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Background: 3D printing is an additive manufacturing (AM) technique that converts CAD design to a solid object. Likewise, bio-printing is also an AM technique where cells and biomaterials are deposited simultaneously in the prints. The aim of the study is to compare the effect of different process parameters i.e. number of layers, time of exposure, and curing time between UV laser 3D printer and 3D digital light printer on printed specimen properties. Such comparison would give an insight on the effectiveness of DLP printer as compared to the traditional UV printer.

Methods: ISO (527-2 5A) samples were printed using both manufacturing techniques at different layer thickness, 0.1, 0.05, and 0.025 mm. Swelling measurements were recorded. Then, FTIR spectra was obtained from 64 scans at 4cm⁻¹ resolution to determine the conversion effectiveness of C=C after UV light exposure by the two printers.

Results: Specimens did not show any swelling indicating optimal curing. A MATLAB code was created to normalize the experimental absorbance peaks at an internal standard 1608 cm⁻¹. The peaks at 1635 cm⁻¹ were compared and the reduction in the peaks absorbance at 1635 cm⁻¹ indicated curing in both techniques. The results indicated increased curing as the thickness of the layers decreased.

Discussion: Failure of the samples to swell indicates optimal curing of the specimens. In addition, the decrease in the 1635 cm⁻¹ C=C peak indicated higher curing of the resin at lower layer thickness.

3. Characterization of Microbial Population in the Nasal Cavity of Pigs during Post-Weaning in Response to Probiotic Supplementation

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Probiotics are commercially available for human consumption. Although it is a common dietary supplement, it has not been approved by the FDA. Probiotics are advertised to affect microbial population in the gastrointestinal system and improve immunity. Additionally, probiotic supplements have been used as an alternate grow and health promoter in domestic animals, such as pigs. However, the definitive effects of probiotic supplementation on these parameters are unknown. The objective of this study was to characterize the microbial population profile of the nasal cavity in post-weaning pigs in response to probiotic supplementation. Nasal swipes were collected from 5 pigs that were fed with commercial feed containing mannan-oligosaccharides plus β-glucans and essential oils for 43 days. Additional samples were collected from 5 pigs that were fed with the identical commercial feed with essential oils alone for the same duration. DNA was isolated from each nasal swipe and V3/V4 region of 16s rDNA was amplified using PCR. Each PCR product was indexed. The indexed PCR products were sequenced to characterize the microbial population profile of each pig. DNA was successfully isolated and amplified for each sample. An analysis of the microbial population profile of each sample is being completed as well as an examination of whether the probiotic supplementation affects the microbial population profile in the nasal cavities of the pigs. In the future, effects of maternal probiotic supplementation on microbial population of piglets during weaning period and their effects on post-weaning immune and stress responses will be examined.

4. An RNAi-based Screen to Identify Novel Molecules in the Nidogen Synaptic Pathway in Caenorhabditis elegans

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Nidogen is a conserved basement membrane protein required for the proper formation and organization of neuromuscular junctions. In C. elegans, nidogen functions to efficiently localize the presynaptic scaffold protein SYD-2/a-ligrin in synaptic active zones. The synaptic phenotype in nidogen mutants is suppressed by loss-of-function in genes that encode a calcium channel (unc-2 or unc-36), the calmodulin kinase (unc-43), or calmyrin (calm-1), demonstrating that calcium signaling is important in synaptic morphogenesis. Previous work identified proteins that associate with CALM-1 in a calcium-dependent fashion, including RACK-1 and multiple ribosomal proteins. Loss of function in rack-1 results in a synaptic phenotype equivalent to the loss of nidogen, and this phenotype can be suppressed by the loss of calm-1. Interestingly, RACK-1 is known to be a translational inhibitor in many contexts, although whether this is how it is functioning in synaptic nidogen pathway is unclear. To test this, we are using RNAi to inactivate the ribosomal proteins isolated in our biochemical screen. RNA interference provides a fast and reliable approach to investigate complex genetic interactions that might be impossible to study using existing mutations. The findings of this study will identify whether all the CALM-1 associated proteins are involved in the nidogen synaptic pathway. This information will be important in better understanding synaptogenesis in invertebrates and vertebrates.

Key Words: Synaptogenesis, Nidogen, Calmyrin, RNAi, C. elegans
5. Identification and Characterization of Messenger RNA Encoding the Prion Protein in Channel Catfish

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The disease-causing nature of prion proteins (PrP) from misfolding is well known in humans and cattle. In cultured human cancer cells, PrP appeared to have a role in modulating inflammatory response. In developing zebrafish embryos, PrP is shown to influence cell-cell adhesion. However, the exact physiological function of PrP has not been well-defined. The objectives of our study were to identify PrP genes in channel catfish, characterize tissue distribution of its mRNA, and to examine whether tissue damage was caused by the injection of Streptozocin (STZ) in channel catfish. Screening of the channel catfish genome database, yielded two distinct transcripts encoding PrP with high sequence similarity. However, the nucleotide sequence of channel catfish PrP did not share high sequence similarities with PrP from other fish or that of mammals. Using real-time polymerase chain reaction, expression of PrP mRNA was examined in the brain, liver, muscle, kidney, heart, spleen, and intestine of channel catfish. The PrP mRNA was highly detectable in the brain and liver of channel catfish. Expression of PrP mRNA tend to be higher in the brain of channel catfish treated with 3.6 mg/kg STZ compared to fish treated with vehicle. Expression of PrP mRNA was similar between fish treated with vehicle and those treated with higher doses of STZ. Currently we are examining the effects of STZ treatment on the hepatic expression of PrP mRNA. In the future, the relationship between the development of inflammation and expression of PrP mRNA will be examined.


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Extracellular vesicles (EVs) are nanoparticles ranging between 50-100 nm in diameter. They are released by almost all cells and can travel to neighboring or distant cells. They contain a cargo of genetic material, proteins, and lipids obtained from their parent cell. Extracellular vesicles have the ability to transfer this cargo to recipient cells, either in the vicinity of the parent cell or to cells in the distance. By doing so, they can influence cell function of the recipient cells. This unique property makes them important mediators of cell-to-cell communication. Extracellular vesicles are highly enriched in tetraspanins, a protein superfamily that organize membrane microdomains termed tetraspanin-enriched microdomains. Tetraspanins, including CD9 and CD81, are known markers for EVs and are used to validate exosomal preparations. Using antibodies CD9 and CD81, we performed Western blot analyses to confirm the presence of these proteins in our EV preparations. In addition, we performed electron microscopy analysis of our exosomal preparation to confirm the size of exosomes.

7. Characterization of the interaction between the Legionella pneumophila effectors Lpg2505 and SidI

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Legionella pneumophila is an intracellular bacterial pathogen that causes legionellosis and Legionnaires’ disease, an inflammatory pneumonia. These bacteria proliferate within phagocytic eukaryotes and are naturally found in freshwater environments. Many disease cases involve contaminated water sources. My project involves an analysis of the relationship between SidI and Lpg2505; two L. pneumophila virulence factors called effector proteins that show an interesting relationship and can affect L. pneumophila intracellular replication. Genes encoding both proteins are found on an operon, and are expressed together in wild-type bacteria. In mutants lacking the lpg2505 gene and expressing the sidI gene, the bacteria do not replicate within host cells. Additionally, SidI is toxic to eukaryotic cells and Lpg2505 can alleviate this toxicity. We discovered that recombinant Lpg2505 and SidI interact with each directly in vitro and aim to characterize this protein-protein interaction. Lpg2505 immobilized on beads could retain wild-type SidI and two non-toxic mutants of SidI. Initially, SidI was truncated To narrow down the binding region, multiple truncations of both SidI and Lpg2505 will be used in our binding assay. Once binding sites between SidI and Lpg2505 are identified, we can determine if direct binding between these proteins is required for Lpg2505 to promote bacterial intracellular replication in the presence of SidI.

8. Annotation of gene features in Drosophila takahashii using a computational-genomics approach

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The genome of Drosophila melanogaster has been a very useful reference for studying many developmental and cellular processes common to higher eukaryotes. The goal of this project is to identify and annotate genetic features in the related Drosophila species Drosophila takahashii using comparative genomics; to ultimately better understand the Drosophila genus. In this study, we analyze contig28, a part of the euchromatin region of the D element in D. takahashii by comparing it to D. melanogaster. Based on gene predictions, this contig is expected to contain 5 genes. We will carefully screen the DNA sequence in this contig, identify specific gene markers and compare our models to their specific D. melanogaster orthologs. Sequence analysis and data collection will be carried out using several open-source computational genomic tools for sequence alignment, gene-prediction and the Drosophila genome browser. The data files and resources for this project were made available through the Genomics Education Partnership (GEP) based in Washington University, Saint Louis. Upon completion of the project, a detailed report will be submitted for incorporation into the GEP data repository.
9. Transcriptional Regulation & Genetic Screening Using dCas9 in Saccharomyces cerevisiae

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While CRISPR may have started as a revolutionary gene editing technique, it has since proven to be incredibly versatile, with several applications beyond cleaving the genome. CRISPR is an immune response that evolved in bacteria to defend against invading phages. The system requires a nuclease (Cas9) and a “guide” RNA that Cas9 uses to target, bind, and cleave a specific DNA sequence. The nuclease deficient mutant of Cas9 (“dead Cas9” or “dCas9”) has the ability to target and bind DNA without inducing a double stranded break. This creates an opportunity to fuse other proteins to dCas9 to be transported to the target locus. In this study, we explore the application of dCas9 as a transcriptional regulator in order to produce an easily detectable phenotypic output. To achieve this, we designed fusions of dCas9 with the repressor domain Mxi1 or transcriptional activator VPR. We developed several artificial reporter constructs containing SpHIS5 (required for growth on medium lacking histidine) or ADE2, (when deleted, yeast colonies turn the color red). By targeting dCas9 fusions to promoter elements, we were able to induce growth or color changes in yeast colonies. This technique will be applied to screen through Cas9 mutants that may escape the action of the anti-CRISPR proteins AcrlA2/A4. These small peptides inhibit the function of Cas9 (or dCas9). Future work will mutagenize dCas9 to isolate functional variants with new biochemical properties that could be used in CRISPR editing applications including gene drives.

10. A novel PD-L1 inhibitory peptide enhances CD8+ T cell-dependent cell death against lung cancer cells

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Programmed death ligand-1 (PD-L1) and PD-L2 are natural ligands for PD-1 and are expressed abundantly in cancer cells. Binding of PD-1 by PD-L1 is linked to cytotoxic T lymphocyte (CTL) exhaustion, and upregulation of surface PD-L1 on cancer cells is associated with immune evasion and aggressive tumor growth. Anti-PD-1/PD-L1 antibodies have been utilized for the blockade of this checkpoint. However, long course treatment with anti-PD-1/PD-L1 antibodies is associated with immune-related adverse events (irAE). We propose a novel PD-1/PD-L1 blockade via an inhibitory peptide that can be administered at the cancer site, limiting adverse systemic effects of immune checkpoint inhibition. We designed two PD-L1 inhibitory peptides (PD-L1ips) that interact with the binding site of PD-L1, and investigated the effect of the PD-L1ips on functional upregulation of both human and murine CTL. IFNγ treatment was shown to stimulate PD-L1 expression in H1299 human lung carcinoma cells. Upon stimulation with anti-CD3/CD28 antibody bead cocktail, human T cells (Jurkat cells) demonstrated upregulation of pro-inflammatory cytokines, including IFNγ, Granzyme B, and IL-2, confirming T cell activation. In co-culture with H1299 cells and anti-CD3/CD28 antibody-activated human T cells, a small stimulation in PD-L1-dependent H1299 cytotoxicity was detected after 48 hours of co-culture for both PD-L1ip-1 and -2. However, PD-L1ip-1, but not PD-L1ip-2, increased CTL-induced cell death of H1299 cells when H1299 antigen-specific T cells were co-cultured. Results suggest that newly designed PD-L1ip-1 is effective in promoting CTL-induced cell death in lung cancer cells. This study was supported by NIH grants 1 R15 CA21991901 and P20 GM103418.

11. Studying the role of T_{reg} cells in mucosal tolerance using the DEREG transgenic mouse model

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From birth, the microbial community within the small intestine forms a symbiotic relationship with the host, mediated by the immune response. An important immune cell in this relationship is the regulatory T (T_{reg}) cell. To study this relationship, we acquired transgenic mice (DEREG) that allow for temporary depletion of T_{reg} cells using diphtheria toxin (DT). The intent of our initial studies with this model is to evaluate methods for measuring T_{reg} cell depletion. We determined the time course of T_{reg} cell arrival in the small intestine after birth, validate genotyping protocols, and to titer DT quantities that effectively decreasing T_{reg} cell levels. To examine methods for measuring T_{reg} cell depletion, spleen samples from transgenic and wildtype mice were processed to measure GFP expression by both qRT-PCR and for flow cytometry. Both methods resulted in significant and measurable GFP expression in transgenic animals. To determine the T_{reg} cell presence in the small intestine over time, expression of FoxP3 was determined using qRT-PCR. FoxP3 expression was measurable as early as three days after birth and significantly increased during weeks two to four. Genotyping studies confirmed the validity of our multiplex qPCR protocols. Lastly, DT effectiveness and toxicity were determined by injecting one day old mice with varying concentrations of DT. Observational data and weight gain was used to measure toxicity, and RT-qPCR was used to measure the extent of T_{reg} cell depletion in each sample. Collectively the results of these experiments confirms the utility of the DEREG mouse model for investigating the role of T_{reg} cells in perinatal small intestine.


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Abstract:
ADP-forming acetyl-CoA synthetase (ACD) is an enzyme that participates in fermentation of acetyl-CoA to acetate. Catalysis conserves energy from the hydrolysis of the thioester bond of acetyl-CoA to generate ATP from ADP via substrate level phosphorylation. ACD activity is most common in archaea and acetate-producing bacteria, however it is also present in a select group of protozoan parasites, such as Entamoeba histolytica (dysentery) and Giardia lamblia (giardiasis). As amitochondriates, both of these parasites lack oxidative phosphorylation; consequently, they must rely on glycolysis and amino acid degradation as key pathways for ATP generation. Biochemical characterization of ACD suggests it may play a role in extending the glycolytic pathway to provide additional ATP for the cell and regenerate CoA.
13. Effect of luteolin on AKT/GSK3-?beta

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Around 1 in 8 deaths United States women may develop breast cancer in their lifetime, of which approximately twenty percent are triple negative breast cancer (TNBC). TNBC lacks the estrogen, progesterone, and HER2/neu receptors which are current chemotherapeutic targets, and thus there are less treatment options for TNBC patients. Novel approaches to combat TNBC are needed to expand our chemotherapeutic repertoire. Luteolin is a naturally occurring tricyclic aromatic compound that has been shown to alleviate metastasis of breast cancer, possible through disruption of epithelial to mesenchymal transition (EMT). Recent findings show that luteolin reduces mRNA and protein levels of a known regulator of EMT, ?-catenin, in TNBC. Typically, regulation of ?-catenin is performed through phosphorylation of ?-catenin by glycogen synthase kinase 3-? (GSK3?), thus targeting it for ubiquitination. The normally constitutively active GSK3? enzyme is inhibited by protein kinase B (AKT) phosphorylation, which luteolin has been shown to reduce AKT activity in breast cancer cells.

We hypothesize that luteolin increases GSK3? activity through AKT inhibition, and thus disrupts EMT in TNBCs. We know that increased ?-catenin drives EMT, and through these experiments we will develop an improved understanding between how LU might suppress EMT. Preliminary results from mRNA isolations suggest that LU may affect GSK3? mRNA levels. In the future, the combination of the mRNA results and western blot analysis will help further elucidate the involvement of LU on GSK3? activity.

14. Ecology and Prevalence of Ticks and Tick-Borne Bacterial Pathogens in Southeast Kansas

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Ticks transmit a wide variety of pathogens including viruses, bacteria, protozoa, and helminthes to vertebrates. Their life cycle depends on blood meals from various hosts as well as on environmental conditions such as the temperature and habitat type. The goals of the present study were to assess the prevalence of various tick species and tick-borne bacterial pathogens in southeast Kansas and adjacent area. Ticks were collected during May-August in 2016 and 2017 from three types of tick habitats using the flag-drag method. Adults and nymphs were sexed and identified using taxonomic key and PCR. Selected bacterial species were also detected by PCR. Differences between tick species prevalence in woodland versus pasture land cover types were analyzed using Arc-GIS. Out of a total of 1678 ticks collected, the majority of ticks were identified as Dermacentor (50.3%) and Amblyomma (47.3%); very few (2.4%) ixodes females and nymphs were also identified. For all the species, more females were found than males. While A. americanum were more frequently found in pasture (42.6%), Q. variabilis and I. scapularis were found in woodland (68.6%) and (80.9%), respectively. The rate of detection in Amblyomma (total pooled isolation, n = 39) for Francisella tularensis was 2.56%, for Rickettsia rickettsii was 28.2% while the same in Dermacentor (n = 28) for F. tularensis was 3.57%, for R. rickettsii was 7.14%. The data obtained in this study would help in implementing comprehensive surveillance and management programs for ticks and tick-borne disease risk for humans and animals in this region.

15. The Oncogene cMYC Suppresses non-AUG Translation

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cMYC is the major oncogene encoding the transcription factor driving oncogenic transcription. To study cMYC function in promoting cell proliferation, we generated the derivatives of the retinal pigment epithelium 1 (RPE1), a model differentiated cell line, expressing the cMYC through initiation from various non-AUG codons and that of Renilla luciferase is initiated from an AUG codon. Our dual luciferase reporter assay indicated the cMYC expression can do so in the RP E1. For this purpose, we used dual luciferase report assays, in which translation of firefly luciferase is initiated from various non-AUG codons, while that of Renilla luciferase is initiated from an AUG codon. Our dual luciferase reporter assay indicated that the expression of cMYC represses non-AUG translation regardless of the type of non-AUG codon (such as CUG, GUG or ACG) or the context driving non-AUG translation (such as Kozak consensus). We discuss how the repression of non-AUG translation might provide the benefit for cancer cells to survive and metastasize.

16. Clostridioides difficile SinR' regulates toxin, sporulation and motility through protein-protein interaction with SinR

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Clostridioides difficile is a gram-positive, anaerobic bacterium. It is known that C. difficile is one of the major causes of antibiotic associated diarrhea. The enhanced antibiotic resistance observed in C. difficile is the result of highly resistant spores produced by the bacterium. In Bacillus subtilis, the sin operon is involved in sporulation inhibition. Two proteins coded within this operon, SinR and SinI, have an antagonistic relationship; SinR acts as an inhibitor to sporulation whereas SinI represses the activity of SinR, thus allowing the bacterium to sporulate. In a previous study, we examined the sin locus in C. difficile and named the two genes associated with this operon sinR and sinI', analogous to sinR and sinl in B. subtilis, respectively. We have shown that SinR and SinR' have pleiotropic roles in pathogenesis pathways and interact antagonistically with each other. Unlike B. subtilis Sinl, SinR' in C. difficile carries two domains, the HTH and Multimerization Domain (MD). In this study, we first performed a GST Pull-down experiment to determine the SinR' interacting domain within SinR. Second, the effect of these two domains on three phenotypes; sporulation, motility, and toxin production was examined. The findings of this study confirmed that as predicted, Multimerization Domain (MD) of SinR' is responsible for the interaction between SinR and SinR'. It was also discovered that SinR' regulates sporulation, toxin production and motility primarily by inhibiting SinR activity through the Multimerization Domain (MD).
17. Preliminary Studies on More Affordable Effective Means of Homogenization and Quantification of Tetrahydrocannabinol (THC) in Marijuana Case Samples

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Since the legalization of marijuana for both recreational and medicinal use has become more prominent throughout the United States in recent years, there is growing concern regarding the actual amounts of cannabinoids in the wide variety of plants that are bred for such purposes. Most especially concerning is the concentration of tetrahydrocannabinol, or THC, the active ingredient found in the sticky resin produced by the marijuana plant, which varies over a wide range (Upton et al., 2014) depending upon the type of plant. Since some states attempt to set a threshold above which marijuana is more strictly controlled, a need arises for an effective homogenization and sample preparation method for the determination of purity and quantity of THC at low analysis costs. There are a wide variety of published methods, but few studies have evaluated the homogenization process itself, which is difficult to achieve with the sticky resin. In this preliminary study, homogenization with and without liquid nitrogen was evaluated, since cryogenic freezing is used in several published studies. A variety of marijuana case samples were dried for 20 hours in an oven (35-40°C), then homogenized using a mortar and pestle both with and without liquid nitrogen. The samples were extracted with ethanol and the THC quantity was determined using Liquid Chromatography-Mass Spectrometry (LC-MS) and Gas Chromatography-Mass Spectrometry (GC-MS). Preliminary results suggest that the liquid nitrogen does not improve the homogenization process.

18. Bioinformatics of Major Depressive Disorder

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Major depressive disorder is a mental health disorder characterized by persistently depressed mood or chemical imbalance in the brain, causing significant impairment in daily life. Major depressive disorder is one of the most treatable psychiatric illnesses but is still the leading disability among 15 to 44-year-olds. Major depressive disorder occurs in approximately 16.1 million Americans and is present in about 50% of people that commit suicide. But if this disease is so treatable then why these staggering statistics? It is because the brain is the great unknown. And with major depressive disorder being a major mental disorder it makes the quest for treatment and scientific understanding that much harder. The goal of this research is figure out what role, if any, does the gene SLC6A4 play in major depressive disorder. The second objective was to see if comparing the crystal structure of a drug target, fluoxetine, to a patient’s amino acid sequence could benefit people who suffer from this disorder. The role of SLC6A4 was investigated by the use of bioinformatics. Bioinformatics being the science of collecting and analyzing complex biological data such a genetic code. The procedures include but were not limited to supercomputing, multiple sequence alignments, phylogenetic tree generation, and BLAST. After research was conducted results showed that the SLC6A4 gene does play a significant role in major depressive disorder by being responsible for reuptake of serotonin. Low expressions of the SLC6A4 gene was also linked to lower levels of serotonin. Results also showed that personalized medicine could be vital to better treatment of major depressive disorder patients.

19. Heavy metals threatening aquatic ecosystems and human health in sediments in the Tri-State Mining District

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Tar Creek is the primary drainage stream for the Tri-State Mining District (Oklahoma, Kansas, and Missouri) which was one of the world’s largest producers of lead and zinc for over 100 years. Elevated toxic metal concentrations associated with the mining process have severe impacts the region’s environmental and public health. Mine pollution at Tar Creek has deteriorated the quality and diversity of aquatic life. Toxic metals accumulate in the fish and may be transferred to humans upon consumption. This study analyzed heavy metal concentrations, such as zinc and lead, in sediments at Tar Creek. Four sediment cores in a transect were collected across Tar Creek in Miami, Oklahoma and analyzed using Inductively Coupled Plasma (ICP). In 1998, the U.S. Environmental Protection Agency (EPA) implemented sediment-quality guidelines to support the functioning of healthy aquatic ecosystems for a nearby stream draining the mining district, Spring River. The two EPA recommended guidelines are the Threshold Effects Level (TEL), which represents occasional adverse effects and the Probable Effects Level (PEL), which indicates frequent toxic effects. Concentrations of lead, zinc, arsenic, and cadmium exceeded the TEL at Tar Creek. This study showed that, cadmium, lead, and zinc concentrations were greater than the PEL, with concentrations as high as 106, 1610, and 9142 mg/kg, respectively. These results were used to guide the selection of a second round of extractions for the samples obtained nearest to the stream. These additional samples will be incorporated in our ongoing analysis.

20. Developing a controlled behavioral assay to test bilateral cortical plasticity in intact rats

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Large scale developmental changes that occur in complex cortical networks after unilateral brain injury may be the basis for recovery. Many changes in the ipsilesional hemisphere have been described after cortical injury in regions responsible for motor control, but the role of the intact hemisphere is still debated. There is some evidence that in these types of injuries, increased activity in the intact hemisphere causes inhibition of the injured hemisphere, impairing function. However, there is also evidence that activity in the intact hemisphere, particularly during the acute post-injury period, may be neuroprotective. To directly interpret the role of population activity from both hemispheres during recovery, neural recordings obtained during a skilled behavior in a scalable preclinical model such as rodent may be the most appropriate experimental platform. Previous studies have demonstrated the utility of a pellet retrieval task in rats as an assay of developmental progress following ischemic infarct. We propose training rats on a modified version of the retrieval task which requires the alternating use of each forelimb to circumvent this problem through the switched role of each hemisphere as the task is mirrored. Trained rats will then be implanted bilaterally in motor areas of neocortex with microelectrode arrays in order to acquire neurophysiological spiking activity during the bilateral reaching behavior. The proposed studies will characterize the modified behavior and neural data acquired from intact rats performing the task.
21. Investigating the importance of proline-rich motifs in palladin for VASP binding

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Actin is a central player in cell motility and shape. Eukaryotic cells use over 150 different proteins to regulate the assembly, crosslinking, and turnover of actin filaments. Palladin is a recently discovered protein associated with actin and other actin-related proteins. Located at the amino terminus is a proline-rich motif, which serves as putative binding site for Vasodilator-Stimulated Phosphoprotein (VASP). Both VASP and palladin are known to catalyze actin polymerization; however, there is no data that establishes a direct interaction between the two proteins. Recently the Beck and Guttman labs collaborated to show that interactions between VASP and palladin are critical for cell motility in Listeria, therefore this research will use biochemical techniques to investigate the direct binding of VASP to palladin. Furthermore, we will use site-directed mutagenesis of the polyproline region to compare binding by wild type (FPDP) and mutated palladin (FPAA). The binding interaction is tested using a pull-down assay with concentrated proteins, followed first by far-western blotting and/or western blotting to monitor the presence of both proteins. Surprisingly, we found that VASP continued to bind both WT and FPAA mutant palladin. However, further testing is required to ensure consistent amounts of the proteins. Establishing a direct connection between these two proteins, as well as defining the site of interaction, is critical to understanding the mechanism employed by highly motile cells such as metastatic cancer cells that are known to overexpress palladin.

22. NMR Solution Structure of Ecdysis Triggering Hormone Peptide from the African Malaria Mosquito Anopheles gambiae

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Infections by mosquito-borne diseases represent one of the leading causes of death in third world countries. The rapid progression of resistance to conventional insecticide causes a significant threat to the highly efficient preventive methods currently in place. Deletion of the Ecdysis Triggering Hormone (ETH) encoding gene has been proven to result in fatal deficits in insects. We propose to develop insecticidal compounds that disrupt the ecdysis triggering hormone (ETH) system that is a crucial component of larvae development specific to the insect species. The ETH of the African malaria mosquito (AgETH), a small peptide hormone with 17 amino acid residues (SESPGFFIKLSKSVPRIamide), was studied to elucidate its molecular structure. We have determined the solution structure of AgETH using 2D H-H Nuclear Magnetic Resonance (NMR) spectroscopy and Nuclear Overhauser Effect (NOE) derived constraints. Our findings show that AgETH contains a short alpha helix between residues 3S and 11S. The NMR solution structure of AgETH will be of significant assistance for designing a new class of insecticidal compounds that acts on the AgETH receptor aiming for in-silico docking studies.

