48th Annual Meeting

of the

Texas Genetics Society

Virtual Conference
March 25-26, 2021

www.texasgeneticssociety.org

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Mark Kirkpatrick, The University of Texas at Austin

I am a population geneticist who has worked on sexual selection, quantitative genetics, and chromosome evolution. I was born in New York City, grew up in New Jersey, and graduated from Harvard University. On a one-year sabbatical from college, I spent several months in the lab of Robert Selander, who interested me in population genetics. I went to graduate school at the University of Washington, where I was advised by Monty Slatkin and also interacted extensively with Joe Felsenstein. When beginning at UW, I was unsure what area I wanted to pursue, and so I tried several things. I soon discovered that although I enjoyed fieldwork, I was not good at it, and I was even worse in the lab. But mathematical modeling was something I could do, and I loved it, so that became my career. After getting a Ph.D. in 1983, I went to U.C. Berkeley as a Miller Postdoctoral Fellow, where David Wake sponsored me as the first (and last?) theoretical biologist in the history of the Museum of Comparative Zoology. I then joined the faculty of the University of Texas at Austin, where I have been ever since. Over the last decade, our research has pivoted to developing new methods to analyze genomic data to study questions such as how new sex chromosomes originate. I am a Fellow of both AAAS and a Member of the National Academy of Sciences.

Georgios Karras, MD Anderson Cancer Center

I am an Assistant Professor in the department of Genetics at MD Anderson Cancer Center. I pursued my PhD studies under the supervision of Dr. Stefan Jentsch, a pioneer in the ubiquitin field. In his lab, at the Max-Planck Institute for Biochemistry in Munich Germany, I studied the timing and mechanism of mutagenesis and postreplicative DNA repair in budding yeast. There, I became enamored of genetics, and genetic screens and the approaches to study the contextual effects of genetic variation in evolution. For my postdoc I wanted to apply my knowledge to the study of human genetic variation, so I joined the lab of Dr. Susan Lindquist at MIT in Cambridge MA, who was a pioneer of the protein homeostasis field. There, I demonstrated that the molecular chaperone Hsp90 "buffers" human genetic variation and can alter the course of disease. I started my lab in 2018, since then I was fortunate to recruit many outstanding trainees and we are currently a healthy group of eight scientists. In my lab, we employ functional genomics and robot-assisted proteomics approaches to study age-related disease and cancer evolution. We also use budding yeast and CRISPR-engineered human cancer cell lines to understand Hsp90's broader impact on shaping evolutionary processes from the population level all the way down to the molecular level. I would like to acknowledge the following awards: CPRIT recruitment award, IT STARs award, K22/NCI award.

Texas Genetics Society 48th Annual Meeting
March 25-26, 2021
Virtual Meeting Via Zoom

Thursday, March 25

1:30 – 3:00 pm **Board Meeting**

3:00 – 3:05 pm **Welcome and Announcements**
David P. Aiello, Ph.D., TGS President

3:05 – 4:30 pm **Contributed Papers Session I**
Moderated by: Joseph Manthey, Ph.D., Texas Tech University

3:05 – 3:25 **An 8.22 Mb assembly of the alpaca (*Vicuna pacos*) Y chromosome**
Matthew Jevit, Texas A&M University, College Station

3:25 – 3:45 **The rate and spectrum of EMS-induced germline mutations in the microcrustacean *Daphnia*: on the prospect of forward genetics**
Marelize Snyman, University of Texas at Arlington, Arlington

3:45 – 4:05 **PROTECTION OF TELOMERES 1b regulates chromatin structure to prevent genomic oxidative damage during development in *Arabidopsis thaliana***
Claudia Castillo-González, Ph.D., Texas A&M University, College Station

4:05 – 4:25 **Ethnicity-specific effects of DNA methylation-based biological age clocks**
Talisa Silzer, M.S., Ph.D., University of North Texas Health Science Center, Ft. Worth

4:30 – 5:50 pm **Poster Session and Sponsors' Meet-and-Greets**
Poster Session Zoom Breakout Rooms (see below)

4:30 – 5:10 Poster #s 1-23 presenting authors in breakout rooms

5:10 – 5:50 Poster #s 24-46 presenting authors in breakout rooms

5:50 – 6:00 pm **Break**

6:00 – 7:00 pm **Barbara Bowman Award Recipient**
Mark Kirkpatrick, Ph.D.
Department of Integrative Biology, The University of Texas at Austin
"Fish with three tales: Evolution of recombination on the sex chromosomes of sticklebacks?

7:00 – ? **Informal Zoom Social Time**
Social breakout rooms or main meeting room

Friday, March 26

3:00 – 3:05 pm **Welcome and Announcements**
David P. Aiello, Ph.D., TGS President

3:05 – 4:05 pm **Invited Lecture**
Georgios Karras, Ph.D.
Department of Genetics, The University of Texas MD Anderson Cancer Center
"Illuminating Hsp90's Selection Shadow"

4:05 – 4:15 pm **Break**

4:15 – 5:15 pm **Contributed Papers Session II**
Moderated by: Tina Gumienny, Ph.D., Texas Woman's University

4:15 – 4:35 **Fitness Effects of a Potentially Selfish Mitochondrial Deletion in *Caenorhabditis elegans***
Abigail Sequeira, Texas A&M University, College Station

4:35 – 4:55 **The dopaminergic stress response limits dopamine oxidation and toxicity following mechanical stress by preventing cytosolic dopamine accumulation**
Kielen Zuurbier, UT Southwestern Medical Center, Dallas

4:55 – 5:15 **Differential Regulation of Glycolytic Genes in Glioblastoma Multiforme**
Shreya Udawant, University of Texas-Rio Grande Valley, Edinburg

5:15 – 5:30 pm **Break**

5:30 – 6:30 pm **Contributed Papers Session III**
Moderated by: Megan Keniry, Ph.D., University of Texas-Rio Grande Valley

5:30 – 5:50 **Determining the function of the transcription factor Cmr3 in wild type *Saccharomyces cerevisiae* and its role in the *spt4*Δ-mediated rescue of *pgm2*Δ phenotypes**
Mandy Eckhardt, Austin College, Sherman

5:50 – 6:10 **Cleavage Efficiency of Flavivirus NS3 Proteases and Protease Recognition Sequences in *Aedes aegypti* Cells**
Alexius Dingle, Texas A&M University, College Station

6:10 – 6:30 **Calcium ions trigger the exposure of phosphatidylserine on the surface of necrotic cells**
Yoshitaka Furuta, Baylor College of Medicine, Houston

6:30 – 6:50 pm **Break**
Judges Meeting (in breakout room)
Trainees Meeting (in breakout room)