23. Motorless myosin V transcripts expressed in mouse brain

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Abstract
The actin associated myosin V motor proteins take part in many biological processes in eukaryotic cells. We previously identified an alternatively spliced, motorless myosin V transcript in Caenorhabditis elegans that interacts with an RNA-binding protein. The widespread expression of this motorless transcript in the C. elegans nervous system led us to look for motorless myosin V transcripts in other organisms. We initially performed multiple sequence alignments of homologous introns of the three mammalian myosin V genes, MYO5A, MYOSB and MYOSC, from five different mammals, including the common house mouse Mus musculus. Several highly conserved sequence and transcription factor-binding sites were identified within one intron of MYO5A. We then isolated mRNA from mouse brain and used primers within and downstream from this conserved region to perform RNA Ligase Mediated Rapid Amplification of cDNA Ends (RLM-RACE) to identify the 5′ end of the transcript. The sequence of the products from this amplification showed that a portion of the intron upstream of exon 20 is expressed but does not appear to code for a peptide. Instead, the expressed intronic region may act as a 5′ UTR, with a start codon occurring near the beginning of exon 20. The resulting protein would lack a motor domain but still have the potential of forming homodimers. This is similar to the structure of the truncated myosin V in C. elegans.

24. Restoring Magnesium to the world’s food source through soil nutrient additions

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Lack of nutrition is a problem that faces numerous people across the world. To add to this problem many are unaware that they are being deprived in their diet of fundamental sources of bodily nourishment. One of the most important macro minerals that much of the world’s population is lacking is Magnesium. Research has shown the importance that Magnesium has on several key functions within the body and more importantly, the issues caused by the absence of sufficient amounts. Past and current research shows that Magnesium is critical in performing hundreds of activities within the body. Humans receive only a percentage of the minimum amount of Magnesium needed on a daily basis from their diets. It may seem unusual that a mineral so important is so scarce in our food sources when in actuality, it is one of the most abundant elements on Earth. This decline is due to the overuse of agricultural soil throughout the last two centuries depleting many of the nutrients within the soil and as a result, in our food. We aim to determine what results traditional and Magnesium fertilizers have on a few common food crops. This experiment will focus on nutrient content of edible grain in plants exposed to magnesium fertilizer, traditional fertilizer (nitrogen, phosphorus, and potassium), and both fertilizers in combination. By increasing the Magnesium content in common crop species, we hope to help counteract the magnesium deficiency in the diet of much of the world’s population.
25. Analysis of Non-AUG Translation Initiation using Molecular Dynamics Simulations

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Given the superiority of computer processing power, memory, speed, and accuracy compared to that of humans, computer science is paving the way to a deeper level of research that has yet to be explored. Specifically, this study uses molecular dynamics simulations to reconstruct and analyze the interaction between mRNA and the ribosome during translation initiation. It is well established that non-AUG start codons are capable of initiating translation, but the role of regulatory proteins (specifically eIF1) in this process isn’t well understood. By compiling and programming a molecular dynamic simulation that reconstructs the translational environment, we are able to observe stability parameters for alternate start codons such as CUG and GUG in comparison with AUG. Furthermore, we can run the simulation in the presence or absence of eIF1 to understand its effect on non-AUG codons, and explain the role of system stability in translation initiation.

26. Effects of sulfide, ethanol, and lactic acid on cytochrome c oxidase and citrate synthase activities in plant roots

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Sulfide, ethanol, and lactic acid are well known as metabolic toxins, yet specific effects on respiration enzymes have not been characterized. The objective of this study was to analyze sulfide, ethanol, and lactic acid toxicity on activities of the enzymes cytochrome c oxidase (CytOx), active in oxidative phosphorylation and citrate synthase (CS), active in the citric acid cycle in mitochondria. Enzyme activity was measured in tissue homogenates from roots of several plant species in the presence of sulfide (0 to 20 µM), ethanol (0 to 100 mM), and lactic acid (0 to 100 mM). Increasing concentrations of sulfide and lactic acid significantly decreased activity in CytOx, however sulfide toxicity had a greater effect on the enzyme compared to lactic acid toxicity. Sulfide has a potent metabolic toxic effect on CytOx, while ethanol had a lesser effect on CytOx and CS activity. Ethanol and lactic acid toxicity had a lesser effect on the enzyme compared to lactic acid toxicity. Sulfide was a potent metabolic toxin; activity of CytOx was measured as high as 0.106 μmol/min/g in the absence of but was reduced to nearly undetectable at 10 µM sulfide. CytOx activity was also reduced by lactic acid but the inhibition constant (K) was 104 times that of sulfide toxicity. Increasing lactic acid concentrations caused a threshold response, where CytOx was not affected until 100 mM lactic acid. Ethanol had no effect on CytOx activity. CS activity was not affected by lactic acid and only marginally affected by ethanol. Both sulfide and lactic acid influenced activities of CytOx, but to different degrees, indicating environmental and physiological constraints on plant metabolism, particularly with respect to sulfide exposure and its effects on respiration.

27. Synthesis of Doxorubicin-Based Prodrug and Activatable MR Nanoprobe For the Imaging and Treatment of Cancer

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Magnetic Resonance Imaging (MRI) is increasingly being used as a diagnostic tool for cancer. We propose a novel molecular probe, Gadolinium-DTPA disulfide-bonded Doxorubicin (Gd-DTPA-SS-Doxo) encapsulated in iron oxide, which could provide a dual modality for detecting malignant growth while simultaneously targeting treatment options. We first synthesized Gd-DTPA-SS-Doxo followed by encapsulation within the poly (acrylic acid) (PAA) coating of iron oxide nanoparticle (IONP), producing a nanoprobe (IO-Gd-DTPA-SS-Doxo) with quenched longitudinal spin lattice magnetic relaxation (T1). After receptor-mediated internalization, Gd-DTPA-SS-Doxo is released from the nanoprobe’s polymeric coating due to the acidic microenvironment of the tumor. When the molecular probe is subjected to various enzymes present in the cancerous cells, the disulfide bond of the molecular probe is cleaved. This results in an intracellular release of Gd-DTPA complex and Doxo with subsequent T1 activation and magnetic relaxation (T1). After receptor-mediated internalization, Gd-DTPA-SS-Doxo is released from the nanoprobe’s polymeric coating due to the acidic microenvironment of the tumor. When the molecular probe is subjected to various enzymes present in the cancerous cells, the disulfide bond of the molecular probe is cleaved. This results in an intracellular release of Gd-DTPA complex and Doxo with subsequent T1 activation and magnetic relaxation (T1). The current study explored the gene expression of Cyp2b10 during a window of susceptibility. In our previous studies, fetuses whose mothers were exposed to CP and restrained showed significant reductions in the expression of Cyp2b10, as compared to saline or restraint-only control animals. Animals exposed to both CP and restraint had a 4-fold increase in the expression of Cyp2b10, as compared to saline or restraint-only control animals. Animals exposed to both CP and restraint had expression levels slightly lower than either control group means. Due to intra-treatment group variability, no statistically significant difference was observed (P > 0.05); however, these results agree with previous studies. The current results suggest that one possible mechanism for the reduction of defects seen in our previous studies is a reduction in the biotransformation of CP to its teratogenic metabolites by Cyp2b10.

28. Changes in Cyp2b10 Gene Expression During Cyclophosphamide Exposure and Maternal Restraint Stress

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Physical restraint can be used to induce consistent psychological stress during gestation and has been shown to cause developmental anomalies. Cyclophosphamide (CP) is an anticancer agent and model proteratogen that causes limb, digit, and cranial defects if prenatal exposure occurs during a window of susceptibility. In our previous studies, fetuses whose mothers were exposed to CP and restrained showed significant reductions in specific CP-induced morphological defects. The current study explored the gene expression of Cyp2b10 (mouse ortholog to human CYP2B6), one of the Phase I enzymes responsible for CP activation. Mated dams were assigned to one of 4 treatment groups: control (saline via IP injection), control + restraint, CP only (20 mg/kg, IP), or CP + restraint. On GD 17, the dams were sacrificed, and maternal livers were collected and immediately frozen at -80°C. Gene expression of Cyp2b10 was determined using quantitative RT-PCR. Dams exposed to CP only had an almost 4-fold increase in the expression of Cyp2b10, as compared to saline or restraint-only control animals. Animals exposed to both CP and restraint had expression levels slightly lower than either control group means. Due to intra-treatment group variability, no statistically significant difference was observed (P > 0.05); however, these results agree with previous studies. The current results suggest that one possible mechanism for the reduction of defects seen in our previous studies is a reduction in the biotransformation of CP to its teratogenic metabolites by Cyp2b10.

29. Progress Toward the Synthesis of Porphyrins with a β-Azo Linkage to Other π-Conjugated Systems

Hannah Fairchild and Sam Leung
Washburn University Chemistry Department

Photodynamic therapy (PDT) is a procedure performed to combat cancer. This therapy involves using light to activate photosensitizers, which excite oxygen to its excited state. This excited oxygen proceeds to eliminate the cancer nearby. There are several photosensitizers that have been developed, but additional research is being done to synthesize photosensitizers that can absorb visible light at longer wavelengths (>650 nm). This research focuses on attempting to synthesize a porphyrin with a β-azo linkage to a π-conjugated system. This type of porphyrins is expected to absorb visible light at longer wavelengths. Currently we are working on developing the methodology to synthesize the key precursor (a dipyromethane with an azo linkage to a substituted benzene ring) to the target porphyrin.
30. The Impacts of High Blood Pressure

Johnny Fuller\textsuperscript{1}, Ginny Ke\textsuperscript{1}, Catherine Raacke\textsuperscript{1}

Hays High School: KU Medical School Night @ the Lab\textsuperscript{1}

This project investigated “The Impacts of High Blood Pressure.” The Hays High School team investigated the effects of hypertension on bodily functions including the human heart/cardiovascular system, blood flow, blood pressure, hypertension, elastic arteries, muscular arteries, dementia, heart attack, heart failures, strokes, aneurysms, kidney failure, osteoporosis, and peripheral artery diseases. Also investigated were the results of medications, diets, and new technology on high blood pressure (HBP) treatment. High blood pressure, commonly known as the silent killer, is diagnosed if the systolic pressure reads above 130 mm Hg or the diastolic pressures is measured at over 80 mm Hg consistently. HBP mainly damages the tunica intima in the elastic and muscular arteries. This damage, which includes cholesterol plaque buildup, will lead to blood clotting in the arteries and veins throughout the body. Blood clotting can lead to the main impact areas plus atherosclerosis. When maintaining HBP, subjects will most likely be given medicine and diets, however, subjects can also invest in new and upcoming technology that can help manage HBP. These new adaptations include Heart Guide by Orman and the Kardia band by Apple both of which have on the spot blood pressure readings.

31. Community-based research: Investigation and comparison of dissolved oxygen and water chemistry between Haskell and Baker wetlands

Ian Gambill, Dr. Bridgett Chapin, and Josh Meisel

Haskell Indian Nations University

Wetlands play vital roles in an area’s ecology. Many wetlands have been destroyed or are in danger of becoming destroyed due to agricultural use and urbanization factors. Also, there is limited systematic research data for our local wetland system. The Haskell Wetland is a culturally significant wetland complex that provides an outdoor classroom and research opportunities for students and faculty at Haskell Indian Nations University. It is a palustrine, emergent wetland system just south of Lawrence, Kansas which has been impacted by historical disturbance and recent large-scale disturbance including the construction of 31st St and South Lawrence Trafficway. We used a water quality meter and statistical analyses on several water quality parameters to compare the Haskell Wetlands with the Baker Wetlands. Using dissolved oxygen (DO) as a water quality indicator, and the previously contiguous Baker Wetlands as a reference site that could represent least disturbed conditions, data collection and statistical analysis displayed supportive evidence that Baker Wetlands showed higher concentrations of DO than Haskell Wetlands ($T$-Value $= -5.36$, $P < 0.05$). Additionally, a Pearson correlation showed that there is statistical significance in a relationship between distance from 31st St and concentration of DO ($P<0.05$). Regression analysis displayed a linear positive relationship showing that the farther north away from 31st St. into the Haskell Wetlands, the higher the DO concentration. The evidence suggests a potential increasing disturbance gradient from south to north related to urbanization factors.

32. Presence of Halotolerant Bacteria in Oligohaline Environments

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Abstract:

We are investigating the prevalence of culturable halotolerant aerobic heterotrophic bacteria in various inland oligohaline soils from Wichita, KS. It is unclear whether halotolerant bacteria will be present. Soil samples were utilized as inocula in liquid SP media supplemented with 10% and 20% NaCl. If any microbes grow in these salinity concentrations, they could be derived from genera that are associated with hypersaline environments, genera associated with common soils, or novel phyla. We are performing most probable number counts (MPNs) to statistically determine the abundance of bacteria exhibiting tolerance to 10 and 20% salinity. MPNs also will be performed on soil samples and wipes from the Jet Propulsion Laboratory of NASA. These samples will be used as inocula in liquid SP media supplemented with NaCl, Na Chlorate, and Na Perchlorate. Bacterial abundance in 10% NaCl and 20% NaCl media was approximately $10^5$ and $10^6$, respectively, with abundances of $10^5$ and $10^6$ in 1% and 5% Na chloride and $10^4$ and $10^5$ in 1% and 5% Na Perchlorate, respectively. Additional soils will be tested to include a larger variety of oligohaline environments. The ecological role of halotolerance in common soils is not clear and the halotolerant microbes may be present as dormant cells that then proliferate under lab conditions. Finding halotolerant microbes in oligohaline soils is relevant to the contamination of spacecraft in assembly clean rooms. Microbes delivered on spacecraft may be able to grow in the harsh chemical conditions of Mars. This study is supported by NIH KINBRE.
34. Molecular Target Validation of Active Fragments for RNA-Binding Protein Musashi-2

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RNA binding proteins are essential regulators of mRNA translation and stability. The RNA binding protein Musashi-2, is known to be upregulated in many cancers, including leukemia, glioblastomas, colorectal, lung, and pancreatic cancers. The upregulation of MS2 allows cancer cells to acquire a more aggressive cancer phenotype and induce drug resistance by interceding mRNA stability and the translation of target proteins in oncogenic pathways. Several fragments have been screened to assess the activity of binding to MS2; disrupting MS2-RNA binding can lead to the translation of specific genes that are critical for inhibiting cancer cell growth and proliferation. Currently, validation of these active fragments is being determined to see 1. whether these active fragments bind properly to MS2 and 2. what biological consequences occur from this binding. Our hypothesis states that these active fragments who disrupt MS2-RNA binding will block MS2 function, leading to the translation of target genes that are essential for the reduction of cancer cell survival.

35. Spatial distribution and regulation of CCL19 and CCL21 expression in rat uteri in preparation for embryo implantation

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Activation of the T cell homing receptor, CCR7, regulates multiple aspects of adaptive immunity. Deletion of CCR7 reduced T regulatory cell (Treg) migration into the uterus and decreased embryo implantation. We hypothesized that the CCR7 ligands, CCL19 and CCL21, attract Treg cells into the uterus and provide local immune suppression prior to implantation. Sprague-Dawley rat uteri were isolated from pregnant rats (Days 3-6, implantation Day 5) and RNA was isolated. Spatial distribution of the ligands was assessed using immunocytochemistry and ligand expression quantified by real time PCR. At Day 3 of pregnancy, CCL21 expression was limited to the glandular epithelium. Expression appeared in the antimesometrial uterine stroma at Day 4. Expression of CCL21 peaked at Day 4 of pregnancy (Mann Whitney, p <0.05). The distribution of CCL21 was similar between Days 4 and 5 but was less robust at Day 5. CCL21 expression was lost from the glands at Day 6 but maintained in the antimesometrial stroma. CCL19 was expressed in the luminal and glandular epithelium of ovarioctomized rat uteri. Progesterone pretreatment (2 mg daily, 3 days) stimulated expression of CCL19 in the periluminal stroma. Estradiol (0.2 µg) administration to progesterone-pretreated rats increased the expression but not the distribution of CCL19. CCL19 and CCL21 were hormonally controlled in a rat uterine stromal cell line. Unlike the constitutive expression of these ligands in lymphoid tissue, female sex steroids regulate uterine CCL19 and CCL21. An intrinsic program of immune-mediated events appears to operate in the mammalian uterus prior to implantation.

36. Remediation of Tar Creek: Ecological diversity and potential human health impacts.

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A variety of human health and biodiversity risks are often the result of habitat alterations in human-dominated ecosystems, particularly due to greenspace removal and increased pollutant concentrations. In this study, we asked how habitat remediation of a heavy-metal contaminated area impacts biological diversity and nearby residents. We surveyed 21 locations in 2017, plus 3 new sites in 2018, at the Tar Creek Superfund site in various stages of remediation. We sampled each location’s vegetation and bird communities in May through July of 2017 and 2018 and we analyzed community data through Bray-Curtis ordination plots, and a series of regressions and ANOVA. In 2017, we observed 58 bird species and 21 tree species across the mined area. In 2018, we observed 62 bird species and 9 tree species. The remediated locations were composed of grasses and forbs, in contrast to the un-remediated locations which were dominated by mining waste and trees. Bird species richness varied significantly across the remediation gradient with the greatest species richness in the post-remediation sites. A similar trend was observed with species abundance, with the most birds found in the remediated locations. Community composition differed across remediation level, often following life history traits and habitat associations. Regarding human health, the effects of mining-caused elevated lead exposure to humans and wildlife are well documented. A survey is being developed to assess how the residents of Tar Creek and the Quapaw tribe view the change in nature as a result of the remediation. Our goal is to combine the data from the Tar Creek sites and from the survey to determine if these changes are beneficial to the public and to wildlife.

37. Targeting RNA-Binding Protein HuR Regulates Characteristics of Human Cervical Cancer Cells

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Cancer-related gene expression is intrinsically and efficiently mediated by post-transcriptional regulation at RNA level. Human antigen R (HuR), a ubiquitously expressed RNA-binding protein (RBP) encoded by embryonic lethal abnormal vision-like RNA Binding Protein 1 (ELAVL1) gene, has become a crucial post-transcription regulator that targets mRNAs that encode proteins involved in key cellular processes, and has been found to be overexpressed in many types of cancers. To explore the function of HuR in cervical cancer, we generated isogenic cervical cancer cell line, HeLa, with doxycycline-dependent inducible HuR knockdown, and HeLa HuRB3 is one of the clones we selected. As HuR was knocked down by doxycycline, expression of Id1 (Inhibitor of differentiation/DNA binding) is a member of the helix-loop-helix protein family expressed in actively proliferating cells. It was also found to be overexpressed in over 20 types of cancers. While HuR regulation of target mRNAs is based on the interaction between the three specific domains of HuR protein and one or several U- or AU-rich elements (AREs) in the target mRNAs, Id1 mRNA contains several putative HuR ARE sites. Our goal is to examine the impact of HuR on Id1 expression. Using CRISPR knockout system, we further knocked out HuR gene in multiple cell lines, including cervical cancer cell line HeLa, glioblastoma cell line GBM3 and U118. Monoclones were isolated. Our main purpose is to prove that silencing HuR decreased cancer proliferation and invasion both in vitro and in vivo.
38. Progress Toward the Synthesis of Expanded Oxophlorins

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Expanded oxophlorins are substances that could absorb within the visible spectrum of light. These compounds can be useful as photosensitzers for cancer treatment in photodynamic therapy (PDT). One way to synthesize expanded oxophlorins is to synthesize a dipyrroyl-\(\alpha,\beta\)-unsaturated ketone. This can then be combined with a dipyrromethane in a MacDonald-type synthesis. Since there is often a \(\alpha,\beta\)-unsaturated ketone, the reaction scale is too small, the MacDonald-type synthesis could not be attempted. Further studies must be performed by increasing the reaction scale.

39. Virulence evolution of low and high virulence Stenotrophomonas maltophilia strains

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Virulence is the degree to which pathogens harm hosts. Theory predicts and experiments demonstrate a general trend for pathogens to evolve towards a state of intermediate virulence in order to balance the benefits of high virulence with the costs of killing the host. However, these studies focus primarily on the evolution of obligate pathogens, and omit key life history features of facultative pathogens. We plan to use interactions between Caenorhabditis elegans hosts and various strains of Stenotrophomonas maltophilia to investigate how the ability of these pathogens to live independent of hosts influences the evolution of their virulence. To do this we will use an evolution experiment in which treatments vary in the degree of selective dynamics occurring in environmental reservoir environments as oppose to in host environments. Additionally, lines will be initiated with high, low, and intermediate virulence S. maltophilia strains. In this experiment, only the pathogen will be able to evolve as all hosts will be naïve. We will quantify the virulence, shedding rate, and bacterial load of parental strains and derived genotypes arising in our experimental evolution lines. We expect to observe virulence trend toward similar intermediate virulence levels across all strains. Our parameter estimates and observations will be used to further develop evolutionary epidemiology models describing and predicting the virulence evolution of facultative pathogens.

40. Base Hydrolysis of Tosylamide to form Secondary Cyclic Amines Using a Microwave Reactor

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Present research is focused on the synthesis and characterization of polycyclic cage systems containing amine functionalities such as adamanzene. Similar macrocyclic complexes have been used as both imaging agents and in radiotherapy. These macrocycles serve as a “host” to carry the metal ion “guest” through the body. Successful generation of these cage structures has been accomplished by alkylation of an amine followed by ring closing metathesis (RCM) to form macrocyclic structures. Deprotection needs to occur due to the presence of tosyl groups on the amines found in the macrocycle. This has proven to be difficult, however, with previous research often resulting in complete destruction of the compound. Partial detosylation of a tetraazamacrocyle has been achieved via the use of microwave assisted base-hydrolysis using isomayl alcohol and sodium metal. This partial detosylation could be due to the presence of a double bond present in the structure that is inducing steric strain and hindering complete detosylation. The purpose of this research was to attempt to remove the remaining two tosyl groups from the tetraazamacrocyle by removing the tosyl groups without the aid of hydrogenation. Differing concentrations of isomayl alcohol to sodium metal were utilized to accomplish this. However, partial detosylation was observed again, as well as other structure rearrangements. Utilizing a hydrogenation reaction might be necessary, or reforming the macrocycle structure using another protecting group other than tosyl.

41. Schiff Base Cyclization to Form a Cross Protected Tetraazamacrocyle

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Macrocyclic coordination complexes have been used in radiotherapy and medical imaging for many years. [4\(^{th}\)]Adamanzene is one such chelator which may have several advantages over current macrocyclic ligands. This is due to the total encapsulation of the metal ion it is expected to provide, protecting the body from exposure to potentially toxic metals. One of the major chemical intermediates in the synthesis pathway for the production of [4\(^{th}\)]adamanzene is a cross-protected tetraazamacrocyle, however this intermediate has proven difficult to synthesize. Synthesis of one such cross-protected tetraazamacrocyle was achieved using a Schiff base condensation reaction between a linear dihydrosine and diamin. The cross-protected tetraazamacrocyle was also allylated and ring closing metathesis was performed as proof of concept. Finally, novel reaction schemes were also investigated in an endeavor to produce a one-pot synthesis strategy.

42. Alkylation of Nosylamide to form Azamacrocycles

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Present research is focused on the synthesis of polycyclic cage systems that contain amine functionalities, such as [4\(^{th}\)]adamanzene. Macrocyclic complexes have been used as imaging agents and in radiotherapy. The macrocycles serve as a “host” that carries a metal ion through the body. Previous research done with this macrocycle included generation of the cage structure by alkylation of an amine followed by ring closing metathesis to form macrocycles. Deprotection of amines in the macrocycle is necessary, however proved difficult when removing p-toluensulfonamide (tosylamide) protecting groups. The purpose of this research is to form macrocycles by alkylation of 4-nitrobenzenesulfonamide (nosylamide) followed by ring closing metathesis and deprotection of the amines. Utilizing nosylamide, instead of tosylamide, may allow for full deprotection of the amines in the macrocyclic structures.
43. **Investigations of Effector-Mediated Growth Restriction in Legionella pneumophila**

Alex Hydock, Dr. Stephanie Shames, Kansas State University

*Legionella pneumophila* (Lpn) is an opportunistic pathogen that naturally infects in freshwater amoeba. This causes severe pneumonia in immunocompromised individuals called Legionnaires Disease. *Lpn* translocates over 300 effector proteins into host cells using a Dot/Icm type IVB secretion system. Effector protein function has been demonstrated to contribute to mammalian host defense against *Lpn*. Mutation of the *Lpn* effector gene *legC4* results in a fitness advantage in the mouse lung and cytokine-activated macrophages. Alternatively, a strain of *Lpn* overexpressing the *legC4* gene from a plasmid (*plegC4*) is attenuated for replication within mammalian cells. This is due to enhanced production of pro-inflammatory cytokines that contribute to host defense against *Lpn*. We hypothesized that *LegC4* increases the activity of the NF-kB transcription factor, which is required for the expression of multiple cytokines. Using immunofluorescence microscopy, we found that bone-marrow derived macrophages (BMDMs) infected with *Lpn* (*plegC4*) had significantly more NF-kB p65 in the nucleus compared to control *Lpn* strains.

44. **Magnetic Resonance Nanosensor for the Investigation of Influenza Binding and Fusion Mechanisms**

Vedant Jain [1], Tyler Shelby [1], Elena Mekhedov [2], Joshua Zimmerberg [2], Prasad Dandawate [2], Shrikanth Ananth [3], Ahinsa Ranaweea [4], David Welký [5], Tuhina Banerjee [i] and Santimukul Santra [1+i]

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**Abstract:** Fusion between membrane of an enveloped virus and that of the host membrane is one of the very important steps for pathogenesis of the virus the host. This step is critical and is usually protein mediated. For influenza virus, the trimeric hemagglutinin (HA) glycoprotein mediated the membrane fusion of influenza with the host cell. This glycoprotein has two functional peptides HA1 and HA2. HA1 is responsible for helping the virus dock and bind on the sialic acid molecules on the host cell membrane, whereas HA2 participates in membrane fusion by undergoing a conformational change at pH lower than 5.5. Here we present a magnetic relaxation (T2) based technique that uses Liposome Coated Iron Oxide Nanoparticles (LIONs) to better understand this mechanism. Liposomes have recently emerged as an important tool to understand various aspects of biological system and especially to mimic the lipid bilayer of host cell membrane.

In our study, the HA trimeric protein was incubated with LIONs in pH environments ranging from 7.5-5.1 and the T2 changes accompanying the fusion were recorded. The effect of different environmental factors such as trypsin (1%), cholesterol composition of Lipid membrane would be evaluated. Ultimately we would demonstrate our newly formulated LION could be reliably used as a new tool for the rapid screening of fusion inhibitors.

45. **Growth of Bacterial Isolates in Iterative Matrix of Salts at High Concentrations Relevant to Mars**

MD Joad, Hassan Zbeeb, Mark Schneegurt PhD

Wichita State University Department of Biological Sciences

We have been testing bacterial isolates collected from the Great Salt Plains in Oklahoma and Hot Lake in WA. We have chosen 18 salinotolerant bacterial isolates for testing. Mars is rich in salts beyond NaCl, especially MgSO4. We have been using an iterative matrix of salts relevant to Mars in order to test the growth of these select bacterial isolates in the presence of various mixtures of ions at concentrations high enough to lower water activity. Liquid broth media was made for each salt at different concentrations and inoculated with each of the 18 bacterial isolates. Growth was recorded over a 12-day period using A600 measurements of turbidity. The shake-tube cultures were incubated in the dark at room temperature. The completion of this research project will give us an idea of the chances of bacterial growth on Mars and will determine qualities of the life forms that can potentially inhabit the planet. Our work on bacterial growth will assist in developing planetary protection protocols for the Red Planet and limits to habitable regions to explore for evidence of extant life.