6:50 – 7:15 pm **General Business Meeting and Awards Presentation**

7:15 pm **Adjourn**

Contributed Papers Abstracts

An 8.22 Mb assembly of the alpaca (*Vicuna pacos*) Y chromosome

Matthew Jevit[1], Brian Davis[1], Caitlin Castaneda[1], Andrew Hillhouse[1], Rytis Juras[1], Vladimir Trifonov[2], Ahmed Tibary[3], Jorge C. Pereira[4], Malcolm A. Ferguson-Smith[4], Terje Raudsepp[1]

[1]*Texas A&M University, College Station, TX;*
[2]*Institute of Molecular and Cellular Biology, Novosibirsk, Russian Federation;*
[3]*Washington State University, Pullman, WA;*
[4]*University of Cambridge, Cambridge CB2 1TN, UK*

The unique evolutionary dynamics and complex structure make the Y chromosome the most diverse and least understood region in the mammalian genome, despite its undisputable role in sex determination, development, and male fertility. Here we present the first contig-level annotated draft assembly for the alpaca (*Vicugna pacos*) Y chromosome. We sequenced flow-sorted Y DNA on PacBio Sequel and Illumina MiSeq (2x300bp reads) platforms. Long reads were assembled with CANU and short reads were incorporated with PILON. The resulting hybrid assembly was 20,060,146 bp in 652 contigs. Flow sorted Y chromosomes were also used for cDNA selection providing Y-enriched testis transcriptome for annotation. Contigs with known Y genes were confirmed to be a part of the male specific region of Y (MSY) by BLAST search against the female reference genome VicPac3.1 and by PCR on male and female genomic DNA. To be incorporated in MSY, the contig must not BLAST to the reference genome and the primers had to amplify in males only. Pseudoautosomal (PAR) contigs were identified by the annotation of known PAR genes and high homology with the X chromosome. The final assembly of 8.22 Mb comprised 4.5 Mb of MSY and 3.7 Mb of the PAR. In MSY, we annotated 15 X-degenerate genes and two novel transcripts, but no transposed sequences. We also identified 20 known PAR genes. Several MSY genes appeared multiple times in MSY contigs including HSFY (26x) and RBMY (3x), EIF1AY (2x), CUL4BY (2x), and TSPY (3x). The relative copy number was tested with qPCR using UTY as a single copy control. RBMY and HSFY had approximately 13 and 18 copies, respectively. EIF1AY, CUL4BY and TSPY did not show a significant copy number change when compared to UTY. While defining MSY and PAR contigs, we were also able to identify the pseudoautosomal boundary (PAB). The PAB was located in contig419, part of which showed high homology to the X chromosome and the known PAR genes SHROOM2 and GPR143. The other half of this contig was male-specific by PCR and contained known Y genes EIF1AY and HSFY. Based on this, we located PAB-Y between SHROOM2 and HSFY, and PAB-X between SHROOM2 and WWC3. Comparative analysis showed that the small and cytogenetically distinct alpaca Y shares most of MSY sequences with the larger dromedary and Bactrian camel Y chromosomes. Most of alpaca X-degenerate genes are also shared with other mammalian MSYs, though WWC3Y is Y-specific only in alpaca/camels and the horse. The partial alpaca Y assembly is a starting point for further expansion and will have applications in the study of camelid populations and male biology.

Matthew Jevit

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Graduate Student
Contributed Papers Session I
Award Competition: Yes

The rate and spectrum of EMS-induced germline mutations in the microcrustacean *Daphnia*: on the prospect of forward genetics

Marelize Snyman, Trung Huynh, Matthew T. Smith, Sen Xu

Department of Biology, University of Texas at Arlington, Arlington, TX

Forward genetic screening is a common strategy used for defining gene function through the production of mutants with an altered phenotype. In this work we performed the first mutagenesis experiments and whole-genome sequencing analysis in the microcrustacean *Daphnia* to estimate the whole genome mutation rate and spectrum induced by a commonly used mutagen, ethyl methanesulfonate (EMS). A total of 43 *Daphnia* isolates were exposed to two different EMS concentrations, 10mM and 25mM, and sequenced by Illumina with 150 bp paired-end reads. The mean base substitution rate for the 10mM treatment lines were 1.17×10^{-6} per base per generation and the 25mM treatment lines 1.75×10^{-6} per base per generation. Mutations were randomly distributed across the genome with the majority being G/C to A/T transitions, a characteristic of EMS induced mutations. The base substitution rate and spectrum was also analyzed between the first three consecutive broods of females exposed to either 10mM or 25mM EMS concentrations. Our results showed and increased base substitution rate for all three consecutive broods, and the spectrum remained constant and mirrored the previous results of an increased G/C to A/T transition rate. For the 25mM treatment lines, an average of 86 function affecting variants were induced per line, drastically reducing the number of mutants to be screened for a phenotype of interest. Our findings establish *Daphnia* as a model organism for mutation research and our proposed forward genetic screening approach paves the way to study novel and existing genotype-phenotype relationships.

Marelize Snyman

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Graduate Student
Contributed Papers Session I
Award Competition: Yes

PROTECTION OF TELOMERES 1b regulates chromatin structure to prevent genomic oxidative damage during development in *Arabidopsis thaliana*

Claudia Castillo-González, Borja Barbero, Ji-Hee Min, Sreyashree Bose, and Dorothy E. Shippen

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

Genome integrity requires an orchestrated response to environmental and cellular cues through highly conserved multifunctional proteins. Telomeres are the capping structures at the ends of chromosomes, indispensable for their role in genome integrity and cellular senescence. However, emerging studies indicate that telomere-associated factors may have a broader role in modulating the response to oxidative stress. PROTECTION OF TELOMERES 1 (POT1) is one of the most conserved telomeric proteins, essential for both chromosome end-protection and telomeric DNA replication. *Arabidopsis thaliana* harbors two highly divergent POT1 paralogs: POT1a which fulfills conserved telomeric roles, and POT1b. Here we explore the functions of POT1b. Unlike POT1a, POT1b is not a major player in telomere homeostasis, as loss of POT1b function did not lead to changes in telomere length. Instead, POT1b appears to play a unique role in redox biology. Plants null for POT1b were hypersensitive to chemical and environmental stresses, including temperature and salt; were sensitive to the absence of sucrose in the germination media; and, exhibited reduced fitness, as seen by defective pollen viability and lower seed biomass. To gain insight into the mechanism of POT1b, we combined transcriptomic, biochemical, genetic and cell biology assays. We found that POT1b expresses in seeds, roots, gametophytes, and flowers; moreover, it is upregulated by oxidative stresses, and the POT1b protein accumulates in the nucleus in clusters surrounding the nucleolus. Loss of POT1b leads to a global increase in endogenous reactive oxygen species. Within the nucleus we observed regions of abnormally relaxed chromatin during anaphase. The observed anomalies in genome architecture were accompanied by increased chromatin accessibility, decreased DNA methylation, increased transcription, and elevated DNA oxidation. All of these phenotypes were partially complemented by the expression of POT1b in the first generation, supporting the conclusion that POT1b functions as a regulator of the epigenome, and the alteration of epigenetic marks in pot1b mutants cannot immediately be restored to wild type status. We propose a model wherein POT1b regulates chromatin condensation and prevents genomic oxidative stress in tissues where endogenous reactive oxygen species are required for development.