Supported by NASA ROSES PPR and K-INBRE

46. **Anti-inflammatory drugs inhibit bradykinin-induced changes in epithelial ion transport**

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The current investigation seeks to characterize the link between prostanoid exposure and epithelial function in hormone sensitive tissues. In vivo intestinal exposure to non-steroidal anti-inflammatory drugs (NSAIDs), which inhibit prostaglandin (PG) synthase, compromise the epithelial barrier. We will test the hypothesis that PGs modulate ion transport and tight junctions in epithelia derived from hormone-sensitive tissues. Epithelia from reproductive and intestinal tissues are grown on permeable supports and electrophysiological parameters (transepithelial potential difference and short circuit current; Isc) are evaluated in Ussing-style flux chambers, to determine transepithelial electrical resistance (Rr), a sensitive indicator of tight junction integrity. Proteins and mRNA are harvested from cells to assess expression of elements in the prostanoid signaling pathway along with transport and junctional proteins. Chronic exposure of porcine vas deferens epithelia to NSAIDs slows tight junction formation and decreases expression of MRP4, which is thought to export prostaglandins. T84 cells, derived from human colon carcinoma, respond to lysyl bradykinin (LBK) with a transient increase in Isc, PGE2, and in one case, PGD2 caused a sustained increase in Isc, indicative of sustained anion secretion. NSAID exposure abrogates the response to LBK, but does not affect responses to prostaglandins, suggesting that T84 cells express PG synthase in basal conditions. Ongoing studies are designed to determine LBK-induced changes in prostaglandin production/export and to determine whether NSAID-associated changes in LBK-induced ion transport are correlated with changes in PG synthase activity, MRP4 expression, cytosolic cAMP generation and/or tight junction integrity.
47. Using fluorescent tags and TIRF microscopy to monitor palladin interactions with actin

Abby Jurgensmeier, Linh Nguyen, MarcArthur Limpiado, Moriah R. Beck
Chemistry Department, Wichita State University

Research in the Beck lab focuses on the actin-associated protein palladin, which plays a critical role in cytoskeletal organization in both normal and cancerous cells. We have recently shown that palladin contributes to actin dynamics in three distinct ways: nucleation of actin, crosslink formation, and filament stabilization. We hypothesize that palladin directly influences cell motility through simultaneous regulation of actin polymerization and organization. Our current work involves direct visualization of actin assembly and protein dynamics using Total Internal Reflection Fluorescence (TIRF) microscopy and Fluorescence Resonance Energy Transfer (FRET). Both experiments require the engineering of fluorophores onto terminal palladin domains. Here, we will describe our work with several different fluorophores that can be used for TIRF and FRET experimentation. Preliminary TIRF and co-sedimentation analyses have been carried out to determine if the fluorophores interfere with the actin-binding domain of palladin. Through these methods, we hope to understand how palladin contributes to actin-based cell motility in metastatic cells, which will be important for developing new therapies to specifically target this step in cancer progression.

48. Continued development/testing of improved methods for immunohistochemistry

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Our approach to development of an improved human cancer patient “avatar” system is based on a uniquely convenient xenotransplantation site (the hamster cheek pouch) and we are currently focusing it on high-grade serous ovarian cancer (HGSOC) and head and neck squamous cell cancer (HNSCC). An important developmental aspect of that approach involves characterizing potential donor tumor cell lines and tissues at the proteomic level via immunohistochemistry (IHC) analyses. While applying an established IHC protocol to the profiling of our current collection of HGSOC and HNSCC cell lines with a number of antibodies, we recently noted some anomalous results. That is, some IHC runs performed with newly made batches of blocking agent solution and/or detection reagent dilution solution yielded either heavy generation of non-specific signals or complete lack of positive signals. Using an antibody directed against an established housekeeping gene product (β-Actin protein), we resolved those solution anomalies and, surprisingly, determined that they primarily depended on the commercial source of a general protein blocking agent (non-fat dry milk). We then used the positively tested solutions to perform IHC analyses of two HGSOC cell lines (Kuramochi and Ovsaho). The regulatory gene protein products targeted in those IHC analyses included BAG-1, SRC-1, IRF-1, and RAR-β. For those targets, we detected specific binding signals that were primarily cytoplasmically localized in both cell lines and, overall, they were considerably stronger in the Ovsaho than the Kuramochi cells. These observations demonstrate the need to routinely assess the performance characteristics (specificity, sensitivity) of antibody-based proteomic profiling studies.

49. Investigation of congenital hydrocephalus in Specc1l-deficient mice

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Congenital hydrocephalus is a genetically heterogeneous condition affecting 1/2000 live-births in which cerebrospinal fluid (CSF) accumulates in the ventricles of brain, resulting in expansion of the skull, compression of brain tissue, and impaired brain function. Patients identified with autosomal dominant SPECC1L mutations develop craniofacial abnormalities, including some with enlarged brain ventricles. To understand the role of SPECC1L in this process, Specc1l-deficient mice were generated and analyzed. On a C57BL6/J background, homozygous mutants show perinatal lethality. However, on an FVB background, mutants survive and develop hydrocephalus with a dome-shaped head, enlarged ventricles, and stunted growth. Histological analysis confirms development of enlarged lateral ventricles in homozygous Specc1l mutants as early as embryonic day 14.5. This embryonic onset suggests an early defect in CSF production from the choroid plexus, or in development of CSF drainage system. In mutant embryos, the choroid plexus displays hypochromatic ependymal cells and disorganized branching pattern. A literature search and Ingenuity Pathway Analysis (IPA) of known hydrocephalus genes predicts SPECC1L to be in an AKT-dependent network. Indeed, we have previously shown that SPECC1L deficiency reduces AKT activity and leads to cell adhesion and migration defects. Thus, disorganization of choroid plexus and ependymal cells, and disruption of SPECC1L-AKT network in ependymal cells may be the underlying cause of hydrocephalus in these mutants. Future work will investigate cell polarity in ependymal cells and cell adhesion in the CSF drainage system. This mouse model validates the human condition and will serve as a valuable tool in understanding congenital hydrocephalus.

50. Cytotoxicity of a novel antibiotic protein

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Purified soybean glucanase protein expressed exogenously in yeast displayed powerful antibiotic activity against multiple species of gram-negative bacteria in a dosage dependent manner. We are testing the protein for adverse toxic effects on mammalian cells, using the human T47D breast cancer line. Previously, we employed the CCK8 kit to measure cell viability after exposure to 17, 33, and 67 mg/ml concentrations of protein within the effective range of antibiotic activity. Active protein was compared to equal concentrations of mutant inactive protein for direct comparison of cytotoxicity results. Other negative controls included buffer without protein and equivalent concentrations of BSA. This previous data did show evidence of toxicity but has not been consistently reproducible. We are now turning to flow cytometry to determine percentages of viable cells to obtain an unambiguous cytotoxicity result.
51. **Proteo-genomic analysis of butterfly sperm using shotgun mass-spectra data.**

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This research uses bioinformatic analysis of mass-spectra proteomic data to characterize the molecular composition of dimorphic sperm in Lepidoptera. Males of the Lepidoptera (moths and butterflies) produce two types of sperm. This dimorphic sperm is either nucleated or anucleated, respectively known as eupyrene or apyrene sperm. Apyrene sperm are unique because, along with having no nucleus, they also have no nuclear DNA, which seems counterproductive for it to be a reproductive cell. Previous studies have looked at the predicted protein composition of the sperm based on the official gene set of the Monarch butterfly (*Danaus plexippus*). However, these studies did not explore the potential for sperm proteins originating from currently unannotated genomic regions, which is a primary aim of our current research. To do this, a six-frame translation is performed on the entire genome of *Danaus plexippus*. Previously obtained mass spectra data are searched against all six-frame translations, and then cross referenced against the official gene set. This allows for the identification of previously unannotated peptides that are present in sperm. Identifying the function of the unique proteins found in apyrene sperm should illuminate the overall function of apyrene sperm.

52. **The relationship between food exposure frequency and expression of AMP-activated protein kinase (AMPK) mRNA in the brain, liver, and muscle of channel catfish.**

Kostner, Danica, Megan Dougherty, Abigail Schmidtberger, Yass Kobayashi  
Department of Biological Sciences, Fort Hays State University

Obesity poses serious challenges to our healthcare system with serious consequences of associated diseases. Our laboratory has been examining how the expression of genes involved in food intake and tissue nutrient metabolism is associated with the development of obese-like phenotypes in channel catfish. The objective of the study was to examine changes in expression of AMPK mRNA in response to changes in feeding frequency. Fish were fed every 12 (overfed), 24 (control), or 48 (underfed) hours (n= 4 tanks per treatment, 8 fish per tank) for 28 days. Brain, liver, and muscle tissue were collected at the end of the study. The expression of the AMPK mRNA was examined using quantitative RT-PCR. The muscle expression of all AMPK subunit mRNA was unaffected by feeding frequency. In contrast, brain expression of alpha 1 and beta 1 subunit mRNA had a linear tendency for increase in response to increased feeding frequency. Hepatic expression of alpha 2 and beta 1 mRNA was lower in the overfeed group compared to those assigned as control or underfeed. Hepatic expression of alpha 1 mRNA decreased linearly in response to increased feeding frequency. Our results demonstrated that the hepatic and the brain expression of AMPK mRNA was influenced by increase in exposure to food in a transcript-specific manner. Given that the AMPK serves as a regulator of tissue nutrient sensing and metabolism in mammals, it is possible that AMPK may share a similar function in the brain and liver but not in the muscle of channel catfish.

53. **Relationship between O-linked N-acetylglucosamine Transferase (OGT) mRNA Expression and Nutritional Status in the Channel Catfish Muscle.**

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Various intracellular “switches” such as O-linked N-acetylglucosamine Transferase (*OGT*) serve as a nutrient sensor and activate or inactivate numerous intracellular metabolic pathways in response to the nutritional status of an organism. In our previous studies, brain and liver expression of OGT mRNA was influenced by the nutritional status in channel catfish. However, expression of OGT mRNA in response to changes in nutrition is unclear in the muscle. The objective of the current study was to examine the relationship between expression of OGT mRNA and the nutritional status in the muscle of channel catfish. The muscle samples were collected from two different feeding studies. In the first 28 day study, fish were assigned to the control (fed once a day), fasted (no feeding), or refed (fasted for the first 14 days and fed once a day for subsequent 14 days) treatment. For the second separate feeding study, fish were fed once every 48 or 24 hours, or twice every 24 hours for 28 days. Muscle expression of OGT mRNA was measured from the samples collected on day 28 of the respective studies using real-time polymerase chain reaction. Changes in feeding frequency did not influence the expression of OGT mRNA in the muscle, but fasting fish tended to increase the muscle expression of OGT mRNA in channel catfish. The results of this study along with other studies from our laboratory indicate that muscle expression of molecular switch genes such as OGT is somewhat insensitive to the nutritional status.

54. **Examination of Structure Function Patterns in Non-Conserved Regions of Lactate Dehydrogenase (LDH).**

Frank Kutilek, Eric Guo, Dr. Moriah Beck  
Wichita State University Department of Chemistry

Non-conserved regions of proteins have been shown in the lab to act as a rheostat in protein function. When mutations are made to non-conserved residues, the functionality of these proteins change gradually. The main purpose of this research is to observe the effects that mutations to the non-conserved regions of the enzyme lactate dehydrogenase (LDH) have on enzyme kinetics, protein structure, and stability. LDH catalyzes the conversion of lactate into pyruvate as well as the reverse reaction. Here, we compared the thermal stability, catalytic rate, as well as the substrate affinity of wild type LDH as well as several mutations at glycine 68 (G68). Glycine 68 in LDH is in a non-conserved region and we substituted with histidine because the pKa is close to neutral pH and alternately to alanine because it is found in other species of Barracuda. In order to produce the specific mutants, site-directed PCR mutagenesis was utilized. All LDH proteins were purified from bacterial cultures and the enzyme activity was measured spectrophotically by monitoring production of NADH. Circular dichroism was utilized to determine the temperature at which these proteins unfold to compare stability. The benefit of this research is to develop a better understanding of what these mutations will have on the protein as well as being able to distinguish between the major and minor consequences of such mutations.
55. **RNA Interference of X-Box Binding Protein 1 in Acyrthosiphon pisum**

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Pea aphids, *Acyrthosiphon pisum*, are a significant pest to legumes, *Fabaceae*, throughout the world, primarily due to the species serving as a vector to many *Fabaceae* viruses. *A. pisum* is a model organism for biological investigation because its genome is sequenced and annotated. Current management of *A. pisum* includes use of insecticides and the introduction of natural predators. The utilization of RNA interference (RNAi) presents an alternative, pest-specific targeting of *A. pisum* rather than introducing natural predators or insecticides that can affect a wide variety of species. X-Box Binding Protein 1 (XBP1) is involved in the regulation of the unfolded protein response to promote proper folding in the endoplasmic reticulum (ER). RNAi targeting the XBP1 gene could result in the accumulation of misfolded proteins in the ER lumen, which downstream results in death of the cell via apoptosis. RNA was isolated from *A. pisum* and reverse transcribed to synthesize cDNA. The cDNA was used as a template for XBP1 primers to synthesize XBP1-dsRNA for use in RNAi feeding studies. The XBP1-dsRNA will be conjugated to Branched Amphipathic Peptide Capsules (BAPCs), which will serve as a delivery unit to prevent degradation of the dsRNA by RNases in *A. pisum*. Death curves of treated aphids, aphids fed only BAPCs, and a control group will be compared to determine if the interference of XBP1 results in *A. pisum* death, which is expected.

56. **Synthesis of Unsymmetrical 2,2'-bipyridine Derivatives Via a Phosphorus Extrusion**

Skyler Markham, Zachary Araki, Benjamin Wicker, Bruce Atwater
Fort Hays State University Department of Chemistry; Southeastern Louisiana, Department of Chemistry and Physics

2,2'-bipyridine derivatives are a common motif in inorganic ligands, natural products, and pharmaceutical agents. The synthesis of these important species has become of increasing interest due to their use in photocatalysts. Herein we report a novel synthesis for unsymmetrical 2,2'-bipyridine derivatives via a general phosphorus extrusion method which can be run under ambient conditions without the need to employ exotic ligands or transition metal catalysts.

57. **Amplification and Analysis of Genes for hFSH Glycoform-Specific Antibodies in Hybridoma Cell Lines**

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Human follicle stimulating hormone (hFSH) plays a role in female and male fertility. FSH has α and β subunits, which can be N-glycosylated to produce different glycoforms with varied bioactivities. Dr. Bousfield’s laboratory discovered that the β subunit could be differentially N-glycosylated, creating hFSHβ1 and hFSHβ2 glycoforms. Intriguingly, as women age, the more potent and active hFSHβ1 becomes less abundant, whereas the less active hFSHβ2's abundance increases. Because hFSH is important for follicle maturation in females, the ratio of the glycoforms may carry indications of fertility and reproductive age. The characteristics of hFSHβ1 and hFSHβ2 has the potential to revolutionize fertility assessments and IVF treatment. To characterize these glycoforms and develop effective assays for clinical diagnosis, pure forms of hFSHβ1 and hFSHβ2 are needed. To solve the bottleneck in glycoform purification, glycoform-specific antibodies were generated. Because the expression level of these antibodies in hybridoma lines was low, we proposed to clone the antibody genes from various hybridoma cell lines and use recombinant DNA technology to produce recombinant antibodies. The research focus is to amplify the variable regions of antibody genes and obtain their sequences. We began this process by isolating RNA from the hybridoma cells and using RT-PCR to amplify our targets. The amplified DNA fragments were then cloned, sequenced, and analyzed using BLAST. Up to date, seven cell lines have been characterized. We hope to use the obtained sequences for engineering antibody genes in the future.

58. **Cannabidiol Oil Does Not Have Adverse Effects on Fetal Development in ICR Mice**

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Cannabidiol (CBD) is a non-psychoactive, cannabis-derived compound that is becoming increasingly popular as an alternative to traditional pharmacotherapies for anxiety, depression, and pain. It is widely and legally available in most states, and as a “natural alternative”, it is not unlikely that it might be used by pregnant women seeking to avoid exposure to prescription drugs. The purpose of this study was to determine the effects of chronic prenatal exposure to ICR mice. Mated females were randomly assigned to one of 4 treatment groups: 1) vehicle control (DI H2O) 2) 25 mg/kg/d CBD 3) 50 mg/kg/d CBD or 4)100 mg/kg/d CBD (from THC-free full spectrum water-soluble hemp oil). Dams were orally dosed by gavage from gestation day (GD) 6-16, and sacrificed on GD 17. The fetuses were removed and examined, then preserved for further study. No differences in maternal weight gain, fetal weight, litter size, or embryotoxicity were observed among the groups (p > 0.05). Initial exams indicate that morphological abnormalities were not present in any of the treatment groups or in the vehicle controls. CBD oil did not cause any clinical signs of maternal toxicity, and appeared to be well tolerated. The results of this study indicate that CBD oil does not appear to cause adverse maternal or fetal effects in ICR mice, even at pharmacological dosages.
59. Optimization of Custom-built Capillary Electrophoresis Coupled with Laser-induced Fluorescence Detection

Alec McDaniel, Qiyang Zhang
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Capillary electrophoresis (CE) is a process that takes advantage of narrow-bore fused-silica capillaries to perform high-efficiency separations of ions under electrical field. Ions separate differently with a change in size, shape, and charge. Electrophoresis is especially useful when working with amino acids and DNA because molecules containing amine groups can be easily separated by electrophoresis. Electrophoresis is an essential part of forensic science and electrophoresis techniques are continually being optimized. The aim was to improve the limit of detection (LOD) on a custom-built CE system coupled with laser-induced fluorescence (LIF) detection.

Research is in progress to best optimize this CE-LIF system. First, to help block light, the stage was improved, and the objective was surrounded to limit light interference. Second, several objectives were tested and more than 100-fold difference in signal strength was observed. Third, to minimize the light reflection between the objective and the capillary, oil-immersed objective was used here. However, the oil tends to move and causes error in the readings. The next step to fix this could be a spray to use on the capillary that repels the oil, or a sleeve around the capillary. More optimization of the instrument can be performed towards laser voltage, separation voltage and capillary length. For future work, this CE-LIF system will be used to analyze the amino acids in soy sauce and use the results to prove accuracy, precision and stability.

60. The Effects of OGT Knockdowns on Mitochondrial Functions in SY5Y Cells

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The addition of a single β-N-acetylgalactosamine (O-GlcNac) to the serine/threonine residues of proteins found in the mitochondria, cytoplasm, and nucleus influences many processes within the cell including transcription, translation, and signaling. The addition and removal of this modification is carried out by two specific enzymes, O-GlcNac Transferase and O-GlcNase, respectively. The cycling of this molecule is highly dynamic and responds quickly to changes in the extracellular environment. Previously, we found that O-GlcNac was a critical regulator of mitochondrial function with over expression of either enzyme causing a disruption of mitochondrial protein expression, including those found in the Electron Transport Chain. These experiments showed a critical link between O-GlcNac levels and overall mitochondrial metabolism. Now, we have generated OGT knockdowns in SH-SY5Y neuroblastoma cells. These cell lines show 30-50% reduction in OGT levels as well as a decline in OGA. Interestingly, we found a reduction in Sirt1 and Sirt3 deacetylases, as well as the mitochondrial transcription factor, TFAM. To determine the effects that these changes had on the cell, we ran mitochondrial and glycolytic stress tests and found changes in the overall energetics of the cell. Overall, it was found that the reduction in OGT expression had an impact on cellular energetics, which could possibly be linked to acetylation. These findings open the door for a new mechanism in which O-GlcNac regulates metabolism and cellular energetics.

61. The Effects of Sleep Deprivation on Gene Expression in Drosophila melanogaster

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All animals that have been sufficiently studied have been shown to exhibit a sleep-like rest state. Drosophila melanogaster is no exception, showing all hallmarks of mammalian sleep, including sleep rebound—an increase in sleep following a period of sleep deprivation. Sleep deprivation has been shown to negatively impact learning and memory, and is associated with various physical and mental disorders such as depression and obesity in humans. Despite its evolutionary conservation, the mechanisms underlying sleep rebound remain unclear. To investigate sleep rebound in flies we used a mechanical sleep deprivation device and monitoring system. Over multiple experiments we routinely observed significant sleep rebound in flies from the A4 strain of D. melanogaster, with flies showing a significant increase in rest following sleep deprivation compared to non-deprived animals. We identified two other strains exhibiting the same pattern, and a fourth that showed no rebound, confirming the observations of other research groups that the response to sleep deprivation is genetically variable. To identify genes impacted by sleep deprivation we used RNAseq to examine brain-specific, genome-wide expression patterns. In a pilot experiment using two sleep-deprived and two control samples, we identified 74 genes significantly differentially expressed following a period of sleep deprivation. This set includes genes involved in development, immune function, and the nervous system, including genes known play a role in maintaining sleep and circadian rhythms. In ongoing work we are generating additional replicates to increase our ability to find genes that regulate the response to sleep deprivation.

62. Hold

63. Bioinspired synthesis of molybdenum carbide for hydrogen evolution reaction

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Hydrogen is one of the greenest fuel which can be generated via water-splitting, however, this process requires an efficient electrocatalyst to reduce the overpotential. Currently, precious metal such as platinum is being used in this process which makes hydrogen production costly. The challenge in hydrogen generation via water-splitting is to reduce the use of precious metals or replace them with earth-abundant materials. Molybdenum is one of the most abundant transition metals which could be used for the synthesis of cost-effective electrocatalyst for hydrogen production. In this work, we have used carbon from almond for the synthesis of molybdenum carbide as an electrocatalyst for sustainable and affordable hydrogen production. Structural characterization using X-ray diffraction confirmed the crystalline nature of the molybdenum carbide and its phase purity. Electrochemical characterizations were carried out to understand the electrochemical behavior of the molybdenum carbide as an electrocatalyst. Linear scan voltammetry, electrochemical impedance spectroscopy, and chronoamperometry were implemented to inspect the hydrogen evolution activity of molybdenum carbide. Molybdenum carbide showed an overpotential of 117 mV and 180 mV in alkaline and acidic media, respectively to achieve a current density of 10 mA/cm², which are among the best-reported results. Our facile method for the synthesis of molybdenum carbide using bio-based and earth-abundant materials for hydrogen evolution reactions opens a new pathway for cost-effective production of hydrogen as a fuel via the water-splitting process.
64. **The Q neuroblast transcriptome reveals novel transcription factors involved in Q descendant migration.**

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The migration of neurons is controlled by a complex system of differential gene expression, the understanding of which is fundamental to understanding the conditions that arise from improper migration. The Q neuroblasts and their descendants undergo long-range anterior-posterior migrations. On the right side of the animal, the QR descendant AQR migrates anteriorly to the anterior deirid behind the pharynx, and on the left, the QL descendant PQR migrates posteriorly past the anus to the phasmid ganglion. We used FACS and RNA seq to determine the Q cell transcriptome. We used cell-specific genome editing and mutant analysis to score AQR and PQR defects of knockdown of genes identified by RNA seq. Transcripts of four genes encoding Onecut-class homeodomain molecules (ceh-29, ceh-38, ceh-39, and ceh-41) were enriched in Q cells, and ceh-21 and ceh-39 controlled AQR and PQR migration. Early enriched were three High Mobility Group transcription factor family members, hmg-1.1, hmg-1.2, and hmg-1. Two alleles of hmg-1.2 displayed PQR migration defects. These results show a previously unknown role of ceh-21, ceh-39, and hmg-1.2 in Q descendant migration, and that genes with transcripts enriched in the Q cells represent a fertile new source for identification of genes involved in neuroblast migration. We thank the members of the Lundquist lab, the Kansas Infrastructure Network of Biomedical Research Excellence NIH P20GM103418, and the KU Genome Sequencing Core Laboratory, part of the Center for Molecular Analysis of Disease Pathways, an NIH Center for Biomedical Research Excellence NIH P20GM103638.

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65. **Magneto-Plasmonic nanosensor for the Detection of Ebola Virus**

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Ebola virus is one of the hemorrhagic fevers that has a high mortality rate with no cure or vaccine. The virus is spread via direct contact with body fluids from an infected individual or contact with infected bats and primates. To increase survival rate against the virus, a rapid detection method must be available. Though there are few point-of-care methods available for Ebola detection, they are not quick and economical. They require more sophisticated methods and resources for identification of the virus.

Herein, we present a novel antibody-conjugating magneto-plasmonic nanosensor (MPnS, gold/iron oxide nanocomposite) for the multiparametric detection of Ebola virus within minutes. Low limit of detection (LOD) of virus is possible utilizing MPnS due to its tri-modal detection capabilities via colorimetric, surface plasmon resonance (SPR) and magnetic relaxation (MR) systems. The gold nanoparticles in the MPnS offer SPR and colorimetric detection, whereas iron oxide nanoparticles enable the use of T2 magnetic relaxation technique. Early detection of Ebola and binding receptors will be analyzed using this technology, results will be discussed in this presentation.

66. **Peptide-polymer Amphiphiles with Different Properties for Studying Biological Interactions**

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Peptide-polymer amphiphiles (PPAs) are tailor-made polymers that can be formulated into nanoparticles with different properties. The peptide component imparts biological functionality to the polymers. Such nanoparticles have the potential to be used for various biomedical applications including novel therapeutic and drug delivery applications. Thus, in addition to developing new strategies to treat diseases, it is important to investigate how these nanoparticles interact with and affect biologically-relevant systems. The goal of this work is to determine how the physical properties of PPA-based nanoparticles affect their interactions with blood and cells. Nanoparticles with different size, surface charge and shape will be synthesized. To that end, we have synthesized the components of the PPAs. Four different monomers have been synthesized. One is a phenyl monomer that will form the core of spherical nanoparticles. The other three are peptide monomers that will give the PPAs and thereby the nanoparticles an overall positive, negative or net neutral charge. In addition, a modified Grubbus second-generation catalyst has been synthesized to make PPAs by ring-opening metathesis polymerization. Different PPAs are being synthesized for formulation into nanoparticles with different properties to then study their biological interactions.

67. **Cellular responses to chronic oxidative stress in Poecilia mexicana**

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Extreme environments are characterized by harsh physiochemical stressors that can affect the mechanisms an organism uses for its normal biological processes. *Poecilia mexicana*, an extremophile fish that inhabits hydrogen sulfide (H2S)-rich environments, is an ideal system to study how organisms modify physiological processes in response to an environmental stressor. H2S is a naturally occurring toxin in these springs that reversibly binds to cytochrome c oxidase (COX) in Complex IV of the mitochondrial respiratory chain, inhibiting ATP production and increasing reactive oxygen species (ROS). Sulfide-tolerant fish that lack a modified COX structure have the same concentration of ROS as intolerant fish. Current data suggests that extended exposure to H2S (5 hours) results in minimal differences in ROS production across tissues and across populations in lab-reared individuals. To assess if there are differences in acute exposure, we will conduct an acute exposure assay and measure ROS from collected tissues. Lipid peroxidation will also be used as a metric to assess levels of oxidative damage in the presence and absence of sulfide in two population pairs of *P. mexicana*. By comparing cellular responses to environmental stress in fish tolerant to H2S conditions and their ancestral non-tolerant populations, we can shed light on mechanisms used to mitigate the negative effects of oxidative stress in a natural system.
69. HD-Zip transcription factors: an evolutionary engine

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About 450 million years ago, land plants evolved from freshwater charophycean green algae. This was potentially facilitated by transcription factors of the homeodomain leucine-zipper (HD-Zip) family that are highly conserved across all land plants. These developmentally important proteins contain a stereodioxidic acute regulatory (STAR) protein-related lipid transfer (START) domain that is required for transcriptional activity. HD-Zip proteins regulate epidermal differentiation and drought tolerance in several crops, including rice, cotton, and maize. Arabidopsis thaliana (gl2) mutants that exhibit defective trichomes due to deficiency of a class IV HD-Zip transcription factor were transformed with two other HD-Zip family members in an attempt to rescue the mutant phenotype. Transformants with SpHDZ24, from the charophycean green algae Spirigera pratenis, failed to rescue the defects despite high levels of nuclear expression. However, multiple lines transformed with Arabidopsis PROTDERMAL FACTOR2 (PDF2) displayed an unexpected gain-of-function phenotype: stunted growth and malformed leaf rosettes. START domain mutation PDF2Q267K was identified, but was determined by comparison with a PDF2 control not to be causative. A new mutation, PDF2R1002, was generated, changing a key amino acid in the third helix of the homeodomain necessary for DNA binding. Plants transformed with this construct showed nuclear expression and normal growth. This result indicates that the transcription factor, and the novel phenotype associated with its ectopic expression, is dependent on the homeodomain’s DNA binding ability. Future work will focus on the function of ancestral transcription factors in the charophycean green algae Penium marginatum, representing the closest evolutionary lineage to land plants.

70. Microbiome of a kleptoplastic dinoflagellate

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ABSTRACT

Kleptoplasmy, gaining nutrition from the photosynthates of “stolen” chloroplasts, appears to mimic the early stages of chloroplast acquisition. We are interested in characterizing cellular processes involved in maintaining these functional, foreign chloroplasts. Gymnodinium acidotum is a freshwater, kleptoplasticid dinoflagellate that emerges from sediments as an aplastidic cell, but soon thereafter becomes photosynthetically by ingesting chloroplasts and nuclei from cryptomonad algae in the genus Chroomonas. We are only able to maintain G. acidotum in co-cultures with Chroomonas sp. Our short-term goal is to formulate an organic growth medium that replaces chloroplast nutrition in order to maintain unialgal populations. Before using antibiotics to eliminate bacteria (and any possible obligate symbionts), we characterized the 16S metagenome of individual dinoflagellate cells. Two bacterial genera, Stanieria and Riverketsia, were present at low densities in plastid-containing G. acidotum cells, but these genera were not associated with aplastic G. acidotum cells. It is possible that Stanieria and Riverketsia are symbionts associated with the Chroomonas algal cells ingested by G. acidotum. We have also amplified and sequenced the 18S rDNA of cryptomonad nuclei sequestered in dinoflagellate cells taken directly from the lake. These data indicate that the cryptomonad kleptobiont is 94-98% identical to Chroomonas coerulea. We will begin antibiotic treatments on aplastic G. acidotum cells to eliminate transient microbes so that we can grow the dinoflagellate in an organic medium. We will then use whole genome sequencing to identify genetic elements transferred from the cryptomonad to the dinoflagellate genome.

71. Characterization of the in-vivo functions of putative lysoglycerophospholipid acyltransferase in Arabidopsis thaliana

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The protein encoded by Arabidopsis thaliana gene At1g78690 has lysoglycerophospholipid acyltransferase activity when overexpressed in Escherichia coli, but its function in planta remains elusive. At1g78690 shows homology to TAFAZZIN (TAZ), a human gene involved in cardiolipin remodeling. Mutations in TAZ are linked to the human genetic disorder Barth syndrome, but the disease mechanism is unclear. It is hoped that characterization of At1g78690 will lead to a better understanding of TAZ, expanding the basis for designing treatments of TAZ-related conditions. Analysis of leaf lipids from WT and T-DNA insertional mutants revealed lipid changes that suggest a possible role of At1g78690 in the acyl editing cycle of the Kennedy lipid biosynthetic pathway. The hypothesized role will be tested by stable isotope labeling to determine acyl editing flux in WT and mutant samples. In addition to lipid changes in the At1g78690 mutants, an early senescence phenotype was observed. To confirm that the lipid and senescence phenotypes are indeed due to the knockout of At1g78690, mutant x WT crosses were performed and progeny analyzed for co-segregation of the mutation and phenotypes. Additionally, production of mutant lines with a WT At1g78690 insertion is in progress to demonstrate complementation of mutant phenotypes. A cDNA version of At1g78690 with an enhanced yellow fluorescent protein tag is also being inserted into Arabidopsis to localize the protein product intracellularly and shed additional light on its in-vivo role. Future work includes analysis of mRNA expression and domain switching based on TAZ-At1g78690 homology. Support is from K-INBRE (NIH P20GM103418) and NSF (MCB 1413036).
72. Structure-Guided Design and Optimization of a Novel Series of Dipeptidyl Inhibitors of Norovirus 3CL Protease

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Human noroviruses are the leading cause of acute gastroenteritis ("stomach flu") in the United States, resulting in greater than 20 million reported cases annually. The symptoms associated with norovirus infection (vomiting, diarrhea, and generalized gastrointestinal discomfort) are especially dangerous for young children, immunocompromised patients, and the elderly. The impact of norovirus on public health worldwide is significant and further exacerbated by the current lack of therapeutics or prophylactics. Targeting norovirus 3CL protease (3CLpro), an enzyme essential for virus replication, may lead to the emergence of effective anti-norovirus therapeutics. A novel series of dipeptidyl transition-state mimics of 3CLpro was designed and shown to have efficacy in a small animal model of norovirus infection. Further optimization of potency, oral bioavailability, and toxicity of prospective inhibitors using a structure-guided approach is anticipated to result in the identification of a drug candidate suitable for further development as a human norovirus therapeutic. Acknowledgements: This work was supported by the National Institutes of Health (AI109039, P30GM110761 and P20GM103418), the U.S. Department of Energy (DE-AC02-06CH11357), and the Industrial Macromolecular Crystallography Association.

73. Defining the mechanism of eEF1A regulation by the Legionella pneumophila effector SidI

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Legionella pneumophila is an intracellular pathogen that causes an inflammatory pneumonia in humans called Legionnaires’ disease. SidI is one of over 300 effector proteins that are translocated into infected host cells by L. pneumophila. SidI interacts with eukaryotic elongation factor 1A (eEF1A). The protein SidI can dysregulate eEF1A, which can result in serious cellular pathologies that can damage the host cell. To understand how SidI regulates eEF1A a genetic approach will be taken to try to reveal molecular details of the interaction between SidI and eEF1A. PCR will be used to amplify the two predominant domains of the SidI protein. After these domains are amplified, they will be cloned into a plasmid to create a fusion to glutathione-S-transferase (GST), which binds to glutathione. Next the GST-SidI proteins will be purified from Escherichia coli and used to determine which region of SidI interacts with eEF1A. The purified GST-SidI proteins will be immobilized on glutathione-coated beads and then will be incubated with lysates from mammalian macrophage, which contain eEF1A. Western blot analysis will be performed to determine which amino acid residue(s) are important for the interaction. Which regions of eEF1A that are targeted by SidI need to be determined. This will be done by generating truncations of eEF1A. eEF1A is important for numerous cell biological processes in eukaryotic cells, all of this will lead to a better understanding of how eEF1A is regulated by SidI.

74. Annotation of Transcription Start Sites for Motif Analysis

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Since the completion of its genomic sequence in 2000, Drosophila melanogaster has served as a model organism for studying many developmental and cellular processes common to higher eukaryotes. Among these, the Genomics Education Partnership (GEP), of which this research is a part, is investigating the anomalous expression of genes within the Muller F element by cross-species comparative analysis of gene and transcription start site annotations. In this in silico study, the transcription start sites for genes within a contig of Drosophila biarmipes were annotated using Drosophila melanogaster as a reference. In addition, several open-source databanks were used, including results from DNase hypersensitivity, ChIP-seq, RNA sequencing, RAMPAGE and CAGE experiments. The transcription start site search regions were defined for each gene; however, the data did not provide conclusive evidence for exact locations. Data files and resources were made available through the GEP sponsored by Washington University, Saint Louis. The findings were submitted for inclusion into the Drosophila database to the Genomics Education Partnership. Supported by the Kansas INBRE, P20 GM103418

75. Effect of sulfide, lactic acid, and ethanol on respiration enzyme activities in catfish tissues

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Cellular respiration is influenced by various chemicals. Effects of sulfide on cellular respiration are well characterized. Previous studies also have shown that lactic acid and ethanol influence cellular respiration, but specific mechanism(s) that affect enzymes involved in cellular respiration have not been characterized. The objective of this study was to characterize effects of sulfide, lactic acid, and ethanol on the activity of two enzymes essential for cellular respiration: cytochrome c oxidase (CytOx) and lactate dehydrogenase (LDH). Activity of both enzymes was measured in catfish muscle and liver homogenates in the presence of increasing concentrations of sulfide (0 to 20 µM), lactic acid (0 to 100 mM), and ethanol (0 to 100 mM). CytOx activities in catfish liver samples were as high as 6.98 µmol g⁻¹ min⁻¹, while LDH activities in catfish muscle samples were as high as 176.8 µmol g⁻¹ min⁻¹. Increasing sulfide concentration significantly decreased CytOx activity but not LDH activity. Increasing lactic acid concentration significantly decreased activity in both CytOx and LDH. The inhibition constant of CytOx in sulfide was 500,000 times lower than in lactic acid. Activities of both enzymes decreased to a similar degree with increasing lactic acid concentration, whereas exposure to ethanol did not affect the activity of either enzyme, regardless of concentration. Our results indicated that sulfide and lactic acid differentially inhibited activities of enzymes critical for cellular respiration. Differential response to sulfide and lactic acid on different enzymes may have potentially influenced evolutionary and physiological function of different enzymes involved in fish respiration.
Analyzing the effect of repurposed drugs on the progression of Polycystic Kidney Disease

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Introduction: Signaling pathways activated by a loss of function mutation in polycystic kidney disease (PKD) constitute important targets of therapy for PKD. We have recently shown that Notch3 pathway was activated in PKD and was associated with disease severity. Here we explore the effects of Notch inhibition in PKD.

Methods: We selected two repurposed drugs Quinomycin A (Quin) and Ciclopirox-olamine (CPX), both of which have recently been shown to inhibit the Notch pathway and ameliorate the progression of cancer. After testing the in vitro efficacy of these drugs in PKD cells, we intraperitoneally injected CPX (10mg/kg body weight), Quin (10mg/kg body weight) or vehicle in 21day old PKD or wildtype mice. Mice were euthanized after 27 days of consecutive treatments. Kidneys and blood were harvested for further studies.

Results: Treatment of PKD mice with CPX or Quin for 27 days both resulted in a significant reduction in the percent cystic index. Both treatments were associated with decreased cell proliferation of the cyst lining epithelial cells and overall decrease in fibrosis. While Quin worked through Notch pathway inhibition in PKD kidneys, CPX did not appear to work through Notch pathway in kidneys. In fact, CPX mediated protection appeared to work through degradation of ferritin and increased autophagy.

Conclusions: Ciclopirox-olamine and Quinomycin A work through different protective mechanisms in PKD. Nevertheless, both drugs may constitute drugs of choice for PKD in the clinic.

Mutant p53 sensitizes cancer cells to stress inducers by inhibiting stress granule formation

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Abstract:
One major hurdle for cancer therapy is drug resistance. A crucial factor for drug resistance is mutation in the tumor suppressor tp53, which regulates cellular metabolism, DNA repair, cell cycle progression, and cell death. Mutations in tp53 occur in approximately 50% of human cancers, hence greatly contributing to drug resistance. Another mechanism of drug resistance is the formation of stress granules (SGs), cytoplasmic mRNA and protein aggregates, induced by various cellular stresses and mitosis-targeting chemotherapy drugs (mitotic inhibitors). Furthermore, despite that mutant p53 (mutp53) enhances chemoresistance by its gain-of-function activity, its role in SG-induced drug resistance is unknown. Initially, we hypothesized that mutp53 promotes SG formation, thereby sensitizing cancer cells to stress-induced cell death. Our findings reveal a novel GOF activity of mutp53 and may lead to the application of stress-inducing chemotherapy agents (eg, vincristine) in the treatment of mutp53-carrying cancers.

Effects of Intrinsic Aerobic Capacity on Resistance Exercise Performance in Middle-Aged Rats

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Normal aging is accompanied by physiological changes that often result in diminished cognitive and motor function. Exercise can protect against age-related functional decline. While many studies support beneficial effects of aerobic exercise, resistance exercise has received less attention. The goal of our study was to determine whether aerobic capacity influences resistance exercise engagement and performance using a rat model. We trained middle-aged low capacity runner (LCR) and high capacity runner (HCR) rats to perform a voluntary unilateral forelimb isometric exercise task. Once rats learned to press and hold the isometric force-sensing disc, we increased the force requirements to compare performance across the two groups of rats. We found that task engagement and time integral of force (a measure of “work”) were similar between the two groups at the lower force requirements, but were greater in the LCR rats at the higher force requirements. Force output was greater in the HCR rats, but LCR rats maintained longer disc presses. Body weights were greater in the LCR rats, and ECHO MRI revealed a greater percent of fat mass and lower percent of lean mass in LCR than in HCR rats. Our results show that aerobic capacity does not affect engagement in resistance exercise in this model. Despite differences in exercise performance between the two groups, LCR rats did not exhibit diminished performance in this task.

Measurement of potential Parkinsonian Toxin uptake in C. elegans using HPLC and microscopy techniques.

Romero Elmer, Wimalasena Kandatege, Murphy David, Lickteig Bryan, Department of Chemistry at Wichita State University

Parkinson’s is a neurodegenerative disease that is prevalent in older generations and currently has no cure. Parkinson’s is correlated to cell death in specific dopaminergic neurons in the substantia nigra of humans. The death of these cells causes a decrease or even complete absence of dopamine in the brain which leads to impaired motor movement, rigidity, and tremors, which are defining characteristic symptoms of Parkinson’s. This illness has been known to have a genetic component that makes people more prone to developing it, however, it has been found that environmental factors may play a greater role in development of the disease.

One compound, 1-methyl-4-phenylpyridinium (MPP+), has been studied because it inhibits complex 1 in mitochondria, and has been found to cause oxidative damage in dopaminergic cells. MPP+ has since been used as a model for the environmental causes of PD and its mechanism of action hosts a variety of mysteries. Our research consists of studying the chemical uptake of MPP+ and its derivatives in the model organism Caenorhabditis elegans and how they lead to further degeneration of these dopaminergic neurons. In addition to uptake measurements via HPLC, microscopy is used to attest for uptake and neurological damage. The nematodes are assessed under the microscope and their axons are checked for blebbing, a bulge/protrusion in the plasma membrane indicating neuronal degeneration.
80. To evaluate whether composite extracellular matrix scaffolds enhance the proliferation, migration, and differentiation of human Wharton jelly cells over single material scaffolds used in wound healing

Grant Ryals and Dr. Aj Mellott
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K-INBRE Abstract
2019 Symposium

Human Wharton Jelly Cells (HWJC) are mesenchymal stem cells that have demonstrated the ability to differentiate down a variety of different lineages provided the right environment. HWJCs are of particular interest in wound healing applications due to their differentiation potential and accessibility. Significant effort has been invested into seeding HWJCs into different acellular matrices such as decellularized tissues and Matrigel for wound healing applications. However, no one has investigated whether these materials can be combined to create a more advantageous composite material that stimulates cell proliferation, migration, and differentiation over individual materials. Here, we investigated different ways in which decellularized human Wharton's jelly matrix and Matrigel could be combined into a composite construct containing HWJCs. Our results showed HWJC's were distributed homogenously throughout the composite construct. Additionally, HWJCs were able to survive for the duration of the 10 days the experiment was conducted. The initial results suggest that the combination of materials is a viable scaffold for HWJCs, which is encouraging for exploring how composite scaffolds could be leveraged to modulate HWJCs for wound healing applications.

81. The interaction of nanofibers and oligodendrocyte progenitor cells derived from induced pluripotent stem cells

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Induced pluripotent stem cells (iPSCs) are generated from somatic cells and could provide a new source of patient-specific stem cells for replacement therapies. Though human iPSC-derived stem cell therapies have been used in various in vivo models of neurodegenerative disorders, few studies have evaluated efficacy of iPSC-derived neural stem cells (NSC) transplantation in models of spinal cord injury (SCI). Additionally, the application of human iPSCs in SCI research of preclinical animal model has been limited by the immune-response to xenografts. The nanofibers and continuous porous structure generated by electrospinning enhance neural regeneration because the nanofibers mimic the extracellular matrix and provide guidance for axonal growth at nanolevels. Nanofiber-based scaffolds may simultaneously provide immediate contact guidance for neural regeneration and act as a vehicle for therapeutic cell delivery. Additionally, nanofibers can serve as a neuron-free model to study myelination for the growth of oligodendrocyte progenitor cell (OPC) derived from iPSCs-NSC. This study showed that the nanofibers can support OPC growth. OPCs maintained their phenotype and viability on nanofibers. Cells were positively labeled with OPC markers A2B5 and MBP.

82. The evolution of Agrobacterium tumefaciens in opine and plant cue containing environments

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Host environments impose strong selective pressures on pathogens. Agrobacterium tumefaciens causes the formation of tumors in plants. Pathogenic agrobacteria harbor the Ti plasmid that encodes pathogenesis functions and the catabolism of opines—substances plant tumors release after infection. The VirA/VirG two component system regulates the expression of these virulence functions in response to cues associated with the rhizosphere of wounded plant roots. The most important of these cues are plant produced phenolic compounds, which are important in plant wound healing and innate immunity. We evolved a strain of A. tumefaciens in four environments varying in the presence or absence of opines and phenolic plant cues. As predicted avirulent mutants, notalby lacking the ability to induce the virB promoter, spread within environments containing plant phenolic cues. Counter to expectations these populations are polymorphic, with a minority of cells maintaining PvirB expression. To better understand the selective pressures associated with this polymorphism we characterized isolates from each population using a potato tissue pathogenesis assay, an octopine catabolism assay, and a beta-galactosidase assay. We found that agro bacterial pathogenesis and opine catabolism functions can evolve independently in specific environments, suggesting that the linkage of the genes encoding these functions on the Ti plasmid does not constrain the evolution of avirulent, opine catabolic genotypes. Genomic sequences of avirulent clones from several phenolic cue evolution lines indicate that transposon insertions into either virA or virG often underlie the avirulence of these clones.

83. Microsatellite Analyses of Specific Linkage Groups in Perennial Wheat Wide Hybrid Lines

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Perennial wheat lines are generated by crossing annual hexaploid bread wheat (Triticum aestivum, 2n = 6x = 42, AABBDD) and perennial wheatgrass species such as Thinopyrum elongatum (2n = 14, EE) or Thinopyrum intermedium (2n = 14, EEJSS) and doubling chromosome content with colchicine to generate amphiploid hybrids. These hybrids are promising for sustainable agriculture systems because they reduce soil erosion, despite unstable karyotypes and depressed yield and agronomic qualities in many lines. A major goal is to optimize the ratio of perennial and annual chromosomes to achieve perennial regrowth and high agronomic qualities in one plant. We sought to augment the laborious cytological methods for determining chromosome content in perennial hybrids with a PCR-based method. We examined twenty Expressed Sequence Tag / Simple Sequence Repeat (EST-SSR) markers establish in the Thinopyrum genus and assessed their polymorphism in Chinese Spring chromosome addition lines and the promising perennial wheat hybrid Salish Blue. We conducted PCR using primers flanking potentially polymorphic EST-SSR sites and detected alleles using 6% denaturing polyacrylamide gel electrophoresis (PAGE), using addition line control samples to determine marker position.
84. Genetic and cell biological analysis of eight possible alleles of Mcr in Drosophila

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Epithelial tissues cover body surfaces, internal cavities, and organs. They serve as barriers to the outside environment and create unique compartments for organ functions. For epithelial tissues to provide this function their cells must be tightly packed while also maintaining a physiological seal. This seal is achieved through cellular junctions called tight junctions in vertebrates and septate junctions (SJs) in invertebrates. The Ward lab is interested in the organization and biogenesis of the SJ in Drosophila melanogaster. Previous work in the lab identified Macroglobulin complement-related (Mcr) as a component of the SJ. A subsequent genetic screen found eight mutations that may be new alleles of Mcr. The purpose of this project is to determine if any of those eight mutations are new alleles of Mcr through three experimental techniques: complementation testing, cuticle preparations, and antibody staining. Two of these mutations fail to complement, meaning they are alleles of the same gene, while the other six are in different genes. Unfortunately, none of these mutations fail to complement a known allele of Mcr (Mcr\textsuperscript{EY07421}), suggesting that they are not alleles of Mcr. The cuticle preparations, however, show that five of the mutations have a high penetrance of head involution defects, a phenotype associated with Mcr mutations. We are currently conducting antibody staining to examine the localization of Mcr and Core in the mutant embryos. These experiments have shown that none of the mutations are in Mcr, but they may be in genes that affect the structure and function of the SJ.

85. Small Therapeutic Peptides Reduce Angiogenesis and Melanoma Growth

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Beta2 glycoprotein 1 (β2-GPI) is a high-concentration serum protein which binds to negatively charged proteins and phospholipids on apoptotic endothelial cells. Recent data shows that β2GPI can either induce or inhibit production of new blood vessels (angiogenesis). It is unclear which of the five sushi domains of β2-GPI affect angiogenesis, and the different domains may have opposing functions. We derived peptides from the fifth and binding domain of β2-GPI, as well as a scrambled control peptide. As melanoma growth requires angiogenesis, we hypothesized that the β2-derived-peptides may enhance or reduce tumor growth. We tested this hypothesis with in vitro assays which evaluated peptide toxicity, and determined endothelial cell secretions and migration changes in response to the peptides. B16-F10 melanoma cells increased vascular endothelial growth factor (VEGF) secretion by endothelial cells. The β2-GP1-derived peptides were non-toxic, downregulated the VEGF secretion, and reduced migration of endothelial and tumor cells. Finally, we monitored the effects of the peptides on tumorigenesis in mice. β2-GPI-derived peptide treatment of melanoma reduced tumor growth and attenuated vascular markers compared to scrambled peptide or saline controls. These data demonstrate that β2-GPI-derived peptides limit tumor growth through inhibition of angiogenesis. Future directions will examine additional mechanisms of peptide tumor control.

86. Dimerization Partners of HAF ABC Transporters

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ABC transporters are transmembrane proteins that use ATP hydrolysis to traffic substrates across membranes. Most organisms have a group of ABC transporter genes that are configured as half genes—encoding half of a typical ABC transporter protein. These HAF proteins must homodimerize or heterodimerize in order to be fully functional. Our lab is studying a family of HAF transporters in Caenorhabditis elegans, three of which have been demonstrated to play a role in RNA interference in this organism. As of yet, the dimerization partners of each of these proteins have not been determined. Our goal is to identify each of the dimerization partners of the HAF ABC transporter proteins in this subfamily, an essential step toward elucidating their roles in RNAi. To accomplish this goal, we attached a GFP reporter to each of the haf genes, configured as a split GFP coding region, and designed the constructs such that they can be integrated into the C. elegans genome. Performing pair-wise crosses between each of the established transgenic strains of C. elegans allows us to determine the protein dimerization partners by monitoring for recovery of GFP fluorescence in cross-progeny. Our ultimate goal is to identify dimerization partners for the half-transporters in the B-subfamily, paying particular attention to the three haf genes that are required for RNA interference. This will allow us to develop in vitro biochemical assays designed to detect and confirm potential RNAi-relevant substrates.

87. Overexpression of the RNA Binding Protein HuR Promotes Chemoresistance to mTOR Pathway Inhibitors in Colorectal Cancer Cells

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Post-transcriptional regulation of proinflammatory gene expression is an important feature in colorectal cancer (CRC) development. We have previously identified specific RNA-binding proteins that control oncogenic gene expression in tumor cells. HuR (ELAVL1) is one such RNA-binding protein that binds to AU-rich elements within the 3' untranslated region (3'UTR) of these target mRNAs and shuttles them from the nucleus to the cytoplasm. However, HuR is overexpressed early during colorectal tumor development and abnormally present in the cytoplasm where it can interfere with rapid mRNA decay. This allows for enhanced oncogenic expression that promotes tumor growth. Based on this, we hypothesized that HuR overexpression plays a central role in modulating key CRC signaling pathways via mRNA stabilization. To address this, we screened a library of established kinase inhibitors on HuR-overexpressing wild-type CRC (HCT116) cells and CRISPR/Cas9 HuR-knockout HCT-116 cells and assessed their effect on cell proliferation. Wild-type cells demonstrated resistance to mTORC1/2 kinase inhibitor PP242, whereas deletion of HuR rendered cells sensitive to PP242. mTOR signaling has been reported to increase proliferation and metabolic activity in cancer cells, and single-cell RNA sequencing and mTOR, phospho-mTOR, Pan-AKT, protein expression confirmed significant upregulation of mTOR signaling in HCT-116 cells. Furthermore, the HuR inhibitor Dihydrotanshinone-I (DHTS) sensitized HCT-116 cells to PP242 and downregulated major mTOR pathway protein expression. These findings indicate that HuR provides intrinsic resistance to mTOR inhibition in CRC cells and suggests that combinatorial treatment targeting HuR and the mTOR pathway may be an effective means to inhibit CRC tumor progression.
Proper formation of neural circuits is critical for development of the central nervous system. These neural connections underlie basic functions such as memory and learning, however, a disruption of the same wiring could lead to neurodevelopmental disorders including autism and schizophrenia. During the development of the nervous system, developing neurons extend axons that are led by a dynamic structure known as the growth cone. Thus, understanding how axons are properly guided during development to make specific connections with one another is extremely important. By using Caenorhabditis elegans (C. elegans) as a model organism, molecular pathways in axon guidance can be better understood. UNC-6/Netrin is a conserved dorsal-ventral axon guidance molecule that has been extensively studied in the Lundquist lab, however it was still unknown how the UNC-6/Netrin repulsive receptor molecule, UNC-5, inhibits growth cone protrusion and how an additional UNC-6/Netrin receptor molecule, UNC-40/DCC, works in coordination with UNC-5 to regulate proper polarity and protrusion of the growth cone. Our findings show that UNC-5 inhibits protrusion by restricting growth cone microtubule accumulation and ensures proper filamentous actin accumulation at the protrusive end of the growth cone. We also found that UNC-40/DCC assists with protrusion downstream of the polarity events established by UNC-6/Netrin and UNC-5. These results indicate both UNC-5 and UNC-40/DCC are required to properly shape and polarize the growth cone and are thus critical in guiding the axon to its proper synaptic destination.