Claudia Castillo-González, Ph.D.

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Postdoc
Contributed Papers Session I
Award Competition: Yes

Ethnicity-specific effects of DNA methylation-based biological age clocks

Talisa Silzer[1], Courtney Hall[1], Zhengyang Zhou[2], Jie Sun[1], Nicole Phillips[1], Robert Barber[1]

[1]Graduate School of Biomedical Science, University of North Texas Health Science Center, Fort Worth, TX;
[2]School of Public Health, University of North Texas Health Science Center, Fort Worth, TX

BACKGROUND. The global aging population (> 65 years of age) is growing rapidly. Advanced chronological age (CA) has long been associated with increased risk for chronic conditions such as hypertension, metabolic dysfunction, cancer and Alzheimer's disease. Other risk factors including genetics, environment and familial history also play a role. Given these factors, age-related disease risk and prevalence vary considerably across individuals and populations. A more informative measurement of aging may be 'biological age' (BA), which captures the state of an individual's biological and physiological well-being. Conceptually, individuals with an older BA in comparison to their CA, may be at higher risk for different age-related conditions. Epigenetic changes including alterations to DNA methylation (DNAm) at CG dinucleotides ('CpG sites') are known to occur throughout the aging process. Though several methodologies for estimating BA have emerged in recent years, utilization of genome wide DNAm data has become popular. While a growing number of studies have focused on the link between BA and disease among individuals of European ancestry, a knowledge gap currently exists for racially and ethnically diverse populations. SIGNIFICANCE. Investigating the performance of existing BA clock calculations in diverse populations will enhance our understanding of age-related disease risk across ancestral groups and will help promote equity in personalized science and medicine. PURPOSE. The goal of the study was to 1) identify differences in BA across ancestral groups and, 2) determine whether BA is predictive of cognition. METHODOLOGY. Phenotypic and genome-wide methylation data generated on the Illumina® MethylationEPIC array were obtained for 578 Non-Hispanic Whites (NHWs) and Mexican Americans (MAs) enrolled in the Texas Alzheimer's Research Care and Consortium (TARCC). BA was derived from DNAm data using two different clock calculations - Horvath and Hannum. RESULTS. Linear regression revealed ethnicity-specific effects of BA on cognition. Top CpG sites that were predictive of BA also varied between ethnic groupings (MAs vs. NHWs). FUTURE DIRECTIONS. Feature selection-based methods in a larger, diverse cohort will be ideal for discerning which CpGs are most predictive of BA across different ancestries. Investigating the relationships between BA and existing proteomic biomarkers for cognitive impairment will also be informative for understanding disease pathophysiology and may be used to guide personalized medicine in the future.

Talisa Silzer, M.S., Ph.D.

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Postdoc
Contributed Papers Session I
Award Competition: Yes

Fitness Effects of a Potentially Selfish Mitochondrial Deletion in *Caenorhabditis elegans*

Abigail Sequeira, Vaishali Katju, Ulfar Bergthorsson

Department of Veterinary Integrative Biosciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX

The ancient endosymbiotic event in which proto-mitochondria were taken up by proto-eukaryotic cells some two to three billion years ago has led to mitochondria playing a critical role in the evolution of eukaryotes by impacting fitness, speciation, evolution of sex, and disease. Mutations in the mitochondrial genome increase variation within populations of mitochondria and lead to heteroplasmies at the cellular, individual, and population level. Because the replication of mitochondrial DNA occurs separately from the nuclear DNA, it is possible for mitochondrial mutations that have a replicative or a transmission advantage to increase in frequency within individuals even if they result in individual fitness detriment. Such mutations would be selected against in large populations, but under conditions where the power of individual selection is diminished, such as in very small populations, selfish deleterious mitochondrial mutations may rise in frequency within individuals. This study explores the fitness effects of a deletion that arose spontaneously in the mitochondria during a mutation accumulation experiment that was conducted on *Caenorhabditis elegans* over several generations. One of the *C. elegans* MA lines incurred a 1,034 bp deletion (Δnd-4) in its mitochondrial genome, spanning the 3' end of cox-3, tRNA-thr and a large section of nd-4. This Δnd-4 mitochondrial genome rose to > 80% intracellular frequency over a few generations. This rapid rise in the frequency of a newly originated deletion-bearing mitochondria is consistent with a replicative or transmission advantage of the deletion over wildtype mitochondrial genomes. In order to test for fitness effects, the Δnd-4 mitotype was backcrossed into a wildtype nuclear background. The Δnd-4 mitochondrial genome significantly reduced productivity (64%), survivorship to adulthood (34%), longevity (26%) and developmental rate (29%). In competition experiments with worms containing wildtype mitochondrial genomes, the Δnd-4 mitotype went extinct in less than seven generations. We estimate that Δnd-4 reduced fitness by 23% in competition. This study illustrates how mitochondrial mutations and resulting heteroplasmies can affect fitness and population dynamics, as well as provides further insight into how mitochondrial disease arise and are maintained.

Abigail Sequeira

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Graduate Student
Contributed Papers Session II
Award Competition: Yes

The dopaminergic stress response limits dopamine oxidation and toxicity following mechanical stress by preventing cytosolic dopamine accumulation

Kielen R Zuurbier, Rene Solano Fonseca, Sonja LB Arneaud, Peter M Douglas

Department of Molecular Biology, UT Southwestern Medical Center, Dallas, TX

A blunt force impact to the head inflicts diffuse mechanical stress which propagates throughout the brain. Due to its diffuse nature it could be expected that cellular damage and death would be non-discriminatory among neuronal subtypes. However, our lab has identified dopaminergic neurons to be hypersensitive to mechanical stress in both murine and nematode models of blunt force impact. The molecular mechanism that underlies this hypersensitivity remains unknown. We hypothesize that rather than anatomical architecture or location, the inherent autoxidative chemistry of the neurotransmitter dopamine sensitizes dopaminergic neurons to degeneration and death. We show that the cytosolic presence of dopamine, causing dopamine autooxidation, is required to confer this hypersensitivity. To test if dopamine is sufficient to sensitize neurons to mechanical stress we sought to synthesize dopamine in a different, non-sensitive, neuronal subtype. Normally, serotonergic neurons are remarkably unaffected by mechanical stress but upon ectopic expression of the tyrosine hydroxylase, serotonergic neurons showed similar amounts of degeneration in our *C. elegans* trauma model. Additionally, we have identified an ancestral mechanism of neuronal adaption through transcriptional regulation of dopamine biosynthesis and packaging, which functions across metazoan and mammalian models in response to mechanical stress. This adaptive response prevents cytosolic dopamine oxidation by simultaneously downregulating dopamine synthesis while increasing dopamine metabolism and vesicular packaging. Future work will characterize this dopaminergic stress response and will identify the main transcriptional regulators of this response.