90. Bacterial Keratinase Enzyme for the Treatment of Nail and Skin Disease

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Enzymes extracts. Our goal for this period is to advance assay development, identify the most productive microbes and active enzymes, as well identified 5 novel isolates that display an encouraging ability to degrade keratotic material. We are currently refining a ninhydrin assay to quantify extracellular proteases. The most well-studied keratinolytic organism, Bacillus licheniformis PW1, is used as a positive control for keratinase activity. For nail infections, fungi often embed themselves within the nail matrix, making it challenging to eradicate the infection with topical treatment. Keratinase enzymes have the potential to loosen nail (and skin) tissues, rich in keratin, can be impermeable to the drugs needed to treat fungal infections or underlying lesions. A variety of dermatological conditions are characterized by hyper trophy of keratotic tissues resulting in discomfort or aesthetic impairment of the afflicted, including onychomycoses, actinic keratoses, keratosis pilaris, and onychogryphosis. In nail fungal infections, fungi often embed themselves within the nail matrix, making it challenging to eradicate the infection with topical treatment. Keratinase enzymes have the potential to loosen nail (and keratotic) tissue, such that topical antifungal drugs can penetrate. A collection of ~ 100 bacterial we isolated from wild bird feathers produce extracellular proteases. The most well-studied keratinolytic organism, Bacillus licheniformis PW1, is used as a positive control for keratinase production. Feather microorganisms have been screened for their ability to degrade whole eldipiedized feathers in culture, for caseinolytic affinity of extracellular proteases on milk agar, and for keratolytic affinity of extracellular proteases on feather powder agar. The results of these tests have identified 5 novel isolates that display an encouraging ability to degrade keratotic material. We are currently refining a ninhydrin assay to quantify free-amino acid release from degraded whole feathers as a measure of keratinase activity for each promising organism to select the most potent enzymes extracts. Our goal for this period is to advance assay development, identify the most productive microbes and active enzymes, as well as move toward testing treatments on model nail infections using keratinases and antifungal drugs. We will test nail mechanical properties and porosity in collaboration with WSU engineering and a dermatologist. This work was supported by KINBRE.
92. **Phosphate-Tether Mediated Studies Towards the Synthesis of Leustroducsin B and Its Simplified Analogs**

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Department of Chemistry, University of Kansas

Synthetic efforts towards the bioactive natural product Leustroducsin B using a phosphate tether-mediated approach are reported. Leustroducsin B, a potent colony-stimulating factor inducer belongs to the family of novel phosphorylated polyene-, polyl- and pyranone-containing natural products. Our synthetic strategy towards this natural product includes ring-closing metathesis (RCM), oxymercuration/Jones oxidation, cross metathesis (CM) with a lactol-olefinic subunit, and 1,2 addition of the Li-enolate of EIOAc as the key steps for the installation of the “western segment” of this molecule. This efficient and library- amenable protocol will be further employed for the synthesis of simplified analogues of Leustroducsin B by switching the CM partner lactone with α, β-unsaturated sultone, phostone and their corresponding amide analogs. Furthermore, these compounds will be assessed through an ‘Activity Based Protein Profiling’ platform by our collaborators.

93. **Syntheses and Properties of Cyanopyrazole Metal Complexes of Interest in Modeling Enzymatic Active Sites**

*by a method previously developed in our group and have used this compound to create previously unknown copper and cobalt complexes. In this presentation, we will report the syntheses, structures, and properties of these metal complexes as well as ongoing studies aimed at elucidating the effect of the nitrile substituent on the coordinated metal.

Our research group has pioneered the study of cyano-substituted pyrazoles and their metal complexes. I have synthesized 4-cyano-3-ethylpyrazole and these compounds will be assessed through an ‘Activity Based Protein Profiling’ platform by our collaborators.

94. **Role of β-Propetipeptides in Proteasome Assembly Process**

Alexander Vontz¹, Anjana Suppahia¹, Pushpa Itagi²,³, Jeroen Roelofs¹
Wichita State University Department of Chemistry

Pyrazole, a 5-membered ring with three carbon atoms and two adjacent nitrogen atoms is very similar to the imidazole ring in the side chain of the amino acid histidine. Thus, metal complexes of pyrazoles can serve as effective models for the active sites of metalloproteins and help to provide insight to their structure and function. Pyrazoles can be relatively easily functionalized with substituents at various positions on the ring, and the inclusion of electronically non-innocent substituents, such as the strongly electron-withdrawing nitrile (cyano) group, can alter the properties of the bound metal ion. Judicious selection of substituents can provide an environment for the metal that closely mimics that of the biological system. Our research group has pioneered the study of cyano-substituted pyrazoles and their metal complexes. I have synthesized 4-cyano-3-ethylpyrazole by a method previously developed in our group and have used this compound to create previously unknown copper and cobalt complexes. In this presentation, we will report the syntheses, structures, and properties of these metal complexes as well as ongoing studies aimed at elucidating the effect of the nitrile substituent on the coordinated metal.

95. **Analyzing the Gut Microbiome of Human Populations in Fort Scott, KS and Shoal Creek, MO**

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Department of Biology, Pittsburg State University

The human microbiota is a mutualistic relationship between the human body and microbial cells. These cells distribute themselves along the internal and external surfaces of the human body. These surfaces and their respective microbiota can be divided into regions, an example being the human gut microbiome. The microbial cells in this region largely outnumber the cells of the human host and perform a multitude of functions for the host. They have major roles in host protection and immune system development, but they are also implicated in diseased states. Their exact function in these states is not yet known. This study focused on determining the presence of: *Bifidobacterium*, *K. granulomatis*, species of Bacteroidetes, and *B. timonensis* in the populations of Fort Scott, KS and Shoal Creek, MO.

Previous studies in gut microbiota have shown that certain diseases are correlated with microorganisms isolated from human samples. Our samples were raw sewage water collected from the waste water treatment plants in these areas. We utilized polymerase chain reaction with species-specific primers to analyze the genomic DNA. Their presence was confirmed via gel electrophoresis.
96. PCR Assay to detect Porcine Endogenous Retroviruses (PERV) A, B, and C

Zoeyi Wallis¹, Rashmi Acharya¹, Dr. Eric Gillock¹
¹Department of Biological Sciences, Fort Hays State University, Hays, Kansas

Xenotransplant is the process of transplanting organs from a non-human source into a human and has the potential to relieve the deficit of human allograft donors. Porcine organs and tissues are a good match to humans because they are close in size to human organs as well as studies have shown resistance to autoimmunity rejection. However, like many other organisms, porcine organs contain Endogenous Retroviruses named PERVs that are found across all breeds of pigs and could potentially give rise to lethal zoonotic infections. PERVs are gamma retroviruses closely related to other retroviruses such as leukemia viruses. These viruses are encoded in the genomic DNA of the germline and are spontaneously released in peripheral blood lymphocytes. There are three types of PERVs: A and B are found to be present in all pigs and previous studies have shown that A and B both affect humans as well as numerous other species. While they last type, PERV-C, has only been shown to affect pigs. A cross between A and C can occur and has been shown to affect human cells in the lab. The underlying reason for why some affect human cells and others do not is unknown. This research focuses on the detection of PERVs using PCR assay to visualize distinct bands identifying specific nucleotide length of the three different PERVs.

97. Morphology and Replication of Notiopedio, a Bacteriophage Infecting Bacillus subtilis

Kayden Webb, Andrew Herbig
Washburn University Department of Biology

Abstract:
Bacteriophages (phages) are viruses that exclusively infect bacteria. A phage infecting Bacillus subtilis was isolated from soil collected in southeast Kansas and has been named vB_BsuS-Notiopedio (hereafter Notiopedio). Notiopedio is a virulent phage, replicating by the lytic cycle. Within 10 minutes of infection, more than 50% of phage adsorb to host cells. From one-step growth curve data, we found that Notiopedio has a latent period of approximately 30 minutes. A single replication cycle yields a burst size of 93 virions. Based on transmission electron microscopy of purified phage, Notiopedio belongs to the taxonomic Order Caudovirales and Family Siphoviridae. Isolated genomic DNA from Notiopedio was compared to DNA from the well-characterized Bacillus phage SPO1 using restriction fragment polymorphism analysis. Notiopedio DNA restriction patterns differ from SPO1. Notiopedio is restricted in its host range, as it does not infect other species of Bacillus. We have begun to construct a phage gene expression library in E. coli in order to further characterize Notiopedio. Research with Notiopedio will further our understanding of the role of phages in soil microbiota dynamics.

98. Degradation of BAPCs by fungi

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The ability of branched amphiphilic peptide capsules (BAPCs) to encapsulate and transport into cells offers new approaches to deliver active ingredients (AIs). Until now, the capsules were completely inert in eukaryotic cells. Pictures have been collected which show that BAPCs are able to take up and break down these nanoparticles with encapsulated dye. Experiments have been designed to optimize delivery of fungicides to fungal species. Many conditions were adjusted to find what allowed for degradation. In these experiments it has been seen that one type of BAPCs can encapsulate and deliver the fungicide thiourea. They showed a zone of inhibition when spotted on an Aspergillus spread milk plate. However, consistent results have been difficult to obtain. Thiourea has been shown to be present in the isolated media of liquid cultures after exposing the fungi to BAPCs containing the AI. This result indicates that not only are the BAPCs being opened, but the fungi are also being altered such that they leak. Proteases that are responsible for degradation are now also being sought after in hopes of purification. Mutant strains with derepressed proteases show a greater zone of inhibition when spotted with BAPCs containing fungicides. This information will be used when comparing the sequence of known proteins that break down the capsules to the activity of proteases.

99. Genomic Annotation of 61,000 bp region of 3L chromosome in D. takahashii

Emily White, Washburn University Biology Undergraduate
Takrima Sadikot, Department of Biology Associate Professor

Drosophila melanogaster, the common fruit fly, is one of the most versatile model organisms in biology, used for a wide range of research centered around cellular mechanisms of complex eukaryotes. In this project we use the D. melanogaster genome as a reference to identify and annotate coding regions in Drosophila takahashii. This project is part of a collaborative effort of the Genomics Education Partnership (GEP). Here we use conservation-based analysis and various online tool such as the Drosophila melanogaster genome browser, Flybase, Gene Model Checker and Gene Record Finder to analyze a 61,000 base pair segment of chromosome 3L in D. takahashii labeled contig 7. The BLAST of D. melanogaster indicates the presence of genes Transcription factor AP-2 (TIAP-2), CR6-interacting factor (CRIF), Structural Maintenance of Chromosome 5 (SMC5), and CG32441 genes in the contig. The analysis of the D. takahashii sequence supports the BLAST results for the aforementioned genes. Discrepancy in exon number and placement was resolved using the small exon finder and protein BLAST analysis to select the most conserved model. The results and the data collected from this research will be submitted for review and inclusion to the GEP data repository.
100. Porcine reproductive and respiratory syndrome virus (PRRSV-1) recognition of peptide sequences in CD163 SRCR5

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PRRSV is an important swine pathogen that uses macrophages as important target cells for viral replication. CD163, a macrophage specific molecule involved in several homeostatic processes, was also identified as a receptor for PRRSV. Research shows that a complete deletion of CD163 SRCR5 can produce pigs that are entirely resistant to infection with PRRSV. Therefore, our goal is to find the smallest mutation in SRCR5 that will prevent PRRSV-1 infection, but also conserve CD163’s biological functions. The CD163 constructs used in this study were previously generated and tested for permissiveness to PRRSV-2 infection. Briefly, each construct carries an insertion of Proline-Arginine dipeptides (PR) at every 30 bp along the SRCR5 cDNA. All of the CD163 constructs were fused to a green fluorescent protein (GFP) which allowed visualization of the proper expression for each recombinant protein on non-permissive HEK293T cells. In order to test the permissiveness of each CD163 SRCR5 construct to PRRSV-1 infection, the cells expressing each mutant were infected with a PRRSV-1 strain, Lelystad. Infection results were visualized by IFA staining using an antibody recognizing PRRSV-N protein. The results showed a wide range of infection rates, from mutations that showed no or little effect to mutations that almost completely blocked infection. For example, insertion of TR in positions 15, 38, 62 and 78 had no effect on infection, whereas insertion of PR in positions 58 and 100 showed great reduction in PRRSV-1 infection. These results show the possible contact regions between the PRRSV viral proteins and the CD163 receptor.

101. The role of thiolase in Clostridium difficile biology

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Clostridium difficile is a major nosocomial pathogen and is the principal causative agent of antibiotic-associated diarrhea and pseudomembranous colitis. Wild-type C. difficile produces dormant spores, which facilitate transmission and persistence of the organism in the environment. C. difficile codes for Thiolase enzyme whose function is to breakdown acetoaetoacyl-CoA into two molecules of acetyl-CoA. Evidence suggests that this enzyme is produced extracellularly in C. difficile and may have a role in bacterial colonization. To determine the role of the thiolase gene in pathogenicity of C. difficile, we constructed a thiolase negative mutant by inserting a group-two intron into the thiolase gene of the JIR strain of C. difficile. We are currently performing tests to determine the role and effect of thiolase in C. difficile sporulation and toxin production. We found that the loss of the thiolase gene significantly delays C. difficile sporulation in vitro.

102. Subdomain shuffling in two similar acyltransferases to investigate acyl specificity

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The primary energy storage in many seeds is an ester of glycerol and three fatty acids known as triacylglycerol (TAG). The final step of TAG biosynthesis is the acylation of the sn-3 position of diacylglycerol by enzymes such as diacylglycerol acyltransferase (DGAT). Many DGAT enzymes belong to the membrane-bound O-acyltransferase (MBOAT) protein family, a group of enzymes that acylates a wide variety of different substrates, including lipids and proteins. Despite the importance of these enzymes for different biological functions, the membrane-bound nature of the proteins has hindered attempts to study their structure. Different DGATs with unusual substrate specificities have been discovered in the plant kingdom. For example, Euonymus fortunei acetyltransferase (EfDACt) uses acetyl-coA to acylate the sn-3 position of diacylglycerol, forming an unusual type of TAG known as acetyl-TAG. Interestingly, other MBOATs have high sequence homology to EfDACt but have specificities for regular long-chain fatty acids. To determine which regions of these proteins were responsible for determining acyl specificity, chimeric proteins combining different homologous portions of EfDACt and SaDGAT, a DGAT with specificity for long-chain acyl-CoA, were created and biochemical activity characterized. While chimeras were found to be non-functional, certain modifications maintained the functionality of the wild-type enzymes. Future plans will use different patterns of rearrangement for chimera construction, as well as to use high-throughput selection methods to screen for functional chimeras.

103. Creating Research Infrastructure in the Haskell Wetlands: Installing a Permanent Sampling Grid and Long-term Water Monitoring Sites

Joseph Zupan and Bridgett Chapin
Haskell Indian Nations University
AISES Abstract

A grid and long-term monitoring sites were designed and installed during the summer of 2018 to provide a spatial foundation for research in the Haskell wetlands, which is a critical outdoor classroom for Haskell Indian Nations University. This grid, along with 10 long-term monitoring sites, will play a vital role in any future ecological survey that may be conducted in this unique marsh adjacent to the university. These installations will provide accurate global positioning system coordinates and measurements. This will help with the physical mapping of where data is being or has been collected in the wetlands. In addition, long-term water monitoring sites in the wetlands will be equipped with temperature data-loggers (thermistors). Since the wetlands are a focal point for interdisciplinary research at HINU, the grid and monitoring sites will help with the coordination of different types of research or classroom projects ranging from cultural to technical scientific studies. This research project will help further the understanding and assessment of the ecology and long-term change in the Haskell wetlands, and can be linked with ecological monitoring networks nationwide.
104. Distribution of Porcine Endogenous Retrovirus (PERV) variants in Domestic and Feral Pigs
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Xenotransplantation is considered an alternative to allotransplantation to relieve the current shortage of human organs. Due to their similar size and physiology, pig organs are of interest for this purpose. Endogenous retroviruses are result of integration of retroviral genomes into the genome of infected germ cells as DNA copies (proviruses), which are then carried in all cells of the offspring of the organism. Porcine Endogenous Retroviruses (PERVs) are of concern because they are found in pig organs and tissue that might be used for xenotransplantation. PERV proviruses, already incorporated into the pig’s genome, can be induced to replicate and recombine in pigs, and have been shown to infect human cells in vitro. There are three classes of PERVs: PERV-A, PERV-B, and PERV-C. PERV-A and PERV-B can infect human cells in vitro and can recombine with PERV-C, resulting in a recombinant virus with a higher rate of replication in pig and human cell lines. In this study, a PCR based analysis of 50 domestic and 35 feral pigs was carried out to study the distribution of PERVs A, B, and C. PERV-A and PERV-B were universal in both domestic and feral pigs. The feral varieties of pigs displayed a higher frequency of 85.67% of PERV-C compared to 42.00% in domestic pigs. However, comparative study of presence of PERVs A, B, and C in different breeds of domestic pigs shows there is variation in distribution among breeds, and among individuals of same breeds.

105. Effects of Mechanotransduction on Nuclear Morphology and Chromatin Organization
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Recent advancements in the field of mechanogenomics have led to the discovery of the nucleus as a mechanoresponsive organelle that responds independently of the cell body and cytoplasm1. As a mechanoresponsive organelle, nuclear morphology and chromatin organization are regulated in part by the mechanical stimuli within the cellular environment. The purpose of this research is to develop an understanding of the mechanism by which mechanotransduction affects the regulation of genome structure in endothelial cells found in the cardiovascular system. Understanding how fluid shear stress affects genetic expression in vascular cells is important in developing a basis for how disease states are formed and how to counteract them. It is hypothesized that cells will retain conformational changes that have been caused by mechanical forces when these forces have caused a change in chromatin organization. In this study, Human Microvascular Endothelial cells (HMEC-1) were cultured in ibidi flow chambers and exposed to laminar and oscillatory fluid shear stress for a period of 24 hours. Cell viability and nuclear morphology were analyzed to develop an understanding of the effects of different mechanical stresses on cell structure. Cells under laminar fluid shear stress exhibited morphological differences compared to cells under static conditions, while there was no statistical difference in cell viability between the treatments. Nuclear shape was analyzed using Image J image processing software. Following the study of nuclear morphology, chromatin organization will be analyzed using Hi-C and RNA-Sequencing techniques.


106. Macroglobulin complement-related is required for Drosophila egg elongation
Haifa Alhadyian and Robert Ward
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Morphogenesis describes a development process by which cells change shape, size, and position during an animal formation. Dysregulation of any of these processes can lead to severe developmental defects and embryonic lethality. We identified Macroglobulin complement-related (Mcr) in a genetic screen of mutations that are required for imaginal disc morphogenesis in Drosophila. We determined that Mcr is a core component of epithelial septate junctions (SJs), which are analogous to the vertebrate tight junctions. Mutations in Mcr are embryonic lethal and cause developmental defects that occur prior to the establishment of the SJ. These data suggest a role of Mcr in morphogenesis that is independent of its role in the occluding junction. Here, we are investigating the role of Mcr in morphogenesis using Drosophila egg elongation as a model system. First, we determined that Mcr is expressed in the somatic cells also called follicle cells (FCs). Next, we used stage-specific knock-down experiments using RNAi to examine the function of Mcr in the FCs and its contribution to egg elongation. We find that reducing Mcr expression early on or late in oogenesis results in mature eggs that failed to elongate. By examining the morphology of the FCs of late stage eggs, we find that the membrane of Mcr knock-down FCs breaks apart, resulting in multi-nucleated cells with irregular cell shapes. Also, Mcr knock-down FCs of stage 14 eggs have reduced levels of actin filaments and FSP-integrin at the basal sides. These data indicate a possible role for Mcr in follicular epithelium maintenance.

107. Combination Therapy of Prostate Cancer: PARP Inhibitor Synergizes the Therapeutic Efficacy of Doxorubicin
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Men are most susceptible to prostate cancer in the United States. The general treatment options include surgery, hormone therapy, chemotherapy, and radiation therapy. But in recent days, the nanoformulation have shown promising applications in overcoming the drawbacks of the currently available treatment options. To complement, we tried to enhance the capability of the nanoparticle formulation by loading them with a novel drug combination for the treatment of prostate cancer. Herein, we synthesized folate conjugated iron oxide nanoparticles encapsulated with doxorubicin and olaparib for imaging and targeted treatment of prostate cancer. IONPs coated with polyacrylic acid will be synthesized with water-based precipitation method, followed by functionalization with folate using “click” chemistry. Briefly, the IONPs will be first propargylated using EDC/NHS bioconjugation chemistry followed by Cui catalyzed “click” chemistry using azide-functionalized folic acid. Next, doxorubicin and olaparib will be co-encapsulated in the IONPs using the solvent diffusion method. The resulting therapeutic nanomedicines will be characterized by measuring size, zeta potential, and UV/fluorescence emission and absorbance. The two drug being used together to explore if the synergistic effect they normally have will still be in effect while they are encapsulated in nanoparticles. The cytotoxicity will be explored through MTT assay, and cell uptake studies. The nature of the cell death will be observed through apoptosis, ROS, and comet assay studies. Finally, the antimetastatic potential of the therapeutic nanoparticles will be studied via migration assay and reported.
108. Expression of mutant human tau protein drives synaptic loss in Caenorhabditis elegans

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Tau aggregation is associated with neurodegenerative diseases, and mutations in human tau have been linked to several disorders, including FTDP-17, with varying phenotypic effects. How tau aggregation results in altered neuronal function is controversial and poorly understood, given the difficulties associated with modeling tau aggregation and neurodegeneration in animal models. To address this problem, we have generated multiple C. elegans lines of transgenic animals expressing different variants of the longest isoform of human tau (htau40) to better understand how aggregation might cause subsequent downstream events leading to increased toxicity. We have produced multiple transgenic lines expressing variants of human tau protein in neurons to compare their effects on neuronal function. When broadly expressed throughout the nervous system mutated htau40 caused decreased lifespan, as well as a decreased locomotor capability as they age. Decreased lifespan, along with cognitive and motor defects, are associated with human tauopathies, suggesting our model is reproducing consequences relevant to human disorders. When we selectively express mutated htau40 in the GABAergic motorneurons, we find an age-associated decrease in the number of GABAergic motor neuron synapses, importantly, in the absence of other indicators of neuronal degeneration, i.e. axons and cell bodies were still intact. We have also begun testing mutations in C. elegans orthologs of known Alzheimer’s risk factors and found that loss of the LAR-like receptor tyrosine phosphatase enhances the synaptic loss due to mutations in htau40. Together, these results indicate the utility of our model to understand how tau aggregation impacts neuronal homeostasis and function.

109. Constitutive Musashi-1 (Msi-1) expression alters growth and intestinal epithelial cell characteristics in mice

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Tightly regulated spatial and temporal gene expression is essential for normal tissue development. RNA-binding proteins (RBPs) provide crucial post-transcriptional gene regulation during development by modulating RNA metabolic processes, such as RNA splicing, transport, translation, and stability. One such RBP is Musashi-1 (Msi-1), which displays altered expression in many cancers, including colorectal, brain and ovarian cancers. The most characterized role of Msi-1 is in translational regulation via binding to the 3'-untranslated region of target mRNAs and repressing translation initiation. Since known targets of Msi-1, including p21, Adenomatous polyposis coli, and Numb, are involved in modulating cell proliferation, differentiation, and death, we hypothesize that this post-transcriptional control of expression provided by Msi-1 might be essential for regulation of intestinal development and homeostasis. To test this, we generated a mouse model that allowed regulated and inducible Msi-1 overexpression. Here we provide evidence that the ubiquitous overexpression of Msi-1 in young transgenic mice results in decreased body weight and in premature death. These Msi-1-overexpressing mice (Msi-1+1+OE) failed to maintain normal organ to body proportions; specifically, their colons displayed altered proliferation, differentiation, and intestinal development. Further analysis revealed that the intestinal epithelial tissue of Msi-1+1+OE mice displayed altered proliferation, differentiation, and morphology. Current studies are aimed at further elucidating mechanisms through which Msi-1 controls intestinal tissue development.

110. Loss of REST in Breast Cancer.

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Breast cancer is the most common cancer in women, and is highly heterogenous making early detection and treatment difficult. A subset of breast cancers lack REST protein (repressor element 1 silencing transcription factor) but not mRNA. This loss of REST is seen in ~20% of human breast cancers, and is associated with a poor prognosis and increased aggregation of the tumor. Despite the prevalence, little is known about the role of REST in the molecular pathogenesis of breast cancer. REST normally functions in the periphery to silence neuronal gene expression and has also been shown to be a tumor suppressor in epithelial cells. The loss of REST leads to its target genes being dysregulated, and can cause aberrant activation of signaling pathways that promote tumorigenesis. This has been seen in other diseases including colon cancer, small cell lung cancer, and uterine fibroids. Here, we show that loss of REST in MCF7 cells leads to similar dysregulation that is seen in breast cancer patient samples. We show that many of the genes overexpressed in breast cancer are direct targets of REST. Furthermore, using RNA sequencing data, we show that steroid hormones can also affect REST target gene expression when REST is lost. Our work in uterine fibroid pathogenesis has shown a large number of REST target genes are also targets of progesterone receptor. Loss of REST in these diseases may contribute to changes in hormone sensitivity. Our findings show loss of REST contributes to the pathogenesis of certain breast cancer subtypes.

111. Development of Functional Magnetic Relaxation Nanosensors for the Investigation of Zika Binding and Fusion Mechanism

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The prevalence and the rampant effect of zika virus (ZIKV) is known to everyone. Due to the high risks’ it possesses to human life, it is important to study the cycle of virus in detail. Binding and fusion of a virus with the host membrane is the first and one of the most crucial steps in the life cycle of all enveloped viruses. Therefore, the study of binding and fusion is necessary for development of the antiviral drugs, which can help to block the entry of virus. Although, many studies have been conducted to detect the presence of ZIKV after the completion of fusion, very few studies have been conducted to see the binding and fusion distinctly. Herein, we aim to develop a nanosensor platform which can help to evaluate fusion and binding distinctively via magnetic relaxation (MR) technique. This will be done by developing new magnetic relaxation nanosensors (MRnS)-conjugated with Reporter Virus Particle (RVP), which will be tested in normal human epithelial cells (Vero cells) for the binding and fusion of MRnS-RVP. Any change in the MR depicts the binding or fusion of MRnS-RVP with the membrane of vero cell. Furthermore, we will also conduct neutralization studies with different neutralizing antibodies in order to validate our platform. Together, we propose a robust MR nanosensor for studying ZIKV binding and fusion mechanism, and the results of this project will be discussed in this presentation.
Poster Presentation Abstracts

112. Investigating a role for septate junction proteins in cell polarity during Drosophila dorsal closure

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Polarized epithelia establish distinct compartments within the body of multicellular organisms and regulate paracellular transport of solutes. This diffusion barrier function is executed by septate junctions (SJs) in invertebrate epithelia. Recent studies in our lab demonstrated that core SJ genes are required for Drosophila embryonic developmental events such as head involution and dorsal closure (DC) that occur prior to the formation of a mature SJ. However, the mechanistic role of SJ genes during morphogenesis is unknown. We hypothesize that SJ proteins regulate aspects of cell polarity. SJ genes act redundant to maintain apical/basal polarity in the epidermis of late stage embryos. Whether SJ genes are independently required to maintain apical/basal polarity and if mild alterations in polarity contribute to defects in SJ mutants is unclear. To address this, we are using DC as a model system. DC occurs during mid-embryogenesis and involves elongation and migration of the contralateral epidermal cells to seal a dorsal hole. Mutations in core SJ genes are embryonic lethal, with significant DC defects. We are examining fixed tissues in SJ mutant embryos for defects in apical/basal polarity during DC. We are also attempting to rescue DC defects in SJ mutants by subtle alterations in apical/basal polarity. In addition, epidermal elongation during DC requires planar polarized expression of various proteins. Whether SJ genes are required for any aspect of planar polarization during mid-embryogenesis is unknown. To address this, we are analyzing fixed tissue of SJ mutant embryos for defects in epidermal elongation and planar polarization during DC.