Kielen R Zuurbier

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Graduate Student
Contributed Papers Session II
Award Competition: Yes

Differential Regulation of Glycolytic Genes in Glioblastoma Multiforme

Shreya Udawant, Carl Litif, Bonnie Gunn, Megan Keniry

Department of Biology, University of Texas-Rio Grande Valley, Edinburg, TX

PI3K/AKT/mTOR signaling pathway and altered tumor metabolisms are significantly associated with Glioblastoma (GBM). PI3K/AKT/mTOR pathway reprograms the glucose metabolism to enhance aerobic glycolysis. Our understanding of glycolysis metabolism under PI3K/AKT/mTOR inhibition is limited. To investigate the perturbed glycolysis metabolism under PI3K/AKT/mTOR inhibition, we performed the RNA-sequencing (RNA-seq) of U87MG GBM cell lines in response to NVP-BEZ235. The differential expression analysis under control and NVP-BEZ235 treatments led to the identification of 7803 differentially regulated genes. We revealed that glycolysis-related gene sets were significantly enriched ($P < 0.05$) in control samples compared to NVP-BEZ235-treated samples through Gene Set Enrichment Analysis (GSEA). We also find that 17 and 35 genes were significantly downregulated ($P < 0.05$) in response to NVP-BEZ235 treatment in two different glycolysis related gene sets. Finally, we validated the expression of glycolysis-related genes, proteins, and metabolites in a set of GBM cell lines. Our findings suggest novel glycolytic changes through PI3K/AKT/mTOR inhibition. The identified genes serve as potential targets for developing targeted molecular therapies for GBM treatment.

Shreya Udawant

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Research Associate
Contributed Papers Session II
Award Competition: Yes

Determining the function of the transcription factor Cmr3 in wild type *Saccharomyces cerevisiae* and its role in the *spt4*Δ-mediated rescue of *pgm2*Δ phenotypes

Mandy Eckhardt[1], Sita Ramasamy[1], Keara D. Malone[1], Spencer L. Nystrom[2], Rachel V. Jimenez[1], Ashley Charales[1], Courtney D. Goldstein[1], Ruthann H. Schmiege[1], Madelyn G. Oliver[1], David P. Aiello[1]

[1]*Department of Biology, Austin College, Sherman, TX;*
[2]*Curriculum in Genetics and Molecular Biology, University of North Carolina at Chapel Hill, Chapel Hill, NC*

Phosphoglucomutase (PGM) is an enzyme responsible for the interconversion of the metabolites glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P) in *Saccharomyces cerevisiae*. A mutant yeast strain lacking *PGM2*, the major isoform of PGM, exhibits several defective phenotypes when the cells are grown on galactose media. These phenotypes include slow growth, high levels of G1P relative to G6P, and increased Ca^{2+} uptake and accumulation. EMS mutagenesis was utilized to isolate mutant alleles that rescue *pgm2*Δ growth defects. *SPT4*, which encodes a transcription elongation factor, was identified through this screen. All calcium related phenotypes observed in the *pgm2*Δ mutant are rescued in the *pgm2*Δ*spt4*Δ double mutant. We undertook an RNAseq analysis with the goal of identifying candidate genes that show differential expression between the wild type and *pgm2*Δ*spt4*Δ strains relative to the *pgm2*Δ strain that contribute to *pgm2*Δ mutant phenotypes, or mediate rescue in the *pgm2*Δ*spt4*Δ strain. Analysis of this dataset suggests the *pgm2*Δ mutation causes cells to hyperactivate a variety of cellular stress response pathways. Further, the DREME analysis tool has identified the transcriptional activator, Cmr3, whose binding sequence shows increased representation in the promoters of genes exhibiting differential expression between wild type, *pgm2*Δ, and *pgm2*Δ*spt4*Δ strains. The *pgm2*Δ*cmr3*Δ strain shows slower growth on galactose media, indicating that *CMR3* likely contributes to the viability of the *pgm2*Δ mutant. Eight genes have been identified that have altered gene expression in the *pgm2*Δ mutant and are potentially regulated by Cmr3. Current efforts seek to determine the roles of these genes in the survival of the *pgm2*Δ strain, and chromatin immunoprecipitation is being utilized to determine if Cmr3 is binding to the promoter regions of these genes.

Mandy Eckhardt

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Undergraduate Student
Contributed Papers Session III
Award Competition: Yes

Cleavage Efficiency of Flavivirus NS3 Proteases and Protease Recognition Sequences in *Aedes aegypti* Cells

Alexius Dingle[1], Bianca Kojin[2], and Zachary N. Adelman[2]

[1]*Department of Entomology and Interdisciplinary Program in Genetics, Texas A&M University, College Station, TX*
[2]*Department of Entomology, Texas A&M University, College Station, TX*

Aedes aegypti is responsible for the transmission of dengue, Zika, and yellow fever viruses, all members of the Flavivirus genus. The flavivirus genome is a single stranded positive sense RNA of ~11 kb that encodes three structural proteins and seven nonstructural proteins translated as one long polyprotein that is cleaved by both cellular and viral proteases. NS3 is multifunctional, acting as a serine protease and helicase. We seek to take advantage of the proteolytic activity of NS3 by developing a transgene that will result in the virus-induced death of *Aedes aegypti*. Our desired construct will express an insect specific neurotoxin in the mosquito's nervous system. The toxin peptides will be tethered to the endoplasmic reticulum by an intervening NS3 protease target sequence, only to be cleaved away by the protease in mosquitos that have developed a systemic infection. The aim of this project is to test the cleavage efficiency of each NS3 target site by proteases from several flaviviruses in *A. aegypti* cells. The most widely recognized sequence will be incorporated into our final transgene construct. We hypothesize that the internal NS3 and NS4A target site sequences will allow for optimum cleavage by a greater number of proteases due to their high level of conservation. To determine cleavage efficiency, we designed a reporter plasmid that expresses an ER-tethered, eGFP-mCherry fusion protein. NS3 target sites were inserted at the eGFP-mCherry junction. A T2A peptide was incorporated for the separate translation of the NS2B-NS3 protease complex. With no protease present, we expect to see green and red fluorescence colocalized at the ER. When NS3 is expressed we expect to observe a decrease in co-localization with proportionally more green fluorescence in the cytoplasm. A20 cells were transfected with the reporter plasmid, stained with ER Tracker Blue-White reagent, and imaged after 48 hours, confirming correct localization of the fluorescent proteins to the ER membrane. Western blot analysis confirmed independent expression of the viral protease. We then proceeded with transfections of each test plasmid and imaging at 24, 48, and 72 hours, using these images for colocalization analyses. Critically, we have observed a decrease in colocalization with at least one target site. Our results indicate that we can successfully observe NS3 proteolytic activity in vivo, therefore making this an effective method for evaluating NS3 target site cleavage. This should allow us to create a death upon infection transgene that can kill mosquitos infected with any flavivirus, effectively selecting for those insects that are resistant to infections and viruses that replicate slower or to lower levels.