113. Migration of induced pluripotent stem cells and their derivatives in 3D matrices

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Spinal cord injuries (SCIs) are debilitating conditions for which no effective treatment or cure currently exists. Stem cell replacement therapies may offer hope to people suffering with SCIs. A relatively new type of stem cell, induced pluripotent stem cells (iPSCs) originate from adult somatic cells and bypass the ethical and immunological concerns of other stem cell lines. One approach to treating SCI is to combine stem cells such as iPSCs with a biomaterial for use in an afflicted organism. To achieve recovery of function below the site of injury, the choice of biomaterial is critical as it must allow for integration of transplanted cells with host cells. In this study, Matrigel was selected as a possible candidate for 3D culturing of cells. iPSCs were cultured and subsequently differentiated into neural stem cells (NSCs) or oligodendrocyte precursor cells (OPCs). These iPSCs and their derivatives were separately seeded into culture wells with host cells. In this study, Matrigel was selected as a possible candidate for 3D culturing of cells. iPSCs were cultured and subsequently differentiated into neural stem cells (NSCs) or oligodendrocyte precursor cells (OPCs).These iPSCs and their derivatives were separately seeded into culture wells with Matrigel. All cell types were able to survive and grow within the Matrigel. The migration of each cell type in the Matrigel was recorded at regular intervals using a time-lapse microscope and analyzed using NIH Image J software. We demonstrated the ability of these cells to move within the gel makes. The study indicates that it possible that the cells will be able to integrate with host neural cells in vivo and bridge the transected portion of the spinal cord. Such a method serves as a model for treatment of human SCI patients.

114. An RNA binding protein, HuR, in pancreatic cancer EMT, metastasis, and CSC

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Epithelial-mesenchymal transition (EMT) contributes importantly to cancer cell metastasis and formation of cancer stem cells (CSCs). An RNA-binding protein, HuR, regulates gene expressions involved in tumor cell proliferation, metastasis, anti-apoptosis, and drug resistance. Here we aim to investigate the role of HuR in regulating pancreatic cancer EMT and CSC, and develop novel HuR inhibitor to suppress pancreatic cancer EMT and CSCs. We demonstrated that HuR knockdown inhibited migration and invasion of pancreatic cancer cells Panc-1 and Mia PaCa-2. Expressions of signature EMT genes in both cells were altered: the epithelial marker Claudin-1 was significantly upregulated and the EMT enhancing transcription factor Snail was significantly decreased. In contrast, HuR re-expression in HuR knock-out cells significantly enhanced the cell migration. Tumor spheroids were significantly inhibited with siHuR silencing, indicating the inhibition of CSC formation. RNIP assay and reporter assay showed HuR bound to the 3'UTR of Snail by targeting the AU rich element (ARE). We also discovered that a novel compound KH-3 inhibited the proliferation of HuR-high pancreatic cancer cell and had minimal toxicity to the HuR-Low normal pancreas ductal epithelial. KH-3 treatment mimicked the HuR knockdown effects in inhibiting cell migration, EMT, and spheroids formation. In addition, KH-3 inhibited pancreatic cancer progression and metastasis in vivo. In summary, our project demonstrated that HuR directly binds to the 3'UTR of Snail by targeting the AREs, thus the inhibition of HuR inhibits the EMT and CSCs process. KH-3 as a novel HuR inhibitor inhibited pancreatic cancer EMT and CSCs.

115. Combination Therapy of TNBC: The Hsp90 Inhibitor Synergizes the Therapeutic Efficacy of CT20p Peptide

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Triple negative breast cancer (TNBC) is considered to be the most aggressive type of breast cancer. The available treatment options for TNBC including hormonal therapy, chemotherapy and radiation therapy are abortive and have lot of side effects. Herein, we have developed an easy-to-formulate targeted drug delivery system using functionalized iron oxide nanoparticles, which helps in curtailing the side effects and combating cancer. In our design, we have formulated ICAM-1 antibody functionalized magnetic nanoparticles (MnPs) for targeting TNBC and MDA-MB-231 cells were used in these experiments. A small peptide, CT20p, which inhibit a protein-folding complex, is commercially available in many cancers, called Chaperonin-Containing TCP-1 (CCT). Gedunin, an Hsp90 inhibitor, is also incorporated to synergize the therapeutic efficacy of CT20p peptide. Experiments indicated that the novel combination of CT20p and gedunin induced reactive oxygen species (ROS) in MDA-MB-231, leading to cell death. To further assess the synergistic therapeutic effect, cell viability assays were performed and more than 85% cells were found to be dead within 48 h of treatment. Additional experiments indicated for the apoptotic cell death, where the migratory nature of TNBC was inhibited as a result of this combination therapy. These results will be summarized and highlighted in this presentation.
116. Ulcerative Colitis Patients Display Increased Levels of APC in Goblet Cells

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While best known as an antagonist of the Wnt signaling pathway, other functions of the tumor suppressor gene Adenomatous Polyposis Coli (APC) have yet to be fully investigated. We have found that there are increased levels of APC in a subset of intestinal Goblet cells of both mice and humans. Goblet cells produce and secrete mucins, which serve to protect the intestinal epithelium from pathogens and mechanical damage. The number of intestinal Goblet cells showing high levels of APC increased in mice treated with Dextran Sulfate Sodium (DSS) to induce colitis as well as in human inflammatory bowel disease patients. The goblet cells in these ulcerative colitis colon crypts also have non-O-glycosylated Muc2 present, indicating perturbation of normal goblet cell function. Muc2 is a highly glycosylated 5179 amino acid protein secreted exclusively by Goblet cells and is predominantly responsible for making up the two intestinal mucus layers. We have found that APC regulates Muc2 expression in colon cancer cells. This may give more insight into the production of Muc2, and how its reduction is linked to spontaneous colitis, which can increase the risk of colon cancer. Muc2 production has also been shown to be tightly controlled, as overproduction or accumulation could lead to ER stress and result in the activation of the unfolded protein response (UPR). Here we show that high levels of APC in patients with UC correlate with the accumulation of non-O-glycosylated Muc2 and ER stress.

117. pwrEWAS: A user-friendly tool for comprehensive power estimation for epigenome wide association studies (EWAS)

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Abstract
When designing an epigenome-wide association study (EWAS) to investigate the relationship between DNA methylation (DNAm) and some exposure(s) or phenotype(s), it is critically important to assess the sample size needed to detect a hypothesized difference with adequate statistical power. However, the complex and nuanced nature of DNAm data makes direct assessment of statistical power challenging. To circumvent these challenges and to address the outstanding need for a user-friendly interface for EWAS power evaluation, we have developed pwrEWAS. The current implementation of pwrEWAS accommodates power estimation for two-group comparisons of DNAm (e.g. case vs control, exposed vs non-exposed, etc.), where methylation assessment is carried out using the Illumina Human Methylation BeadChip technology. Power is calculated using a semi-parametric simulation-based approach in which DNAm data is randomly generated from beta-distributions using CpG-specific means and variances estimated from one of several different existing DNAm data sets, chosen to cover the most common tissue-types used in EWAS. pwrEWAS reports the marginal power, marginal type I error rate, marginal FDR, and false discovery cost (FDC). We demonstrate how pwrEWAS can be applied in practice using a hypothetical EWAS. In addition, we report its computational efficiency across a variety of user settings. Both under- and overpowered studies unnecessarily deplete resources and even risk failure of a study. With pwrEWAS, we provide a user-friendly tool to help researchers circumvent these risks and to assist in the design and planning of EWAS.

118. SPECC1L regulates oral epithelial adhesion downstream of IRF6 in palatogenesis

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Clefts of the lip and/or palate (CL/P) is one of the most common craniofacial birth defects. SPECC1L variants identified in patients with syndromic and nonsyndromic CL/P suggest the gene plays a primary role in palate development. To elucidate the role of SPECC1L in palatogenesis, we generated null and hypomorphic Specc1l-deficient mouse alleles. Null mutants are lethal at embryonic day 9.5 (E9.5) with defective neural tube closure and cranial neural crest cell delamination. Specc1l−/−/− compound mutants exhibit delayed palate elevation and fusion in >60% of embryos at E14.5. Importantly, mutant embryos carrying the human T397P mutation showed 100% cleft palate, confirming a specific role for SPECC1L in palatogenesis. At the cellular level, we observed transient adhesions between oral epithelial periderm layers of the palate and tongue or mandible. Normal non-adhesive periderm is devoid of cell adhesion molecules at its apical surface. We observed increased actin filaments and ectopic apical distribution of adherens junction markers in mutant palate periderm. Heterozygous or hypomorphic mouse mutants for known clefting genes, Irf6, Grhl3 and Arhgap29, also show periderm defects and ectopic oral adhesions. Accordingly, we found that SPECC1L expression in the oral epithelium requires IRF6. Together, our data show that SPECC1L acts downstream of IRF6 to regulate embryonic palate epithelial integrity, and that delayed palate elevation may represent a predisposing factor in this complex disease. Our results extend the understanding of human CL/P etiology by placing SPECC1L within the IRF6/GRHL3 pathway, which plays a major role in lip and palate development.

119. In-vivo like Fibroblastic Reticular Cell Growth Determined by 3D Scaffold Material

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Cellular growth, morphology, and proliferation are all largely influenced by their environment. Three-dimensional scaffolds provide an opportunity to more closely mimic the natural environment of cells compared to traditional two dimensional cell culture. Stromal cells, such as fibroblastic reticular cells (FRCs), play an essential role in remodeling the extracellular matrix (ECM) and stimulating cellular response in lymph nodes. By seeding these cells into scaffolds, we hypothesized that they would be able to grow and eventually simulate a native ECM environment. Different materials and microarchitectures were used in scaffolding for FRC culture. Material properties such as stiffness, surface chemistry, and pore structure were shown to play a role in determining cellular morphology and behavior. Collagen appeared to play a vital role in inducing fibroblastic reticular cell branching and in-vivo mimetic morphology. Additionally, FRC cell functionality was observed in all collagen containing scaffolds. These results will allow us to eventually develop a biomimetic 3D microenvironment for immune tissue culture.
120. The Extracellular Matrix of Gonium is Important for Multicellularity

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The evolution of multicellular organisms from unicellular ancestors is considered a major transition. Unicellular Chlamydomonas and its close multicellular relatives in the order Volvocales are an excellent model system for studying the genetic basis of this transition because they exhibit a stepwise acquisition of biological complexity. Surprisingly, the genomes of Chlamydomonas and the Volvocales are highly similar, which suggests that co-option of small sets of genes underlies the evolution of multicellularity. To identify genes important for that transition we performed a forward genetic screen in the undifferentiated multicellular alga Gonium pectorale. From this screen, we isolated two unicellular mutants, uc-1C7 and uc-1H7. Both mutants exhibit defects in assembly of their extracellular matrix (ECM), pointing at the possibility that it has been co- opted for cell-cell adhesion, a key feature of multicellularity. We followed up this analysis by sequencing the genome and transcriptome as well as performing mass spectrometry of proteins in the ECM of uc-1C7. We found that uc-1C7 is lacking critical components of its ECM, suggesting the genetic defect in uc-1C7 affects its assembly in a global fashion. We hypothesize that the development of an extracellular matrix from an ancestral cell wall is necessary for the evolution of colonial organisms in the Volvocales.

121. Identification and Characterization of chemical inhibitors of the Legionella pneumophila metaeffector Lpg2505

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Legionella pneumophila (Lpn) is a facultative intracellular pathogen found abundantly in freshwater environments where it can parasitize and replicate within amoeba. Lpn colonization of mammal freshwater environments has led to accidental human pathogenicity upon aerosolization and successive inhalation. Lpn infects and replicates within pulmonary immune phagocytes -alveolar macrophages- and the resulting infection causes a severe inflammatory pneumonia called Legionnaires’ Disease. In order to infect and replicate within eukaryotic phagocytes, Lpn translocates more than 300 effector and metaeffector proteins into the host cell using a Dot/Icm type IVB secretion system (T4SS). Metaeffectors are effectors proteins that regulate other Lpn effectors within the host cell. We have determined that the metaeffector, Lpg2505, interacts directly with its cognate effector, SidI, in vitro. In the absence of Lpg2505, SidI is detrimental to Lpn intracellular replication. Lpg2505 promotes Lpn replication by regulating SidI, likely through direct protein-protein interaction. Our goal is to identify small molecules that will inhibit regulation of SidI by Lpg2505 as a means to attenuate Lpn intracellular replication. Our efforts will include characterization of the Lpg2505-SidI interaction through crystallization of the SidI-Lpg2505 complex, which will reveal potential small-molecule interaction site targets. We will also screen for inhibitors of Lpg2505-mediated suppression of SidI toxicity in a yeast expression system. This work will lead to novel therapeutics to limit Lpn-mediated disease through inhibition of an essential metaeffectors.

122. Neuron “GPS”: The Canonical Wnt Pathway in Motor Axon Guidance

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The human brain has millions of motor neurons. During development, each of these neurons will migrate and extend axons to establish connections with muscles via synapses. These connections are critical for ensuring proper communication and function. How motor neurons are able to guide their axons is still largely unknown. We utilize the nematode Caenorhabditis elegans (C. elegans) to study 3 of their 302 neurons to map the signals which instruct neurons when to stop growing.

In C. elegans, the growth of the GABAergic D-type motor neurons is highly influenced by the canonical Wnt signaling pathway. Specifically, the most posterior D-type neurons, DD6 and VD13, utilize the canonical Wnt signaling pathway as a unique growth and termination cue. We sought to further categorize how the pathway was involved in growth, and began to search for other factors involved in this process. However, due to the highly similar nature of the D-type neurons, we could not individually view each neuron. Our lab recently obtained a transgene which allows us to view exactly how VD13 is formed.

Here, we show that VD13 is most often C-shaped. Further, in animals mutant for disheveled and the Hox gene egl-5, transgenic expression is lost in VD13. In addition, the morphology of VD13 is aberrant in Wnt signaling mutants. We are working to fully categorize VD13 in canonical Wnt signaling mutant backgrounds. We are also using this marker in a mutagenesis screen to discover additional genes involved in directing the growth of this motor neuron axon.

123. Online Preconcentration of High-Salt Samples Using Pressure-Assisted Field-Amplified Sample Injection in Flow-Gated Capillary Electrophoresis

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Abstract

Capillary electrophoresis (CE) is a powerful separation technique; however, its detection sensitivity often fails to satisfy real-world applications due to the small amount of samples injected for analysis. To enhance the detection capability, we report a new technique developed for flow-gated CE to directly analyze high-salt samples such as cerebrospinal fluid (CSF). First, the high-salt sample was fluorogenically derivatized with 2,3-Naphthalenedicarboxaldehyde (NDA) in the presence of cyanide. Then, a sample plug was hydrodynamically injected by vacuum on the outlet side of the capillary. Third, a reversed-voltage was applied to conduct field-amplified sample injection while maintaining a counter vacuum to elongate the injection time during the pushback. Finally, a normal-polarity voltage was applied for separations. During the injection procedure, the sample solution was continuously supplied to the cross section of the flow gate by a syringe pump, which ensured the inlet of the capillary was immersed in sample; and the sample preconcentration might rely on the electrokinetic supercharging principle. Enhancement factors of 50-100 folds at optimal conditions were obtained for a series of amino acid neurotransmitters including γ-aminobutyric acid (GABA), serine, glutamate and aspartate, and the limits of detection were lowered down to pico-molar range. In addition, this method was applied to the determinations of neurotransmitter levels in CSF by using one-point standard addition method enabled by alternate injections of the two samples; the detection limit was lower down to the pico-molar range. The technique is expected to be useful for analyzing other high-salt containing samples with flow-gated CE.
124. A Bayesian Framework for Assessing Concordance in Microbial Abundance

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Large-scale epidemiologic studies have used high-throughput sequencing of bacterial DNA to identify associations between the microbiome and risk of several human diseases. However, analysis of microbial abundance is complicated and currently there is no general test for concordance between groups (e.g., different tissues) with respect to other grouping variables (e.g., cancer subtype). Here we extend a previous approach that modeled relative abundance via Zero-Inflated Mixed Beta Regression into the Bayesian framework and formally test concordance via posterior distributions of group means. We demonstrate the viability of our method using both simulations and real data collected from different body sites in patients with pancreatic cancer. Our approach successfully identifies specific microbes that exhibit concordant patterns between body sites. The identification of such concordant patterns could be used to generate non-invasive estimations of microbial composition by using the microbial composition in easily accessible tissues to predict those in tissues only accessible via surgery.

125. Optimization of a lead biosensor to test environmental samples in an in vitro and in vivo system

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Pittsburg State University

Lead contamination of water and soil poses a serious health risks to humans. Gas chromatography and atomic absorption spectrometry are typically used to detect lead in environmental (water and soil) samples. These detection methods require costly equipment and expertise. Here, we outline plans to optimize a lead biosensor generated as part of the international genetically engineered machine (iGEM) program in order to facilitate development of a cheaper and easier lead detection method. We will be working with a biosensor within the bacteria Escherichia coli (E. coli) which will allow for detection of lead by adding a soil or water sample into a growing culture of bacterium, making it a cost-effective method that takes minimal training. The biosensor consists of a plasmid containing a constitutively expressed repressor protein which binds to the promoter/operator unit from the chromosomal lead operon of Cupriavidus metallidurans. If lead is present, it binds to and inactivates the repressor, allowing for transcription of green fluorescence protein (GFP). Initial tests indicate that the amount of fluorescence produced by the biosensor is similar for all lead concentrations however, we hope to fine-tune the system to allow for dose dependent detection. We also plan to use the biosensor DNA to set up an in vitro cell free protein synthesis (CFPS) system for detection. We will visualize our results through use of a UV lamp, flow cytometry, Western blotting, or fluorescence microscopy.

126. Natural Products Against E.S.K.A.P.E. Pathogens Isolated from Soil microbes

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Background and Objective: The E.S.K.A.P.E. pathogens (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter species) are a group of bacteria that have developed multiple antibiotic resistances (Boucher et al., 2009). This presents a worldwide health concern as antibiotics that normally treat these infections are now ineffective. By testing bacteria obtained from soil samples against E.S.K.A.P.E. pathogen relatives, the goal is to find new natural products from those isolates that are effective against the relatives and some pathogens themselves.

Methods: Microbes were isolated from soil collected around Hays, KS. Seventeen isolates were selected from each site based on morphological differences to test against the E.S.K.A.P.E. relatives for inhibitory effects. Isolates possessing this ability were further tested with a combination of staining techniques, biochemical testing, and genetic analysis for bacterial identification and characterization.

Results: The isolates were Gram positive and negative organisms with a variety of genera found. The isolates were tested against the E.S.K.A.P.E. pathogens: Methicillin Resistant - Staphylococcus aureus, both with and without the LAC-p03 Operon, and Acinetobacter baumannii with varying levels of inhibition displayed. A total of 14 isolates possessed this ability.

Discussion and Conclusion: Potential natural products were extracted from those isolates that inhibited the aforementioned pathogens. Purification, and synthesis of these natural products will be conducted for product(s) identification and further analysis performed to identify the compound(s) present.

127. Detection of inversely enriched pathways in PBMC cells in Alzheimer’s disease and cancer

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Challenging the paradigm of studying human diseases in isolation has enormous potential to advance our understanding of human disease and pathogenesis. A growing number epidemiological studies confirm the existence of direct and inverse comorbidities between different Alzheimer’s disease (AD) types and certain cancers. Inverse comorbidities (a lower than expected joint occurrence) such as these provide an ideal opportunity to identify the mechanisms through which protection against both diseases might be conferred and exploited. A number of factors are generally considered to impact comorbidities, such as age, lifetime exposures, and socio-economic factors, but none yet have been found to play a role in cancer and AD comorbidity. Thus, overlapping genetic and molecular mechanisms hold the greatest promise in explaining relationships between these diseases. In this work, we demonstrate that inverse patterns of disrupted gene expression, associated with inverse comorbidity, are detectable in the blood of cancer and Alzheimer’s patients. In concordance with prior work, we found these disruptions to be associated with cellular metabolism. However, this is the first time these changes have been demonstrated to be detectable outside of the disease tissues. This suggests the possibility of blood markers that can identify certain individuals with reduced or increased risk for either disease that may be easily detectable through relatively non-invasive tests.
128. Understanding the characteristics of bacterial isolates obtained from commercial poultry feed using whole genome sequencing approach

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In order to reduce pathogen contamination in poultry products identification of overall microbial populations in poultry production processing steps have always been considered an important monitoring tool for assessing sanitizer effectiveness and the corresponding responses of bacteria load levels on poultry carcasses. Presence of pathogens is relatively infrequent, thus gaining a better understand of non-pathogens will help in identification of more representative hygienic organisms. Bacterial isolates recovered from corn-based chicken feed were purified on aerobic plate count agar and 11 morphologically different colonies were selected for whole genome sequencing. The goal of the study was to: 1. Sequence, assemble and annotate the whole genome of these isolates, 2. Compare the protein profile among different strains of the same bacterial species. The isolates were identified as Stenotrophomonas spp. (2), Kosakonia cowanii (3), Enterococcus gallinarum (2), Klebsiella ariilica (2), and Pantoea vagans (2). Whole genome length was calculated as 4.8-5.7 X 10^6 bases; number of rRNA molecules were found to be 8-14; and total protein coding sequences were up to 5500. Genes coding for resistance, stress and toxin were prevalent in all strains. The data obtained from this study would help in identifying characteristics of a hygienic indicator organism in the poultry processing pipeline and thus reinforce application of WGS in food safety.

Keywords: poultry feed, MiSeq sequencing
Category: Food Safety Including Fundamental Understanding of Pathogens (Graduate)

129. Wnt-dependent asymmetric redistribution of the β-catenin-destruction complex

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The intestinal epithelium is highly proliferative and relies on a small population of Wnt-regulated stem cells for its renewal. During division, intestinal stem cells must replenish the stem cell population and produce progenitor cells that eventually differentiate. Asymmetric cell division (ACD) accomplishes this task by generating two daughter cells with different fates. When genes involved in ACD are deregulated or mutated, symmetric divisions arise and have resulted in tumorigenesis in various tissues and organisms. The tumor suppressor, Adenomatous Polyposis Coli (Apc), is mutated in over 80% of colorectal cancers and is a key regulator of Wnt signaling as a core component of the β-catenin destruction complex. Studies have also shown Apc to be involved in asymmetric cell division and proper mitotic spindle orientation. We hypothesize that a localized Wnt source will re-orient an intact β-catenin destruction complex, and that this localization will be disrupted with mutant Apc. Using Wnt3a-conjugated Dynabeads, we have examined the effects of a localized Wnt signal on the localization of the β-catenin destruction complex in human colon cancer cell lines. Preliminary results support our hypothesis that administration of a localized Wnt signal will re-orient the β-catenin destruction complex toward the Wnt source and that complex localization is altered with Apc mutation. Studies will be expanded to determine whether ACD incidence and cell fate outcomes are altered in response to a localized Wnt signal. Results from these studies will provide mechanistic insight into the role of Apc and Wnt signaling in intestinal stem cell maintenance and tumorigenesis.

130. Antimicrobial properties of secondary metabolites from plants in Kansas wetlands

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Antimicrobial resistance is a multifaceted, global problem. To combat drug-resistant pathogens, the array of available antibiotics must be expanded; however, little effort has gone into the discovery of new antimicrobial drugs. Plants defend themselves from pathogenic attacks by producing secondary metabolites, a broad range of compounds including terpenes, phenolics, and nitrogen-containing compounds. These compounds might be utilized as antimicrobial agents. The objective of this research is to survey plants from wetlands in Kansas for antimicrobial properties by using aqueous and ethanolic extracts from different anatomical structures of the plant. Plants found in aquatic regions are of special interest to this research, as they must acclimate to pathogens found above-ground, in soil, and in aquatic biofilms. The extracts were tested with a disc diffusion assay against Gram positive Staphylococcus aureus and Gram negative Escherichia coli. Negative controls consisted of water and ethanol alone, and positive controls of known antibiotics were also used. Zones of inhibition were measured, and minimum inhibitory concentrations (MIC) were determined. This research could assist in developing new antimicrobial drugs, which is especially important with the rise of antibiotic resistance. Additionally, this research could further our understanding of plant-microbe interactions, including ecological and evolutionary applications, both of which remain understudied.

131. RNA Interference of the Unfolded Protein Response in Acyrthosiphon pisum

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Aphid species are distributed worldwide and are persistent agricultural pests. Previous studies have demonstrated the efficacy of dsRNA as a tool to cause selective death in target organisms through RNA interference (RNAi). Branched Amphiphilic Peptide Capsules (BAPCs) are used to complex dsRNA and protect RNA from the aphid digestive system (Balthazor, J. R., et al. 2018). Prior to beginning the study, sequence similarity was evaluated to ensure no undesirable off-target effects would occur. Whole RNA was isolated from adult A. pisum and used to synthesize cDNA. Synthesized cDNA was used as a template to produce dsRNA for use in RNAi studies. dsRNA complementary to PERK mRNA was fed to aphids in three trials with standardized amounts of dsRNA: 0.1µg, 0.001µg, and 0.0001µg, n=200. dsRNA complementary to ATP4 mRNA was fed to aphids in three trials with standardized amounts of dsRNA: 0.1µg, 0.001µg, and 0.0001µg, n=200. The death curves of the treatment aphids were compared to the control group (n=200) via log-rank testing. It is expected that the introduction of dsRNA complementary to the target gene mRNA will cause knockdown of the target and subsequent death of the insect.

Category: Food Safety Including Fundamental Understanding of Pathogens (Graduate)
132. Integrating Multiplatform Data for Improved Prognosis in Cancer Patients.

Duncan Rotich, PhD

Recent advances in technology have generated vast amounts of heterogeneous types of data collected from patients. These data types range from clinical characteristics and patient reported outcomes to high throughput genomic data. Integrating information from multiple sources is important in capturing patient heterogeneity which consequentially can help inform treatment regimens to achieve precision medicine. Currently most studies approach these data independently even though there might exist important relationships which can be revealed through data integration. In this study, a Bayesian approach is adopted to develop a prognostic model which integrates both clinical information and genomic data to identify prognostic biomarkers and predict time to event in cancer patients by utilizing publicly available data from The Cancer Genome Atlas. By incorporating prior information as well as multiple sources of data, Bayesian methods provides increased precision than frequentist approaches in regard to model prediction. We will evaluate model performance in comparison to standard methods that examines data independently or integrate it using non-Bayesian methods.

133. Biomolecular Structure-Function studies of the central binding region of tumor suppressor Adenomatous Polyposis Coli

Aaron J. Rudeen, Minli Xing, Justin T. Douglas, and Kristi L. Neufeld

Mutation of Adenomatous polyposis coli (APC) is widely regarded as an initiating event in colon carcinogenesis, and is found in up to 80% of colorectal carcinomas. APC is a large (2,843 aa), multi-domain scaffolding protein that has been shown to functionally interact with many protein partners. The best-characterized role of APC is in a protein complex that negatively regulates Wnt signaling via β-catenin destruction. This destruction is mediated by β-catenin binding to centrally located 15- and 20-amino acid (aa) repeat regions of APC. Most carcinomas with mutant APC express a truncated APC protein which retains the 15-aa repeat region. It remains to be determined why the 15-aa repeat region is so strongly selected as beneficial to cancer cells. However, this apparent selection raises the potential for unique interactions between this APC region and critical binding partners to serve as targets for future development of anti-cancer strategies.

The large extent of predicted disorder within the 15- and 20-aa repeat regions of APC has deferred structural studies, as illustrated by the dearth of resolved secondary features and precise binding mechanisms. Establishing whether this central domain of APC is a fully intrinsically disordered protein or possesses regions of stability will shape our understanding of the function of this critical protein-binding region. Here, we have utilized solution NMR, an attractive method to begin to probe the structure of the central region of APC and uncover the specific residues that interact noncovalently as a prelude to the development of molecular probe agonist/antagonists.