Alexius Dingle

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Graduate Student
Contributed Papers Session III
Award Competition: Yes

Calcium ions trigger the exposure of phosphatidylserine on the surface of necrotic cells

Yoshitaka Furuta, Omar Pena-Ramos, Zao Li, Lucia Chiao, Zheng Zhou

Baylor College of Medicine, Houston, TX

Intracellular Ca^{2+} level is under strict regulation through calcium channels and storage pools including the endoplasmic reticulum (ER). Mutations in certain ion channel subunits, which cause mis-regulated Ca^{2+} influx, induce the excitotoxic necrosis of neurons. In the nematode *Caenorhabditis elegans*, dominant mutations in the DEG/ENaC sodium channel subunit MEC-4 induce six mechanosensory (touch) neurons to undergo excitotoxic necrosis. These necrotic neurons are subsequently engulfed and digested by neighboring hypodermal cells. We previously reported that necrotic touch neurons actively expose phosphatidylserine (PS), an "eat-me" signal, to attract engulfing cells. However, the upstream signal that triggers PS externalization remained elusive. Here we report that a robust and transient increase of cytoplasmic Ca^{2+} level occurs prior to the exposure of PS on necrotic touch neurons. Inhibiting the release of Ca^{2+} from the ER, either pharmacologically or genetically, specifically impairs PS exposure on necrotic but not apoptotic cells. On the contrary, inhibiting the reuptake of cytoplasmic Ca^{2+} into the ER induces ectopic necrosis and PS exposure. Remarkably, PS exposure occurs independently of other necrosis events. Furthermore, unlike in mutants of DEG/ENaC channels, in dominant mutants of deg-3 and trp-4, which encode Ca^{2+} channels, PS exposure on necrotic neurons does not rely on the ER Ca^{2+} pool. Our findings indicate that high levels of cytoplasmic Ca^{2+} are necessary and sufficient for PS exposure. They further reveal two Ca^{2+}-dependent, necrosis-specific pathways that promote PS exposure, a "two-step" pathway initiated by a modest influx of Ca^{2+} and further boosted by the release of Ca^{2+} from the ER, and another, ER-independent, pathway. Moreover, we found that ANOH-1, the worm homolog of mammalian Ca^{2+}-dependent phospholipid scramblase TMEM16F, is necessary for efficient PS exposure in thapsgargin-treated worms and trp-4 mutants, like in mec-4 mutants. Altogether, we propose that both the ER-mediated and ER-independent Ca^{2+} pathways promote PS externalization through activating ANOH-1.

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Graduate Student
Contributed Papers Session III
Award Competition: Yes

Poster Titles and Numbers

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Hyperlink in the poster title will take you to the poster's abstract
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Minal Jamsandekar	How large inversions contribute to ecological adaptation in Atlantic herring	23	5
Shalini Nair	Media composition and fitness costs of antimalarial drug resistance mutations in *Plasmodium*	24	6
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Austin Daigle and Robert Melde	The Phenotypic and Genomic Consequences of Transposable Elements in *C. elegans* Bergerac strains	37	8
Katherine E. McBroom	Examining growth phenotypes of the pgm2Δ mutation in *Saccharomyces cerevisiae* lacking or overexpressing *TPS1*, *NTH1*, and *ATH1*	38	8
Harsha Tamtam	Examining the effects of extracellular Ca^{2+} and Methylglyoxal on HACS and LACS mutants in *S. cerevisiae* mutants lacking *PGM2*	39	9
Tina L. Gumienny	The DBL-1/TGF-β signaling pathway regulates pathogen-specific innate immune responses in *C. elegans*	40	9
Rebekah Napier-Jameson	RNA binding proteins coordinately control lifespan in *C. elegans*	41	9
Razan Fakieh	The RAP-2 Small GTPase and MIG-15 MAP4 kinase promote tertiary fate in *C. elegans* VPC Patterning	42	9
Sudip Mondal	An ultrahigh-density platform for large-scale in vivo phenotypic screens using *C. elegans* models	43	10

Presenter	Title	Poster Number	Breakout room
Brandon Meadows	An Investigation Using 16S rRNA Gene Sequencing To Characterize The Microbial Taxa Present In Different Body Compartments Of Carpenter Ants	44	10
Elyssa Garza	Using Genomic Strategies to Identify Serpentine Stress Tolerance QTL	45	10
Sydney Versen	Identifying Novel Developmental Genes Using the Undiagnosed Disease Network	46	10

Poster Abstracts

Hyperlink in presenter's name will take you to an uploaded video presentation of the poster

Not all centromeres are equal, or are they?

Michelle M. Jonika[1,3], Sarah N. Ruckman[1,2], Claudio Casola[2,3,4], Heath Blackmon[1,2,3]

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Despite the fundamental role of centromeres two different types are observed across plants and animals. Monocentric chromosomes possess a single region that function as the centromere while in holocentric chromosomes centromere activity is spread across the entire chromosome. Proper segregation may fail in species with monocentric chromosomes after a fusion or fission, which may lead to chromosomes with no centromere or multiple centromeres. In contrast, species with holocentric chromosomes should still be able to safely segregate chromosomes after fusion or fission. This along with the observation of high chromosome number in some holocentric clades has led to the hypothesis that holocentricity leads to higher rates of chromosome number evolution. To test for differences in rates of chromosome number evolution between these systems, we analyzed data from 4,393 species of insects in a phylogenetic framework. We found that insect orders exhibit striking differences in rates of fissions, fusions, and polyploidy. However, across all insects we found no evidence that holocentric clades have higher rates of fissions, fusions, or polyploidy than monocentric clades. Our results suggest that holocentricity alone does not lead to higher rates of chromosome number changes. Instead, we suggest that other co-evolving traits must explain striking differences between clades.

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Graduate Student

Poster #: 1
Poster Session: 1
Breakout Room: 1
Award Competition: Yes

'Molecular Switch' Regulates Mitochondrial Surveillance and Immunity Pathways

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Mitochondria perform crucial cellular functions and their dysfunction contributes to a wide variety of pathologies, including neurodegenerative diseases, cancer, and metabolic diseases, and aging. Active monitoring of mitochondrial status provides information about the general health of the cell to prevent the loss of cellular viability. This task is performed by mitochondrial surveillance or quality control pathways, which are activated by signals originating from mitochondria and relayed to the nucleus (retrograde response) to start the transcription of protective genes. In a model nematode *Caenorhabditis elegans*, several systems exist, including the mitochondrial unfolded protein response (UPRmt), mitochondrial MAPK cascade (MAPKmt), and the Ethanol and Stress Response Element (ESRE) pathway. These pathways are highly conserved and their loss results in compromised survival following mitochondrial stress.
In our study, we discovered that the ESRE pathway is robustly activated in response to increased reactive oxygen species (ROS) concentration. The direct linear relationship between ROS, particularly superoxide, and ESRE activation differentiated ESRE from other mitochondrial surveillance pathways, such as the UPRmt, which largely monitors mitochondrial protein import. We also identified multiple interactions between mitochondrial surveillance pathways, suggesting a partially redundant hierarchy. For example, when the UPRmt pathway was mutated, activation of the ESRE pathway occurred earlier and reached higher levels. Compromising the UPRmt also significantly increased expression of a mitochondrial surveillance pathway that is regulated by PMK-3/MAPK (MAPKmt).
Furthermore, we found a novel interaction between the Box C/D snoRNA core proteins (snoRNPs) with mitochondrial surveillance and innate immunity pathways. We showed that C/D snoRNPs are required for the full expressions of UPRmt and ESRE upon stress. Meanwhile, we found that the loss of C/D snoRNPs increased immune responses. Thus, we proposed that the Box C/D snoRNPs act as a molecular switch that activate quality control pathways while inhibiting immune responses.
The ESRE motif, the mitochondrial surveillance and innate immune systems, and the Box C/D snoRNPs are conserved in humans. Their roles in modulating cell biology during mitochondrial crises is essential for mitigating stress and restoring health after damage. A better understanding of these networks and interactions, and their roles in the constellation of mitochondrial and cellular stress networks, may be important for the understanding of multifactorial processes, including response to infection or aging.

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Postdoc
Poster #: 3
Poster Session: 1
Breakout Room: 1
Award Competition: Yes

Investigation of Differentially Expressed Genes Associated with PI3K inhibition in GBM

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Glioblastoma Multiforme (GBM) is an aggressive, malignant brain tumor with poor prognosis and limited treatment options of life-threatening tumor resection surgery and harmful radiation. However, novel gene therapy treatments that target differentially expressed genes in cell signaling pathways may have potential in treating this metastatic tumor. The phosphoinositide 3-kinase (PI3K) pathway is of interest as it has been known to have upregulated activity in GBM and may lead to GBM proliferation. Therefore, the Keniry Laboratory inhibited the PI3K pathway and studied the differentially expressed genes, Kaplan Meier survival curves, and Pearson Correlations. From these analyses, IGFBP3 is of interest for its upregulation in GBM, association with clinical prognosis and significant correlation to other genes.

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Undergraduate Student
Poster #: 4
Poster Session: 1
Breakout Room: 1
Award Competition: Yes

Investigating the role of Crz1p and Rlm1p on altered gene expression in *Saccharomyces cerevisiae* mutants lacking *PGM2*

Ruthann H. Schmiege[1], Madelyn G. Oliver[1], Keara D. Malone[1], Spencer L. Nystrom[2], Rachel V. Jimenez[1], Mandy R. Eckhardt[1], Paul Mpunga[1], Courtney D. Goldstein[1], Ashley Charales[1], David P. Aiello[1]

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In *Saccharomyces cerevisiae*, the enzyme phosphoglucomutase (PGM) facilitates the interconversion of Glucose-1-Phosphate (G1P) and Glucose-6-Phosphate (G6P). For cells grown on galactose-containing media, the loss of the major isoform of phosphoglucomutase, encoded for by *PGM2*, results in increased G1P relative to G6P when compared to wild-type (wt) cells. In addition, *pgm2*Δ mutants show defects in calcium homeostasis and sensitivity to cyclosporin A. The current working model of the lab hypothesizes that the altered G1P to G6P ratio results in abnormal uptake and accumulation of calcium within the cell which, in turn, hyperactivates various stress signaling pathways that alter gene expression. A list of genes that showed altered gene expression between wt and *pgm2*Δ strains, compiled using RNA-Seq and DESEQ-2 analysis, was analyzed using DREME analysis to determine potential transcription factors that might be coordinately regulating subsets of these genes. Crz1p and Rlm1p were identified as potential transcriptional activators involved in upregulating a number of these genes. Crz1p is the major effector of calcineurin-driven gene expression while Rlm1p is a mediator of the cell integrity MAPK pathway. To experimentally confirm the proposed Crz1p- and Rlm1p-mediated upregulation of genes in the *pgm2*Δ mutants, this project is using chromatin immunoprecipitation (ChIP) to confirm the presence of these transcription factors at the promoters of genes of interest.

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Undergraduate Student
Poster #: 6
Poster Session: 1
Breakout Room: 2
Award Competition: Yes

The Impacts of Pesticide Exposure on Zebrafish Development

Mia Jin Hibner, BIOL 324 Developmental Biology Class Fall – 2020, Kelli J. Carroll

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Pesticides have emerged as a global public health concern due to their wide usage and the frequency of human exposure. Pesticides are used to control and limit a wide variety of organisms such as weeds, insects, and other small pests. Therefore, they are found in numerous places including groundwater and as residues on crops. Understanding the effects these chemicals have on human development is crucial as humans frequently come into contact with them. Zebrafish are an ideal model organism to study developmental biology because of their rapid and transparent development. In order to identify specific pesticides for future study, we used a list from the Environmental Protection Agency of the fifty most commonly used pesticides in the USA, with an emphasis on those that had a potential effect on neural development and function. The top pesticides of interest are Glyphosate, Atrazine, Paraquat, Dicamba, Methyl Bromide, Bifenthrin, Malathion, Metolachlor, Chloropicrin, and Pendimethalin. For each pesticide, zebrafish studies as well as studies using other model organisms were utilized to provide important background information on dosing and morphological abnormalities seen after embryonic exposure. In addition, case studies of high-dose human exposure provided information on the effect these pesticides have on adult physiology. Future work for this project will include an initial characterization of the top 10 pesticides of interest through the generation of dose curves, acute embryonic zebrafish exposure experiments, and initial qPCR-base screening to assess organ development after drug treatment. Promising candidate drugs will later undergo a deeper analysis with behavioral assays and an in-depth characterization of organogenesis, as well as identification of the modes of action for each drug. Lastly, combinatorial treatments may also be conducted in the future, as pesticides are rarely found in isolation in the environment.