134. A novel compound inhibits pancreatic cancer cell invasion, tumor sphere formation and in vivo tumor growth in mice by suppressing EMT

Tao Wang, Ruochen Dong, Ping Chen, Michael J Baltezor, Scott Weir, Qi Chen

Epithelial to Mesenchymal Transition (EMT) has been proposed to contribute importantly to metastasis, cancer stem cell (CSC) generation and drug resistance in many cancers. Targeting EMT may be promising to benefit cancer treatment. Our previous high throughput screening study has identified a potential EMT inhibitor (namely, C150) in pancreatic cancer cells. Here, we sought to investigate the activities of C150 in inhibiting pancreatic cancer cell invasion, CSCs and tumor growth in mice and its mechanisms of EMT inhibition. C150 exhibited a well-separated cytotoxicity between pancreatic cancer cells and normal cancerous cells, with IC50 values of 1–2.5 μM in multiple pancreatic cancer cell lines and >25 μM in a non-cancerous pancreatic epithelial cell line (hTERT-HPNE). C150 significantly inhibited Panc-1 cell migration and invasion in 3-dimension (3D) cell invasion model, in wound scratching assay and in Boyden Chamber trans-well migration-invasion assay. Pancreatic tumor sphere formation was strongly inhibited by C150 at concentrations as low as 0.4 μM in Panc-1 cell and 0.05 μM in MiaPaCa-2 cells, indicating preferable inhibition of pancreatic cancer stem cells. In the orthotopic pancreatic cancer mouse model, C150 significantly reduced tumor growth at the dose of 15mg/kg, 3x weekly IP injection. C150 decreased the mesenchymal marker N-cadherin while increased epithelial markers of ZO-1 and Claudin-1. Further studies revealed that C150 significantly reduced total and nuclear levels of Snail and β-Catenin, two important transcription factors that contribute to EMT and cancer stem cells in pancreatic cancer. This early evidence suggests that C150 potentially inhibits pancreatic cancer stem cell and invasion by inhibiting EMT though Snail and β-Catenin down-regulation.

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135. CuCo2S4 Nanoballs: A solution to clean hydrogen generation and urea elimination from wastewater

Camila Zeguine, Fangzhou Wang, Xianglin Li, Pawan K. Kahol, Ram K. Gupta

Abstract:
Urea is an abundant waste generated in agricultural land, and it is present in industrial and human wastewater as well. Urea is produced from natural gas or can be synthesized from ammonia. When in contact with atmosphere and groundwater, the urea breaks down into ammonia and nitrate, causing health and environmental problems. Owing the crisis energy and the potential degradation of the environment, it is extremely important to produce clean energy helping the world do not be destroyed. Urea oxidation reaction (UOR) is an alternative in the electrochemical generation of hydrogen due to both lower electrochemical potential, decreasing the costs for the cells and plugging urea from wastewater during the hydrogen fuel production. Copper cobalt sulfide grown on nickel foam was synthesized as an electrocatalyst for urea oxidation to generate hydrogen as a green fuel. The electroactivity of CuCo2S4 towards the urea oxidation was evaluated using electrochemical measurements. CuCo2S4 nanoballs showed an outstanding activity for electrooxidation of urea in alkaline conditions exhibiting 60% less energy consumption for hydrogen production in presence of urea. In addition, the electrode presents very stable performance, up to 16 h, ensuring its durability for energy applications. By performing the UOR using the CuCo2S4 nanoballs electrode, it is possible to produce H2 with less energy consumption and eliminate the urea from groundwater systems and environment that are harmful.
ApoE2 promotes neuronal health and wellbeing via modulation of glycolytic pathways

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Continued clinical failures in the search for a successful treatment of Alzheimer’s disease (AD) raise questions about the validity of currently-focused therapeutic targets, underscoring the importance of a novel concept that emphasizes less on the pathological manifestation of the disease but more on the neuroprotective mechanism that promotes brain resilience against the onset of AD. We have recently demonstrated that human ApoE genetic isoforms (ApoE2, ApoE3, ApoE4) differentially modulate brain energy metabolism with the ApoE2 brain exhibiting the most robust while the ApoE4 brain displays the most deficient profile. Studies by other groups have also revealed the evidence in support of the association between glycolytic deficit and high brain glucose with increased accumulation of plaques and tangles in the brains of Alzheimer’s patients. These findings laid the foundation for our follow-up study of the role of human ApoE isoforms in the regulation of glycolytic metabolism in the brain and how it may contribute to the development of AD. In the present study, using two different cell models, we show that hexokinase, the enzyme that catalyzes the initial and irreversible step in glycolysis is significantly affected by ApoE isoforms, and the distinct glycolytic status is directly correlated to the overall health and wellbeing of the three ApoE isoforms-expressing cells. These data indicate that human ApoE isoforms differentially modulate neuronal glycolysis, in large part via regulation of hexokinase, which markedly influences neuronal metabolic activity and health status. The ApoE-mediated glycolytic robustness may serve as a mechanistic rationale for its neuroprotective role and consequently provides a novel therapeutic approach in the prevention and early intervention of AD.

Super-resolution microscopy analysis of neuromuscular junction reveals degeneration of active zones in ALS model mice

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Abstract: Presynaptic active zones play an essential role as synaptic vesicle release sites for synaptic transmission. We used stimulated emission depletion (STED) microscopy to reveal the nanoscale architecture of active zones and ultrastructure of the synaptic terminal at sub-diffraction limited resolution. We identified an unexpected finding of non-overlapping localization of the active zone proteins Bassoon and Piccolo in mouse neuromuscular junctions (NMJs). Piccolo puncta sandwiched a Bassoon punctum in a side-by-side pattern, which could not be resolved using conventional confocal microscopy. P/Q-type voltage-gated calcium channel (VGCC) puncta colocalized with Bassoon puncta. We aimed to reveal the distribution patterns of additional key active zone proteins in wild-type mouse NMJs using STED nanoscopy. Based on the knowledge obtained from wild-type NMJs, we also aimed to elucidate how active zone proteins are altered in amyotrophic lateral sclerosis (ALS). ALS is a neurodegenerative disorder in which denervation occurs before the death of neuronal cell bodies in the spinal cord, suggesting a “dying-back” neuropathy. The mechanisms underlying NMJ denervation in ALS remain unknown. Changes in protein levels, which are important for the maintenance of NMJ active zones and regulation of neurotransmission may play a role in the pathogenesis of ALS. For this purpose, we analyzed active zone proteins in NMJs of a rodent ALS model SOD1G93A mice at an early, pre-symptomatic stage (P85) and a symptomatic stage (P140). Interestingly, we found that the quantity of active zone proteins Bassoon, Piccolo and P/Q-type VGCC decreased in innervated NMJs of ALS mice. Impairments in presynaptic function are likely to contribute to NMJ denervation.

Next Generation Sequencing Pipeline for Metagenomic Data Analysis of Clinical Samples

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Keywords: metagenomic data analysis, bioinformatics, next generation sequencing

Abstract: Rapid detection of common viral pathogens by qPCR is effective, yet these methods can fail to identify highly mutable viral subtypes and cannot identify unknown viruses causing clinical disease. Next Generation Sequencing (NGS) has proven revolutionary in addressing a variety of biological questions at genome scale (1). NGS has become a standard protocol for diagnostic laboratories; however, complex data analysis due to huge volume of data still needs to be addressed (2). Though, several tools exist to support users in manipulation of datasets on various levels, few are built on the basis of vertical integration (pipeline). Here, we present the bioinformatics Next Generation Sequencing Analysis pipeline that allows non-expert users including wet-lab scientists to comprehensively build, run, and analyze NGS data and directly access the genetic content of the sample.

We develop a NGS bioinformatic pipelines for metagenetic analysis of samples received at Kansas State University Veterinary Diagnostic Laboratory. Here we describe the bioinformatic pipelines and report the results from a single enteric sample from each of two alpacas within the same herd. NGS revealed the presence of Betacoronavirus and Bocaparvovirus. Genome assembly and comparative analysis were performed to determine the evolutionary relationship of these viruses.

Protein Phosphatase 1 promotes a collective rather than single cell mode of migration

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Collective cell migration is critically important in embryonic development, wound healing, adult tissue renewal and cancer metastasis. Drosophila melanogaster undergo a developmentally regulated collective cell migration during oogenesis and provide an excellent genetic model for identifying how collectives move inside tissues. Here we show that Protein Phosphatase 1 (PP1) maintains the collective cohesion and migration of border cells. Inhibition of PP1 activity, either through the endogenous PP1 protein inhibitor NipPP1, or by knockdown of PP1 catalytic subunits, causes border cells to round up and completely dissociate from the cluster during migration. These defects are fully rescued by overexpressing PP1 catalytic subunits. The PP1-inhibited border cells switch to an overall slower individual cell motility mode, with altered protrusions that form randomly between cells. E-cadherin between cells is strongly reduced. Activated non-muscle myosin II (myo-II) and F-actin are now enriched around each individual border cell rather than at the outer edges of the entire border cell cluster. We have evidence that the myosin binding subunit (Mbs) of myosin phosphatase, a known PP1 complex, is required for proper border cell shape and collective cohesion. Thus, we propose that at least some of the cellular alterations caused by PP1-inhibition is due to altered myosin phosphatase function. Thus, normal PP1 activity levels are needed so that border cells can migrate as a collective. Given the high degree of conservation of PP1 complexes, this may be a common mechanism for collective cell migration in human development and disease.
140. Genomic dissection of natural variation resistance to copper poisoning in Drosophila melanogaster

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Metals have complex effects on organism physiology and function. Some metals are required for normal development and homeostasis, and deficiencies can result in disease, while metal poisoning poses risks of neurological and acute organ injury. Natural populations harbor genetic variation that influences resistance to metal poisoning. Genomewide dissection of this natural variation can help identify genetic factors that contribute to metal resistance, and provide insight into genes and pathways that regulate metal response. Here, we treat copper as a model metal of interest due to its critical requirement for normal cell function, and similarities between all forms of copper and nonessential metals. Using quantitative trait locus (QTL) mapping in the Drosophila Synthetic Population Resource (DSPR), we identify several candidate genes that are associated with copper resistance, and we show that the genomic architecture of copper resistance varies between DSPR mapping populations. Several of these candidate genes have known copper-specific functions, or are involved in the detoxification (Catsup), transport (Zip42C.2), or homeostasis (Ccs) of metal ions. These candidate genes will be validated using RNAi knockdown. Variation in gene expression between high and low copper resistance DSPR strains will also be further investigated using tissue-specific RNA sequencing of individuals following exposure to copper. Pollution via metal-containing industrial wastes and leaching of metals into ground water presents a pervasive threat to environmental and human health; our study provides valuable insight into the genomic architecture of metal poisoning susceptibility, and provides candidates for future functional and mechanistic validation.

141. The Role of RNA Binding Protein, HuR, in Pancreatic and Breast Cancer Malignancies

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RNA binding proteins (RBPs) are key regulators of gene expression. RBPs bind to mRNA and control mRNA stability, translation, cellular localization and transport. Hu antigen R (HuR) is an RBP, which binds to adenine and uridine rich elements (ARE) of mRNA 3’untranslated region. The overexpression of HuR in cell cytoplasm has been associated with tumor malignancy in various types of cancer, making HuR a promising therapeutic target for cancer. To study the role of HuR in tumor malignancy in pancreatic and breast cancer, we deleted HuR in pancreatic cell line MIAPaCa2 and breast cancer cell line MDA-MB-231 using CRISPR/Cas9 system. The deletion of HuR was confirmed by Western blot and qPCR assays. Two HuR knockout clones and a vector control clone of each cell line were picked for further studies. In both cell lines, HuR knockout clones were less proliferative and less invasive than their control and wild-type counterparts. In addition, loss of HuR resulted in a significant decrease in the colony formation in vitro and tumor formation in vivo. The HuR knockout clones of MDA-MB-231 cells were inoculated in nude mice to test whether the absence of HuR affects in vivo tumor formation capability. Overall, our results show that HuR has a significant role in cancer malignancy and progression, confirming its importance as a promising therapeutic target for cancer.

142. SPECC1L-Deficient Cells Show Impaired Collective Cell Migration Attributes that are Rescued by Upregulation of PI3K-AKT Pathway

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Clefts of the lip and/or palate (CL/P) are common anomalies that occur in 1/700 live births. Pathogenic SPECC1L variants identified in patients with rare atypical clefts and syndromic CL/P suggest the gene plays a primary role in face and palate development. Palate development requires extensive mesenchymal remodeling especially during palatal shelf elevation. We post that this remodeling involves collective cell migration (CCM) of neurocrest-derived palate mesenchymal cells. In vitro wound closure delay and the underlying CCM defect in SPECC1L-deficient cells consistently showed delayed wound closure. Coordinated directional movement of neighboring cells is a hallmark of CCM. In wound-repair assays, movement parallel to the direction of wound-front propagation can be measured as an attribute of CCM. However, trajectories of individual SPECC1L-deficient cells show increased cell movement perpendicular to the direction of wound closure. This diminished directionality causes delayed wound closure even though cells are adequately motile. We previously showed SPECC1L as a novel regulator of PI3K-AKT signaling, with in vitro and in vivo SPECC1L deficiency exhibiting reduced PI3K-AKT signaling. Indeed, wound closure delay and the underlying CCM defect in SPECC1L-deficient U2OS and MEPM cells is rescued by activation of PI3K-AKT pathway using 740Y-P small molecule activator. Our data are the first to show CCM progression, confirming its importance as a promising therapeutic target for cancer.

143. Equipment and Services of the Ralph N. Adams COBRE Core Nanofabrication Facility

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The Adams Nanofabrication Core Lab is a core lab within the Kansas University Nanofabrication Cleanroom Facility, and is supported by the Center for Molecular Analysis of Disease Pathways COBRE. The Adams Nanofab 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 ft² of ISO class 5, 1,700 ft² of ISO class 6 and 1,250 ft² of ISO class 7 cleanroom space, housing tools and materials for techniques including photolithography, nano-imprint lithography, plasma (dry) etching (ICP-RIE), wet etching, metal and dielectric material thin film deposition, ellipsometry, profilometry, wafer dicing, 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, dedicated process fume hoods and filtered lighting for photolithography.

This facility is under the direction of Dr. Susan Lunte. 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.
144. Next Generation Sequencing at KU Genome Sequencing Core

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The Genome Sequencing Core (GSC) is one of three research core labs in the NIH COBRE Center for Molecular Analysis of Disease Pathways (CAMPD) 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 HiSeq 2500 (large scale genome projects), NextSeq 550 (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 wide range of project support, from project consultation, sample quality check, library construction, cluster generation, data generation, to preliminary data analysis. For latest pricing, current job queue, or other info, visit the core’s website: https://gsc.ku.edu/

145. BCL9 protein-protein interactions that drive breast cancer progression

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Background: The mechanism by which ductal carcinoma in situ (DCIS) progresses to invasive ductal carcinoma (IDC) is not well understood. Previous studies show that B cell lymphoma-9 (BCL9) plays a key role in invasive progression of DCIS. In the Wnt pathway, BCL9 serves as a transcriptional cofactor that directly binds to β-catenin and enhances β-catenin mediated transcriptional activity. β-catenin directly binds to BCL9 homology domain-2 (HD2). In order to validate the role of BCL9-HD2 domain in oncogenic functions, we generated a DCIS cell line with deletion of the HD2 domain. We verified that BCL9-HD2 deletion significantly reduces DCIS progression by using our mouse-intraductal (MIND) method. In order to identify other domains BCL9-HD2 domain protein interactions, plasmids encoding flag tagged BCL9 full length and ΔBCL9-HD2 deletions were generated and immunoprecipitation (IP) assay was performed.

Results: Following transfection of full-length BCL9 and HD2Δ plasmids into our DCIS.COM cell line, western blot was used to assess initial protein presence. Our western blot indicated more flagged BCL9 in the HD2Δ construct as compared to the full-length BCL9 construct. HD2Δ construct also showed more active β-catenin when compared to full length BCL9. IP was used to pull down flag tagged BCL9. We found an increased amount of BCL9 in the HD2Δ construct. Active β-catenin was similar across both variants. Total active β-catenin was increased in the full length BCL9 construct and lower in HD2Δ construct.

Significance: These tools allow us to investigate the mechanisms by which BCL9 promotes DCIS invasive progression through unknown protein-protein interactions, allowing for therapeutic strategies in the future.

146. Antiviral evolution and population history of Drosophila innubila

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Hosts and viruses coevolve with each other in an evolutionary arms race at a time scale so rapid that it can often be observed almost in real time. Isolated geographical populations have the potential to observe this coevolutionary arms race in real time across “replicate” populations. Drosophila innubila is often infected with the Drosophila innubila Nudivirus and inhabits mountain woodlands in the southwestern USA. These mountains are often separated by hundreds of kilometers of desert which represent a formidable barrier to migration. To study host/virus coevolution in these distinct populations, we sequenced wild-caught individuals from each of four populations. Strikingly, the host population shows very little population structure across the genome aside from the Muller B chromosome where population structure appears to be driven by segregating inversions. The virus, on the other hand, is quite structured with most viruses fitting into one of three clusters almost perfectly. Given the genome-wide lack of structure, we focused on genes involved in the host immune response to viruses. Surprisingly, the canonical antiviral pathway, antiviral RNAi, shows little evidence of adaptive evolution. However, the JAK-STAT and Toll pathways, both involved in antiviral defense, showed rapid adaptive evolution. Finally, we performed genome-wide association studies in both host and virus to find variants associated with viral titre in the wild. We found no associated host variants, but several tightly linked viral polymorphisms were significantly associated with viral titre. Our results suggest that, asymmetry in gene flow may influence the coevolutionary arms race either with the host able to utilize mutations occurring throughout the species range or the virus able to specialize to a particular region.

147. Regulation of heterotrimeric kinesin-II motor complex trafficking by RanGTP and canonical nuclear import/export signals

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Kinesin is part of the microtubule-binding motor protein superfamily, which exerts crucial functions in cell division and intracellular transport. The heterotrimeric kinesin-II motor complex KIF3A/KIF3B/KAP3 plays diverse roles in cargo transport including anterograde trafficking in cillum. However, the molecular determinants mediating intracellular trafficking of this motor complex itself is unknown. Here we show that, besides ciliary localization of heterotrimeric motor complex, both the motor protein KIF3A and accessory protein KAP3 can shuttle between the cytosol and nucleus. Furthermore, RanGTP regulates nuclear translocation of KAP3. Given the emerging roles of RanGTP in ciliary protein targeting, we also investigate the effect of RanGTP in ciliary trafficking of KAP3 in different models. We demonstrate that, in Chlamydomonas, chemical inhibition of RanGTP/importin β-mediated import or ectopic expression of the dominant negative mutant Ran1(Q73L) can shorten flagellar length and regulate flagellar entry of Kinesin-II associated protein KAP, the orthologous protein of human KAP3. This recapitulates our finding in human retina pigment epithelium cells in which GTP-locked Ran mutant Ran(Q69L) can inhibit cilium assembly. Collectively, our results suggest canonical NLS/NES and RanGTP regulate intracellular transport of the kinesin-II heterotrimeric motor complex in different situations, which may provide new insights into molecular mechanisms regulating intracellular trafficking of the kinesin superfamily proteins.
148. Dissecting the structural basis for inhibitors of RNA-binding proteins

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Elevated RNA-binding protein (RBP) Musashi-2 (MSI2) expression is associated with ectopic oncogenic pathways including but not limited to NUMB/Notch, PTEN/mTOR, TGF-β/SMAD3, making MSI2 a promising therapeutic target for cancer. In the case of NUMB, MSI2 binds to and negatively regulates translation of NUMB, a negative regulator of Notch signaling. So far there is no structure available for MSI2Protein. Recently, we solved the first nuclear magnetic resonance (NMR) solution structure of MSI2 RNA-recognition Motif 1 (MSI2-RRM1) that forms the major long narrow RNA-binding pocket. Using a fluorescence polarization (FP) chemical libraries screening, we identified several groups of hits that disrupt the binding of MSI2-NUMB potently. These compounds induced apoptosis, inhibited cancer cell proliferation, invasion and metastasis in multiple tumor models tested in vitro and in vivo. They also interfered with cancer stem cell functions with reduced tumoursphere formation. Several leads showed promising efficacy in animal tumor models of human breast, prostate and colon cancer. To investigate the specificity of the compounds towards other RBPs, the compounds were tested in biophysical binding assays such as FP, time resolved Fluorescence Resonance Energy Transfer assay, NMR and cell based assays. We compared the affinity of these MSI2 inhibitors towards other RBPs such as MSI1 and HuR. Using docking and molecular dynamics simulation, we analyzed the structure activity relationship of these inhibitors towards different RBPs. This work adds significant information to the structure of MSI2-RRM1 and the structural basis for designing more potent and specific inhibitors of RBPs.

149. An Evolutionary Approach to Understanding Host Interactions with Microbial Pathogen

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Microbial pathogens are a constant threat to organismal survival. Although animals possess physical barriers to prevent entry of microbes, they must also recognize and respond when pathogens have bypassed these barriers. This response is modulated through the innate immune system, a defence mechanism comprised of evolutionarily ancient components. In its natural environment the microbivorous nematode, Caenorhabditis elegans, lives in close contact with numerous, potentially pathogenic bacteria. If systemic or intestinal infection occurs, the animal must respond appropriately. Models of intestinal infection have revealed that, although C. elegans possesses a generic innate immune response, there is no obvious conservation of the microbial defense pathways observed in arthropods and mammals, e.g. Toll or NF-κB. Rather, studies indicate that pathogen detection occurs via many different systems, converging on a core set of physiological responses as well as a set of pathogen-specific responses, some of which are conserved in other organisms (e.g. generation of reactive oxygen species, expression of antimicrobial peptides, etc.). To investigate the evolutionary basis for this discrepancy in innate immunity we use nematodes and fruit flies as model systems. First, we are assessing survival of several Caenorhabditis elegans species infected with various pathogenic bacteria to identify species-specific differences in pathogen susceptibility. Second, we plan to perform transcriptomic profiling of evolutionarily diverged species of nematodes and flies following exposure to microbial pathogens. Ultimately, our study seeks to shed light on the evolutionary origins of innate immunity as well as reveal uncharacterized aspects of mammalian defenses against infection.

150. Using Molecular Docking to Find Potential Anti-Cancer Drugs that Bind a Mutant form of Speckle-Type POZ, SPOP, a Protein Necessary for the Maintenance of a Number of Cancer Cells

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This research involves the development of therapeutic agent to target the protein known as speckle-type POZ, SPOP, that was found to be mutated in a number of cancers that include, kidney cancer and prostate cancer. The native SPOP is involved in carrying out post-translational modifications that are crucial for the vitality of normal cells. Therefore, it is strongly inferred that mutant SPOP found in cancer cells are crucial for the initiation and propagation of the cancer cell. The goal of this research is to find therapeutic agents that target the mutant form of SPOP in an effort to block it action in maintaining cancer cells. This was conducted using virtual molecular docking tools that allowed us to find a number of small molecules that bind mutant SPOP with high affinity. Further studies to experimentally corroborate the binding affinity parameters determined using molecular docking will be conducted. In Vitro cytotoxicity assays will also be conducted to determine the effects of these potential therapeutic agents on the vitality of cancer cells compared to healthy cells.

151. Applying Human Mesenchymal Stem Cells for the Treatment of Amyotrophic Lateral Sclerosis

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Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by a gradual loss of motor neurons that leads to paralysis and death. Studies indicate that neuromuscular junction (NMJ) denervation occurs in the early stages of the disease. We aimed to use a stem cell-based therapy in ALS model, SOD1G93A mice. Stem cells can be used as vectors to express and secrete proteins that are neuroprotective and promote NMJ maintenance. The objectives of this study are (1) to determine whether human umbilical cord derived mesenchymal stem cells (hMSCs) can secrete the synapse organizer, laminin β2, and neurotrophic factors, and (2) to evaluate the effect of hMSCs for maintenance of neuromuscular function and extension of life-span of SOD1G93A mice. We found that laminin β2 decreases in NMJs of SOD1G93A mice, and overexpressing laminin β2 in SOD1G93A mice ameliorates NMJ denervation. We discovered that stimulating hMSCs in media supplemented with growth factors can increase the secretion of laminin β2 and neurotrophic factor secretion. Furthermore, culturing primary motor neurons with the conditioned media of stimulated hMSCs maintained their survival in vitro. We then transplanted the hMSCs in SOD1G93A mice by intrathecal and intramuscular injections. Interestingly, NMJ innervation rate and median survival age of SOD1G93A mice were improved following hMSCs injection. This hMSCs injection could prove to be an efficient and a long-term delivery system for laminin β2 and provide a treatment method to reduce NMJ denervation and increase the quality of life of ALS patients.
152. Fighting Cancer By Targeting Cancer Stem Cells (CSCs) Using Virtual Docking Techniques

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DNAJB8, a member of a family of heat shock proteins, is found to be involved in the initiation and metastasis of cancers. This protein has been shown to be highly expressed in a number stem cells (CSCs), a type of cancer cells that are known to self-initiate, renew and differentiate. CSCs are thought to be resistance to the stress induced by chemotherapy and radiotherapy and are believed to be the reason behind the relapse after treatment. DNAJB8 has been shown to play a role in the survival of CSCs in cancers. The goal of this research is to find therapeutic agents that bind DNAJB8 and potentially block it from carrying out its role in maintaining CSCs in cancers. To do this we carried out a virtual screening study using molecular docking techniques that enabled us to find a number of small molecules that bind DNAJB8 with high affinity. Further studies to experimentally corroborate the binding affinity parameters determined using molecular docking will be conducted. In Vitro cytotoxicity assays will also be conducted to determine the effects of these potential therapeutic agents on the vitality of cancer cells compared to healthy cells.

153. Analysis of Leucine Zipper Mediated Interactions in the HNRNP C Family of Proteins

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Heterogeneous nuclear ribonucleoprotein C (hnRNP C), hRaly, and hRalyl share a high degree of sequence homology and conserve structural motifs that have been identified in hnRNP C. Each of the three proteins have an amino terminal RNA binding domain, an acidic carboxy terminal region, and a 4-heptad repeat of amino acids that is consistent with a leucine zipper motif. In general, leucine zippers stabilize protein-protein interactions and are responsible for the formation of homo or hetero-oligomers. Structural studies on synthetic hnRNP C leucine zippers have shown that they are anti-parallel homo-dimers. The presence of leucine zippers in hRaly and hRalyl that are highly similar to hnRNP C suggests that hetero-oligomers might form as a result of different protomer combinations from each of the three proteins. Molecular docking studies have confirmed support for this latter hypothesis. If the computational studies can be corroborated, then our presumption of three different functions for each protein must be re-examined based upon the number of potential heterologous interactions. We present here a research scheme that is in progress that will either undermine or corroborate our computational observations.

154. Homeodomain proteins linking lipid metabolism to gene expression in plants

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A fundamental question in cell and developmental biology is how lipid metabolism is integrated with gene transcription. In plants, Steroidogenic Acute Regulatory protein (STAR)-related lipid transfer (START) domains occur in class IV homeodomain leucine-zipper (HD-Zip IV) transcription factors that are critical for epidermal development. Initially discovered in mammalian STAR proteins, START domains are implicated in binding a variety of lipids. Our working hypothesis is that START functions as a lipid-binding domain to regulate transcription factor activity. Using EYFP fusions to a representative family member, GLABRA2 (GL2), we showed that deletion of the START domain results in nuclear localization albeit complete loss-of-function. Intriguingly, all START domain loss-of-function mutants including the newly identified missense allele g2(262792) display weak nuclear expression, suggesting that START is critical for protein stability. Deletion or missense mutations in the HD DNA-binding domain also result in loss-of-function while nuclear localization is intact. We are further characterizing a combination of HD and START mutants for protein turnover, DNA binding and dimerization. To discover START domain ligands, we are developing binding assays that use in vitro transcription-translation to produce recombinant protein, and immunoisolation followed by mass spectrometry to identify co-purified lipids. Our studies will provide a mechanistic platform for understanding how HD-Zip IV transcription factors drive epidermal cell fate, revealing the role of lipids in cell differentiation. Knowledge gained from this project could impact public health by providing new information on START domain proteins, which are hypothesized to serve as metabolic sensors and regulators of proliferation in human cancers.