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Undergraduate Student
Poster #: 7
Poster Session: 1
Breakout Room: 2
Award Competition: Yes

The Post-Transcriptional Regulator Musashi Binds Sonic Hedgehog mRNA In The Developing Mouse Palate

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Mammalian palatogenesis requires a high degree of molecular coordination. Palatal shelves must elevate, symmetrically extend, and fuse accurately at the midline for complete soft and hard palate formation. The protein sonic hedgehog (SHH) is expressed in the developing palate and is thought to be essential for palate elongation. Sonic hedgehog is primarily expressed in palatal rugae, signaling centers that are thought to choreograph palate growth. Diffusion of SHH from rugae induces a mitogenic response of inter-rugal cells, promoting tissue proliferation and growth of the palatal shelves. Due to the highly morphogenic potential of SHH, its production must be strictly controlled, and restricted to growth zones. Examination of museum specimens of bat species with different facial morphologies revealed a correlation between palatal rugae number and inter-rugal distance with rostral length, suggesting that rugae establishment and SHH signaling may be important in rostrum length evolution.
The RNA-binding protein musashi (MSI) is thought to post-transcriptionally regulate gene expression through binding RU1-3AG mRNA motifs, which appear three times within the mouse Shh 3'UTR. Immunofluorescence staining of mouse embryos has identified the confinement of MSI to palatal rugae. Also, immunoprecipitation qPCR (RIP-qPCR) with MSI antibodies has confirmed that MSI binds Shh mRNA in the palate during embryogenesis (stage E14.5). Because MSI binds Shh mRNA in the developing palate, we hypothesize that Shh is post-transcriptionally regulated by MSI. The dynamics of this association will be evaluated through in-vitro gene expression assays involving site-directed mutagenesis of MSI-binding motifs, and siRNA knockdown of Msi, to determine the degree and directionality of expression change induced by MSI binding activity.

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Graduate Student
Poster #: 8
Poster Session: 1
Breakout Room: 2
Award Competition: Yes

Biomarker Discovery for Angelman Syndrome Therapeutics: The Role of Extracellular Vesicles

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Angelman Syndrome (AS) is a neurogenetic disorder that arises from the loss of maternal UBE3A in central nervous system (CNS) neurons. An antisense oligonucleotide (ASO) therapy to treat the disorder is in clinical trials; however, questions remain about the proper dosage protocol to maximize efficacy while minimizing toxicity. An efficacy biomarker has not yet been identified due to the challenges associated with studying the state of CNS neurons specifically. Extracellular vesicles (EVs) are currently being explored as potential biomarkers for several neurological disorders because they have cell-type-specific markers, contain bioactive molecules, and cross the blood-brain barrier. In this study, we characterized the contents of EVs isolated from serum of a pig model of AS to identify potential biomarkers for AS therapeutics. We first characterized EVs following ISEV guidelines to promote reproducibility and reliability. We then used RNA sequencing to characterize RNA transcripts in EVs isolated from pig serum. We identified mitochondrial RNA (mtRNA) transcripts associated with mitovesicles, a recently described subpopulation of EVs. There is evidence of mitochondrial dysregulation in Angelman Syndrome, thus an efficacy biomarker may be present within mitovesicles. More sensitive RNA and protein detection methods are being investigated to promote the identification of potential biomarkers within these vesicles. Additional studies are ongoing to characterize the transcriptome of pig EVs and to identify potential biomarkers for different AS therapeutics.

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Graduate Student
Poster #: 9
Poster Session: 1
Breakout Room: 2
Award Competition: Yes

Relatives of Ras regulate function of the *C. elegans* exocyst complex in development

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Among three Ras effectors of roughly equivalent oncogenicity, Raf and PI3K are well studied but the downstream mechanisms of Ras-RalGEF-Ral signaling remain poorly understood. Ral (Ras-like) is a small GTPase related to Ras. The exocyst is a heterooctameric protein complex that mediates the targeting and tethering of transport vesicles to the plasma membrane. Ral uses the exocyst complex as a signaling intermediary (as exocyst components-Exo84 and Sec5 are Ral effectors) and also performs essential activities to regulate exocytosis functions of the exocyst, which precludes conventional biochemical bootstrapping to identify signal transduction components downstream of the exocyst. Rap1 (Ras proximal) is another small GTPase that sometimes signals in parallel with Ras. Delineating the mysterious functions of Ral in signaling and Ral and Rap1 in functions of the exocyst are important for therapeutic targeting of oncogenic Ras and understanding the cell biological functions of the exocyst complex.
We are using genetic, biochemical and cell biological analyses in *C. elegans* to identify roles of Ral and Rap1 in control of exocyst functions during development. The three aims of this project are: 1. Determine the contribution of Ral to functions of the exocyst complex. 2: Investigate whether the Ral signaling function works to regulate exocytic functions of the exocyst separately from the signaling-independent functions. 3: Delineate Rap1 contributions to exocyst functions.
We found that Ral is needed for the exocyst function, as deletion of Ral aggravates developmental defects conferred by maternally rescued (M+Z-) deletion of Sec5. Yet signaling-defective Ral does not alter the phenotype of M+Z- Sec5 mutant animals. Furthermore, constitutively activated Ral reduces the severity of M+Z- Sec5 mutant defects, implicating signaling-dependent and -independent contributions of Ral to exocyst-dependent development. Rap1 similarly interacts with the M+Z- Sec5 mutant and the M+Z- Ral mutant, suggesting a model similar to that of Ral. We will continue to test the contributions of Ral and Rap1 to exocyst-dependent development using the chemogenetic tool Auxin-Inducible degron (AID). We will also use the development of elaborate arborization of the axons of PVD neurons as a more precise readout of exocyst function, and also assay the impact of Sec5, Ral and Rap1 depletion on the transport of marked exocytic cargoes. Besides, we will use biochemical tools (Co-immunoprecipitation, yeast-two-hybrid) and will do confocal imaging to measure the physical interaction and colocalization of HA and fluorescent tagged Ral and exocyst components.
In conclusion, this study will further investigate the role of two Ras relatives, Ral and Rap1, in the exocyst. This will help delineate the downstream effectors of the poorly understood Ras>RalGEF>Ral pathway and help better understand the relationship of Rap1 with the exocyst, as well as the crosstalk of Rap1 and Ras effectors.