155. Overexpression of the Legionella pneumophila Effector LegC4 Attenuates Intracellular Bacterial Replication

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Legionella pneumophila (Lpn) is a Gram-negative, rod-shaped flagellated bacteria that parasitizes and replicates within unicellular freshwater protozoa. Upon inhalation of aerosolized bacteria by immunocompromised humans, Lpn can cause a severe form of pneumonia called Legionnaires’ Disease. Lpn is an accidental human pathogen, and human-to-human transmission is very rare. Legionnaires’ disease results from an uncontrolled Lpn replication within alveolar macrophages, with the help of more than 300 effector proteins that the bacteria translocates directly into the host cell by a Dot/Icm type IVB secretion system. Previous work revealed that the Lpn effector LegC4 is detrimental for Lpn replication. We found that loss-of-function mutations in the legC4 gene enable enhanced Lpn replication within cytokine activated bone marrow-derived macrophages (BMDMs) and in the lungs of a mouse model of Legionnaires’ disease. However, overexpression of the legC4 gene attenuated Lpn replication in wild type BMDMs and in vivo. We found that LegC4 enhances TNF production in BMDMs and this contributes to attenuated replication of Lpn overexpressing legC4 in BMDMs. Inhibition of TNF receptor signaling abolishes the fitness defect associated with overexpressing legC4. Additionally, overexpression of legC4 also further attenuated Lpn replication in BMDMs activated with recombinant IFN-g, suggesting that multiple cellular defense mechanisms are being enhanced by LegC4. Current research in our lab is focused on understanding the molecular mechanism by which this occurs.
156. Elucidation of molecular mechanisms causing aging-related synapse degeneration using RNA-seq transcriptome data

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Neuromuscular junctions (NMJs), the site of communication between motor nerve axons and muscle fibers, play a key role in musculoskeletal impairment that occurs with aging. Several reports have shown gene expression profiling of aged skeletal muscle in human. General findings are that changes in mitochondrial genes in aged muscle and thereby mitochondrial dysfunction is feature of aging. However, synapse formation and/or maintenance-related specific genes and pathways that are affected by aging remain unclear. In the present study, we utilized an RNA-seq approach capable of capturing the whole transcriptome of mouse muscle. Specifically, we analyzed young adult mice and aged mice quadriceps and identified significantly altered transcripts. We identified 332 down-regulated genes and 533 up-regulated genes in aged mouse compared to young wild-type mouse. Ingenuity Pathway Analysis revealed that the expression levels of several synapse-related genes were changed in aged mouse. Among the changed genes, we found that laminin β2 (Lamb2) was significantly down-regulated in quadriceps of aged mouse. Laminin β2 is known to be important for the formation of NMJ in vertebrates. To investigate the involvement of Lamb2 in NMJs, we analyzed NMJs structure in laminin β2 LN-domain deletion mutant mice. We found that laminin β2 LN-domain deletion mutant mouse showed acetylcholine receptor cluster fragmentation and/or axon swelling near NMJs. These phenotypes are akin to NMJ phenotype of aged mouse. Significant downregulation of lamb2 mRNA and important role of LN-domain of laminin β2 protein suggest critical role of this gene in the maintenance of NMJs in aged animals.

157. Variation in DNA methylation of human blood over a 1-year period using the Illumina MethylationEPIC array

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Assessing DNA methylation profiles in human blood has become a major focus of epidemiologic inquiry. Understanding variability in CpG-specific DNA methylation over moderate periods of time is a critical first step in identifying CpG sites that are candidates for DNA methylation-based etiologic, diagnostic and prognostic predictors of pathogenesis. Using the Illumina MethylationEPIC [850K] BeadArray, DNA methylation was profiled in paired whole blood samples collected approximately 1 year apart from 35 healthy women enrolled in the Nurses Study II cohort. The minimal intraclass correlation coefficient (ICC) across all CpG loci was 0.19 [Interquartile Range (IQR) 0.00–0.50]; 74.8% of ICCs were in the low range (0–0.5), 16.9% in the mid-range of ICCs (0.5–0.8), and 8.3% in the high-range of ICCs (0.8–1). ICCs were similar for CpG probes on the 450K Illumina array (median 0.17) and the new probes added to the 850K array (median 0.21). ICCs for CpG loci on the sex chromosomes and known metastable epialleles were high (median 0.71, 0.97, respectively), and ICCs among methylation quantitative trait loci (mQTL) CpGs were significantly higher as compared to non-mQTL CpGs (median 0.73, 0.16, respectively, P < 2 × 10–16). We observed wide variation in DNA methylation stability over a 1-year period. Probes considered non-stable, due to substantial variation over a moderate period of time and with minimal variability across individuals could be removed in large epidemiological studies. Moreover, adjusting for technical variation that arises from using high-dimensional arrays is critical.

158. 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 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 additionally provides access to the model organism Danio rerio (zebrafish), and allows investigators to image embryonic and adult zebrafish treated with molecular probes for phenotypic drug discovery and other projects. SCB core encompasses the Purification and Analysis Laboratory (PAL) that provides purification, analysis and quality control of compounds via HPLC-MS. The core utilizes automated mass directed fractionation for purification in both reversed and normal phases (including chiral separations), and also provides relative purity analysis by UPLC coupled to a high-resolution mass spectrometer for structure confirmation.

159. The RNA Binding Protein HuR Regulates Exosome Secretion in Colorectal Cancer via Rab 27B

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Enhanced secretion of exosomes by cancer cells is recognized as a means of transferring specific RNA and protein cargo to recipient cells, and is promising blood-based cancer biomarkers. We have established that colorectal cancer (CRC) cells and tumors overexpress the RNA-binding protein HuR (ELAVL1) early in GI tumor development. When overexpressed, HuR can promote mRNA stabilization of tumor-promoting genes through binding of 3'UTR AU-rich elements (ARE). These same mRNAs are within tumor-derived exosomes, suggesting role for HuR in RNA trafficking. To test this, HuR-inducible HeLa cells were used to demonstrate that HuR overexpression promoted a 4-fold increase in exosome secretion (50-150nm). Furthermore, HuR was detected in exosomes produced only from HuR-overexpressing cells. To assess this in CRC cells that endogenously overexpress HuR, exosome levels were compared to normal human intestinal epithelial and myofibroblast cells. CRC cells secrete ~3-fold greater exosome levels than normal cells. These findings were reflected in vivo where GI-tumor bearing ApcMin/+ mice produced ~3-fold more serum exosomes, with HuR as exosome cargo in ApcMin/+ mice compared to wild-type mice. Organs derived from ApcMin/+ mice adenomas showed ~3-fold higher number of exosomes released as compared to normal intestinal tissues. Interestingly, HuR was specifically present in adenomas derived organoid exosomes. It has also been investigated that HuR regulates exosomes secretion via Rab27B in CRC cells. Silencing of Rab27B inhibits exosome secretion in CRC cells with decreased multi-vesicular body’s size. This work has identified a novel connection between HuR-mediated post-transcriptional regulation and tumor-derived exosomes secretion, along with providing preclinical evidence of exosomes HuR as a serum-based CRC biomarker.
160. Mechanism of Genome Instability Driven by RB Loss

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The Retinoblastoma protein (RB) is a cell cycle repressor that is broadly conserved in many eukaryotes. RB is defective in over 90% of human cancers. Loss of RB causes de-repression of the cell cycle genes, resulting in uncontrolled cellular proliferation. RB has additional cellular functions that are poorly understood and contribute to tumorigenesis.

The mammalian RB pathway has many redundant factors. Humans have three RB-like proteins, nine E2F proteins, and two DP proteins, each with unique and overlapping functions. In addition to their canonical role in cell cycle regulated gene transcription, RB is also an integral part of chromatin where it is important for genome stability through a mechanism that is poorly understood. Thus, defects in RB usually have compounding effects where cells become hyper-proliferative and defects in their genome increases to a very high rate furthering tumorigenesis and malignancy.

Surprisingly, a unicellular alga Chlamydomonas reinhardtii, is an advantageous model system for studying RB in a simplified eukaryote. It has a single homolog of RB, E2F and DP, where RB null cells show a similar hyperproliferative defects as in human cells, as well as a high rate of genome instability. Thus, C. reinhardtii provides a simplified model system where fundamental aspects of RB mechanism can be much more easily studied that in more complex eukaryotes. We hypothesize that chromatin-bound RB is important for genome stability, where RB loss leads to a non-random loss of genome integrity especially under stressed conditions.

161. Septate junction proteins maintain tissue integrity during dorsal closure

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The process of dorsal closure in Drosophila involves the dorsalward movement of the lateral epidermis to cover a gap temporarily occupied by the extraembryonic amnioserosa and requires continuous interaction between the two tissues to coordinate morphogenetic movements and maintain embryonic integrity. The interface between these two tissues is maintained by both integrins and adherens junctions. Although components of the occluding septate junction (SJ) such as Coracle (Cora) and Macroglobulin complement-related (Mcr) do not appear to be localized to this region, they are required for efficient completion of dorsal closure. Here, we are studying the role of these proteins during closure, including through live imaging of SJ mutants. Despite their absence in the amnioserosa and at the leading edge, we find these proteins are critical for maintaining interaction between the lateral epidermis and amnioserosa, and separation of these tissues along the leading edge is responsible for the closure phenotypes in cora, Mcr, and Neurexin-Iv mutants. Dorsal closure progresses normally in these mutants until the later stages of closure, when the DE-cadherin protein appears to become depleted within the amnioserosa and along the leading edge and tears occur between the two tissues. Laser ablation studies show that tension and the viscosity-elasticity ratio are not affected in these mutants, suggesting the tearing is caused by adhesion defects. These results suggest a role for SJ proteins in adhesion, but the absence of these proteins along the leading edge suggests an indirect role, possibly by regulating levels of DE-cadherin and/or integrins.

162. Bioinformatics for You: A KINBRE-Wide Short Course in Next-Generation Sequence Analysis

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Are you a student who is curious about sequence analysis and bioinformatics? Are you a faculty member who would like to provide your students with bioinformatics experience? The Kansas and New Hampshire KINBRE Bioinformatics Cores are developing a short, approximately two-week course that will provide an introduction to bioinformatics via hands-on analysis of real sequence data from mutant microbes. Students will develop skills in using the bash/command line interface in Linux, quality control/trimming of next-generation sequencing data, microbial genome assembly and annotation, read mapping and SNP calling. Course participants will have the opportunity to be authors on an eventual publication. Our goal is to make this course available to students from all KINBRE institutions by collaborating with faculty at each university such that students can receive course credit through their own institution. If you are interested in this opportunity, please stop by our poster to visit with us.

163. Increasingly complex protein interactions with contracting gene groups may have led to evolution of multicellularity in Volvocales

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The transition from unicellular to multicellular organisms is a major step in the evolution of life on earth. However, the molecular basis of this transition has remained largely elusive. The Volvocine algae consist of several species representing increasing multicellular complexity and thus provide an excellent model for the study of the evolution of multicellularity. The genomes of Chlamydomonas (Unicellular), Gonium (undifferentiated multicellular), Yamagishiella (Partial germ/soma division), Eudorina (size expanded anisogamy) and Volvox (differentiated multicellular) were compared. The genomes were found to be highly similar on the basis of GC content, gene counts, number of Pfam domains and orthologous groups. However, the number of contracting Pfam domains and orthologous groups were significantly greater than the number of expanding ones. Finer scale analysis of histone genes, transcription factors and protein kinases followed a similar trend. Analysis of previously proposed mechanisms of evolution of complexity including phylastrum specific genes, positive selection and intrinsic protein disorder did not show any specific trend with increasing complexity and thus, were rejected. Analysis of Pfam domain network showed an increase in network modularity indicating a greater number of domains per gene with increasing biological complexity. Increase in protein interaction complexity was tested on 2D SDS page which showed that a large number of protein complexes were having more complex interactions with increasing biological complexity. In conclusion, we propose that Volvocales evolved multicellularity by increasing complexity of protein interactions and expression networks leading to newer functions of pre-existing genes.
164. K-INBRE Undergraduate Coordinator Office: Continued Activities & Impact

Sarah E. Velasquez, K-INBRE, Department of Anatomy and Cell Biology, University of Kansas Medical Center

**Background:** The primary goal of the Undergraduate Coordinator Office is to engage undergraduate students in biomedical research at Kansas IDeA Network of Biomedical Research Excellence (K-INBRE) participating universities, developing seamless career pathways leading to graduate education and professional careers in biomedical disciplines.

**Method:** This office employs six initiatives: 1) Solicit programs at participating universities developed to attract and retain promising students in biomedically-related majors while engaging these students in cutting edge research; 2) Assist the Campus Coordinators in implementing these programs; 3) Support the Communications Core in providing professional development to students and faculty alike; 4) Assist in the implementation of the Annual Symposium where students can present their research, hear research presentations by internationally recognized scientists, and network with others; 5) Supervise the awarding of funds to support Bioinformatics Scholars, Community Based Scholars, Star Trainees, Summer/Semester Scholars, and Translational Research Scholars; and 6) Oversee student assessments and evaluations to determine program effectiveness.

**Results:** Valuable data about students in the pipeline are collected to quantify the impact of the K-INBRE on student success at the undergraduate level and beyond.

**Discussion:** The ongoing mechanisms used to implement our program and data showing the K-INBRE effectiveness and impact. This poster is made possible by an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences of the National Institutes of Health under grant number P20GM103418

165. K-INBRE Communications Core: Continued Activities & Assessment

Sarah E. Velasquez, K-INBRE, Department of Anatomy and Cell Biology, University of Kansas Medical Center

**Background:** The Kansas IDeA Network of Biomedical Research Excellence (K-INBRE) Communications Core aims to expand and advance biosciences education and research among Kansas (and Oklahoma) science researchers, educators, and students.

**Method:** With partner institutions and participants dispersed across Kansas and Oklahoma, the Communications Core has the complex role of, 1) evaluating the effectiveness of programmatic and collaborative activities for continued improvement and scholarship of the program; 2) advancing and leveraging the K-INBRE infrastructure for professional development of students and faculty alike, filling a niche that may not be provided in degree programs or experiences; 3) highlighting the role and accomplishments of K-INBRE on bioscience education, research, faculty, and students engaged in the project; and 4) enhancing the daily operational and programmatic communication between faculty, students, and researchers within the K-INBRE and IDeA networks.

**Results:** Continued evaluation and assessment of the K-INBRE, coupled with changes in technology, has illuminated unique opportunities for communications and professional development activities for the program.

**Discussion:** This poster will provide details on ongoing Communications Core activities. This poster is made possible by an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences of the National Institutes of Health under grant number P20GM103418

166. Role of RNA Binding Protein HuR in Non-alcoholic Fatty Liver Disease (NAFLD) Progression

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Nonalcoholic fatty liver disease (NAFLD) includes a spectrum of diseases, from nonalcoholic fatty liver (NAFL) to nonalcoholic steatohepatitis (NASH) with liver dysfunction leading to cirrhosis and hepatocellular carcinoma (HCC). NAFLD is characterized by the accumulation of lipids (steatosis) and cellular inflammation in liver. Human antigen-R (HuR, ELAVL1) is a key RNA binding protein involved in mRNA stabilization of various pro-inflammatory and oncogenic genes. So far, the role of HuR in NAFLD progression with respect to inflammation and steatosis has yet to be explored. Using immunohistochemistry for HuR expression in human liver tissues, we observed a significant increase in HuR expression and cytoplasmic localization during NAFLD progression, with significant HuR immunoactivity score (IRS) occurring during transition from NAFL to NASH, which remain elevated in cirrhosis and HCC. Fatty acid (palmitic acid) induced steatosis in both HuR and HepG2 hepatocellular carcinoma (HCC) cells showed elevated HuR expression (~4 fold) suggesting its transcriptional upregulation upon fatty acid uptake. Using luciferase reporter constructs, we noticed steatosis enhanced both human and mouse HuR promoter activity (2.2- and 2.5-fold, respectively) in HuR cells. Further, knocking-out HuR by CRISPR-Cas9 technique or knocking-down using HuR-siRNA in HCC cells demonstrated significant decrease in fatty acid-induced steatosis, suggesting involvement of HuR in disease progression. Additionally, inhibition of HuR using the small molecule natural product 15,16-dihydrotanshinone-I (DHTS) prevented fatty acid-induced steatosis in HCC cells. These findings suggest that HuR is upregulated at the onset of NAFLD to facilitate steatosis and implicate HuR as potential novel therapeutic target for NAFLD treatment.

167. Targeting viral glycans with lectin mediated immunotherapy

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Enveloped viruses cause a number of life-threatening human diseases. Glycoprotein (GP) spikes, present on viral particles and the exocellular membrane of viral infected cells, facilitate infection by binding to motifs on target cell receptors, leading to membrane fusion, the hijacking of host cell machinery and viral replication. Viral GPs are highly glycosylated and this “glycan shield” diminishes the ability of the host immune system to target viral GP antigenic sites. Lectins, such as Cyanovirin-N and Griffithsin, have been shown to inhibit infectivity of an array of viruses. These lectins bind with high specificity and affinity to high-mannose N-glycans, which are ubiquitously present on viral GPs and are importantly absent on the surfaces of healthy human cells. The ability of these lectins to neutralize the infective potential of a number of different viruses led us to envision a lectin derived antiviral immunotherapeutic. These molecules would maintain the ability to specifically bind viral high-mannose glycans and inhibit viral infectivity, while having the added functionality of being able to recruit the immune system to destroy viral particles, and GP expressing viral infected host cells. This added functionality will arise via the conjugation of these lectins to dinitrophenyl (DNP) or L-rhamnose (Rha) antibody recruiting motifs. DNP and Rha specifically bind endogenous antibodies (Abs) found in human sera. It is envisioned the resulting Cyanovirin-N Antibody Recruiting Molecules would be capable of: (i) inhibiting viral infectivity; and (ii) recruiting Abs to the surfaces of viral particles and infected cells, and thereby inducing an antiviral immune response.
168. In-frame Deletion of SPECC1L Coiled-Coil Domain 2 in Mice Results in Exencephaly and Cleft Palate

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Cleft lip with or without cleft palate (CL/P) and anencephaly/exencephaly are genetically heterogeneous birth defects that affect 1/700 and 1/1000 births, respectively, so there is continued need to identify genes that play a role in their etiology. SPECC1L mutations identified in patients with syndromic craniofacial pathologies cluster in the second coiled-coil domain (CCD2), highlighting the importance of this domain in SPECC1L function. To study SPECC1L function in mice, we generated genomic deletions that resulted in out-of-frame truncations. Homozygous mutants (C57BL6/J background) for these truncations, even those lacking CCD2, died shortly after birth without cleft palate or exencephaly. Given that most patients identified to-date carried CCD2 mutations, we hypothesized that targeted perturbation of CCD2 may be required. Indeed, homozygotes for in-frame deletions involving CCD2 (Specc1lac300) resulted in ~50% exencephaly and ~50% cleft palate. Interestingly, these two phenotypes are never observed in the same embryo, suggesting antagonistic tissue mechanics between neural tube and palate closure. We also generated an in-frame deletion (Specc1lac300) that is downstream of CCD2. Homozygous mutants for Specc1lac300 do not show cleft palate, however, ~20% do display exencephaly. Thus, regions beyond CCD2 can affect neural tube closure, albeit with reduced penetrance compared to when CCD2 is involved. Importantly, our results show that perturbations of CCD2 in the context of the rest of SPECC1L protein specifically affect palate closure, suggesting that human SPECC1L CCD2 mutations are gain-of-function. These mouse models validate the importance of CCD2 in SPECC1L and serve as useful tools in understanding the etiology of craniofacial malformations.

169. Targeting RNA-binding protein HuR to inhibit human breast cancer invasion and metastasis

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Patients diagnosed with metastatic breast cancer have a dismal 5-year survival rate of only 24%. The RNA-binding protein Hu antigen R (HuR) is overexpressed in breast cancer, and elevated cytoplasmic HuR correlates with high-grade tumors and poor clinical outcome of breast cancer. HuR promotes tumorigenesis by regulating numerous proto-oncogenes, growth factors and cytokines that support major tumor hallmarks including invasion and metastasis. Knocking out HuR inhibits breast cancer cell growth and invasion. Therefore, HuR is an emerging target for breast cancer therapy, especially the lethal metastatic breast cancer. Here, we report a novel HuR inhibitor KH-3, which potently suppresses breast cancer cell growth and invasion similar to HuR knockout. In the study of mechanism of action, a transcription factor, FOXQ1, is found for the first time to be a direct mRNA target of HuR and one of the top genes that are reduced by KH-3 treatment. Exogenous introduction of FOXQ1 can rescue cell invasive capability impaired by HuR knockout and abolish the effect of KH-3 on inhibiting cell invasion. Moreover, KH-3 disrupts HuR-FOXQ1 interaction and RNP-IP, RNA pull down and FOXQ1 3′-UTR luciferase reporter assays. In vivo efficacy studies show that KH-3 not only exhibits potent antitumor efficacy in an orthotopic xenograft model of breast cancer, but also efficiently inhibits lung metastasis and improves mouse survival in an experimental metastasis model. Our data provide a proof-of-principle that HuR inhibition by KH-3 may be developed as a promising molecular therapy for inhibiting progression and metastasis of breast cancer with high HuR.

170. Headcase regulates tissue growth and cell cycle progression in response to nutrient restriction

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Nutrient restriction (NR) decreases the incidence and growth of many types of tumors. To date, the anti-tumorigenic effects of NR have been well established and its potential implications in both cancer prevention and treatment have been suggested. Despite these advances, our understanding of the molecular mechanisms underlying the anti-tumorigenic effects of NR remain fragmented. It has been shown that cells with PI3K activation are resistant to NR in both mammalian and Drosophila tumor models. And TORC1 activation has been shown to play a key role in NR resistance of PTEN or TSC-null tissues in Drosophila. The underlying molecular mechanisms of upregulated TORC1 activation on insulin/TORC1 mediated NR resistance is unclear. Here we identified Headcase (Hdc) as a tumor suppressor that regulates tissue growth in response to NR. We found that hdc mutant cells do not show apparent growth advantage under normal nutrient conditions but proliferate much faster than wildtype cells under NR. Our results suggested that Hdc regulates tissue growth through its regulation on cell cycle progression. We further found that Hdc binds to Unkempt (Unk) and forms a protein complex with TORC1 component Raptor. Taken together, our study suggests a functional link between Hdc/Unk and insulin/TORC1 signaling on cell cycle progression and NR resistance.

171. C. elegans hrRNPK homolog is a novel modulator of miRNA activity

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MicroRNAs (miRNAs) are small non-coding RNAs that post-transcriptionally regulate gene expression by base-pairing to the 3′-untranslated regions (3′UTR) of target mRNAs and reducing their translation or degrading the target mRNAs. miRNA activity is essential for animal development and homeostasis, and alterations in miRNA expression or function are associated with pathologies and diseases. Although significant progress has been made in understanding the mechanisms by which miRNAs post-transcriptionally regulate gene expression, little is known about how miRNA activity itself is regulated. Using proteomics approaches followed by genetic analyses in Caenorhabditis elegans, we identified a conserved RNA binding protein, HRPK-1, as a novel regulator of miRNA activity. Loss of hrpk-1 enhances the phenotypes associated with partial loss of miRNA function, suggesting that HRPK-1 acts as a positive regulator of miRNA activity. Additional molecular evidence suggests that HRPK-1 feeds into the miRNA gene-repression pathway at the level of the target mRNAs. To get a better molecular understanding of how HRPK-1 may regulate miRNA activity, we have begun to identify conserved regulatory regions of HRPK-1 protein domains. Here, we will present our current model for HRPK-1 role in miRNA-mediated gene regulation and our ongoing efforts on functional dissection of HRPK-1 protein domains.
172. ESR2-regulated ovarian kisspeptins in oocyte maturation

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Hypothalamic kisspeptins are essential for the onset of puberty and regulation of ovulation. Ovaries also express kisspeptins correlating with puberty and estrous cyclicity. Granulosa cells (GCs) express \( \text{Kiss1} \) and oocytes express the receptor (\( \text{Kiss1r} \)). While ESR1 regulates hypothalamic \( \text{Kiss1} \) expression, ovarian \( \text{Kiss1} \) expression is ESR2-dependent. Gonadotropin treatment fails to induce \( \text{Kiss1} \) expression in \( \text{Esr2}^-/- \) GCs. We characterized ESR2 regulation of \( \text{Kiss1} \) by identifying GC-specific transcripts and regulatory regions. The \( \text{Kiss1} \) promoter, an upstream enhancer, and a downstream enhancer all possessed conserved EREs and showed active histone marks in GCs. The transcriptionally-active \( \text{Kiss1} \) promoter and the enhancers revealed enrichment for ESR2-binding. \( \text{Kiss1} \) promoter-activity was induced after ESR2 overexpression and blocked upon mutating an ERE within the promoter. Gonadotropin administration induced ERK2 phosphorylation (pERK2), which interacted with ESR2, demonstrating a role for pERK2 in ESR2 phosphorylation (pESR2). Gonadotropin treatment also induced AP-1 factors FOSL2 and JUNB. These findings show LHCGR-activated estrogen signaling via pESR2 and AP-1 factors regulate \( \text{Kiss1} \) expression in GCs. Remarkably, cumulus oocyte complexes (COCs) isolated from PMSG-treated \( \text{Esr2}^-/- \) ovaries exhibit inefficient meiotic activation in \textit{in vitro} maturation, which may be linked with the lack of kisspeptins in GCs. To examine the potential role of kisspeptins on oocyte maturation, COCs were treated with kisspeptin-10 \textit{in vitro}. This elevated phosphorylation of ERK in oocytes and expression of Gdf9, Bmp15 and c-Mos indicate an important role for KISS1/KISS1R signaling in oocyte maturation. Our results indicate ovarian \( \text{Kiss1} \) expression is regulated by ESR2 and that kisspeptins play an important role in oocyte maturation. (Supported by KINBRE)

173. Prion Gene Polymorphisms in Feral Pigs

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Prion diseases are a group of infectious, incurable, fatal neurodegenerative conditions, affecting mammals such as, scrapie in sheep, bovine spongiform encephalopathy in cows, chronic wasting disease in deer and Creutzfeldt-Jakob disease (CJD) in humans. Prion diseases are caused by the misfolded form of the normal cellular prion protein (\( \text{PrP}^\text{C} \)). Prion disease occurrence depends mainly on the interaction between the host prion protein \( \text{PrP}^\text{C} \) and the prion strain (\( \text{PrP}^\text{Sc} \)). The closer the sequence, the easier to cause prion disease. Interestingly, some animal species are considered resistant to prion diseases, such as pigs, rabbits, and dogs, since no single case of naturally-occurring disease has been reported in them. However, some recent \textit{in vitro} studies have shown that pigs can be infected with different prion strains such as chronic wasting disease and bovine spongiform encephalopathy. In our research we are looking for polymorphisms in the \( \text{PrP}^\text{C} \) gene by comparing wild pig sequences to those of domestic pigs, and another susceptible species such as deer. This work is important because pigs might be a silent carrier, and they are extraordinarily used in human daily life, as well as an alternative source of organs for transplant.
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<td>Zurek</td>
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