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Graduate Student
Poster #: 10
Poster Session: 1
Breakout Room: 2
Award Competition: Yes

Effects of Tart Cherry on Lifespan of a GMC 101 *C. elegans's* Strain for Alzheimer's Disease

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Background: Alzheimer's disease (AD) is a neurodegenerative disorder characterized by the deposition of β-amyloid plaques and tau protein in the brain. AD risk factors include age and chronic diseases such as obesity and is characterized by neuroinflammation and oxidative stress. Tart cherry (TC) is a rich source of anthocyanin, and exhibits anti-inflammatory and antioxidative activities, which may result in beneficial effects in AD. Nematode *Caenorhabditis elegans* (*C. elegans*) is a prominent model organism for studying aging-related diseases due to their characteristics of free-living, non-parasitic and a short life span of 3 weeks. We previously demonstrated that TC increases lifespan of wild type N2 *C. elegans*, by enhancing mitochondrial function and anti-oxidant markers when adding 6 or 12 µg/ml of TC supplementation to the worm diet. We hypothesized that TC will increase lifespan in the *C. elegans*' AD strain and will provide a preventative treatment option for AD. In this experiment, we use transgenic GMC 101 *C. elegans* strain with a expression of human β-amyloid to investigate the beneficial effects of TC and its role in aging-related disease
Methods: Age synchronized *C. elegans* AD strain were loaded into microfluidic devices and received 20 mg/ml of *E. coli* OP50 and one of the following treatments: control (no supplementation), 6 and 12 µg of anthocyanin/ ml of TC extract. A 90 second video was recorded for each treatment group daily, followed by image analyses using Infinity software to determine the number of live worms. Median lifespan of worms was determined using Prism Graph-Pad.
Results: Experiments are currently ongoing. Preliminary data demonstrates that TC supplementation at 6 and 12 µg/mL extended median lifespan of GMC 101 *C. elegans* by 15.4% (15 days) and 19.2% (15.5 days, $p < .05$), respectively, when compared to control worms (13 days).
Conclusion: Our outcomes indicate that TC may have health benefits in *C. elegans* via increasing their lifespan, which may provide an efficient approach for combating aging-related diseases, like AD. We plan in future to investigate genetic pathways mediate the longevity, like insulin/insulin-like growth factor-1 and the β-amyloid modification.
Funding: USDA NIFA AFRI award # 2018-07980, Pi2 program and Undergraduate Funds from the College of Human Sciences

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Undergraduate Student
Poster #: 11
Poster Session: 1
Breakout Room: 3
Award Competition: Yes

Determining a role for *CMR3* in regulating gene expression due to stress responses caused by the *pgm2*Δ mutation

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Transcriptional elongation factors play an important role in regulating gene expression. Spt4 is a transcriptional elongation factor that plays an important role in transcriptional pausing in mammalian cells, and in regulation of transcription through long trinucleotide repeats in *Saccharomyces cerevisiae*. Previous work in the lab identified the loss of *SPT4* as a suppressor of *pgm2*Δ defects through an EMS mutagenesis screen. Yeast which lack *PGM2*, the major isoform of phosphoglucomutase, lose the ability to interconvert glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P), and exhibit a variety of growth defects when grown in galactose-containing media. The most relevant of these include slow growth, imbalanced levels of G1P relative to G6P, high levels of intracellular Ca^{2+}, and induction of the unfolded protein response (UPR), which is hypothesized to result from reduced ER calcium levels. Deletion of *SPT4* was shown to rescue the Ca^{2+}-related growth defects in the *pgm2*Δ background, but this rescue is indirect. Data collected by RNASeq have shown that Spt4 is important in regulating the expression of genes mediating stress responses such as those created by the imbalanced levels of G1P:G6P found in *pgm2*Δ mutants when grown in galactose. Using bioinformatics tools analyzing our RNASeq data, we have identified various target genes, and transcription factors predicted to coordinately regulate subsets of these stress-induced genes. These are hypothesized to be essential for viability in *pgm2*Δ mutants. Many of these genes exhibit increased expression in the *pgm2*Δ mutant compared to wild type and/or *pgm2*Δ*spt4*Δ strains. Current work in the lab shows that loss of *CMR3*, a putative zinc-finger protein of unknown function and transcription factor identified from RNASeq data, exacerbates the slow-growth phenotype of *pgm2*Δ mutants, implicating a role promoting in their survival. Current work investigates changes in gene expression in *pgm2*Δ and *pgm2*Δ*cmr3*Δ mutants to confirm target gene regulation by Cmr3 and to examine the hypothesis that these stress-induced genes promote the survival of the *pgm2*Δ mutant.

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Undergraduate Student
Poster #: 12
Poster Session: 1
Breakout Room: 3
Award Competition: Yes

Genetic Basis of Variation in Pigmentation Patterns and Feather Morphology in Chickens

McKaela Hodge

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There are few known naturally occurring examples of biological regeneration, particularly with the accuracy and precision of feather regeneration. Birds vary in plumage pigmentation and shape between species and even within a single individual depending on the body region, but the underlying genetics and the cellular regulation of feather production is not well understood. Our research focuses on identifying the mechanisms affecting the variation in avian pigmentation patterns and how cells interact to direct variation in feather morphology. Single cell RNA sequencing was performed on growing feather follicles in a Red Jungle Fowl (RJF) rooster from four different body regions: breast, hackles, leg, and saddle. A small section of feathers was plucked from each section and 10 mm biopsies were taken when these regions displayed feather buds. Expression of HoxC8 and HoxC10 was found to be nearly absent in hackles compared to the three other regions. The dorsal region displayed higher expression of HoxA9, HoxA7, and HoxC10. This is notable because we previously documented that HOXC10 is implicated in feather shape control based on body region: an intronic 198bp tandem duplication in HOXC10 is associated with the production of dorsal-like feathers in the cranial region. The findings of a chicken embryo development study also found that HoxC8 and HoxC10 were absent in crown and neck skin, but highly expressed in the anterior trunk, which is consistent with our data that there is absent expression of these genes in the hackles region and increased expression in the dorsal region. RJF roosters display reddish brown feathers in the dorsal region and that region showed low expression of the genes SOX10 and TYRP1. Pheomelanin is the melanin type that produces red or yellow coloration. Downregulation of SOX10 has been associated with increased pheomelanin and reddish phenotypes in other studies. SOX10 also regulates expression of TYRP1, which is necessary for eumelanin production, but is not needed for pheomelanin production. We hypothesize that within the dorsal region, the reddish feathers is due to increased pheomelanin production compared to the other body regions because SOX10 is being downregulated and decreasing the expression of TYRP1. These preliminary findings identify differences in gene expression between body regions, within the same cell type. Further work is ongoing to capture the variation in expression within and between body regions.

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Graduate Student
Poster #: 13
Poster Session: 1
Breakout Room: 3
Award Competition: Yes

Whole Genome Sequencing of *Escherichia coli* Surrogate Strains: Comparison of Methodologies and Application for the Food Industry

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Gene expression has long been appreciated for its integral role in phenotypic evolution. To ascertain the effect of selection on global gene expression, we evolved *Caenorhabditis elegans* lines under a range of selective constraints by bottlenecking each subsequent generation at population sizes of N = 1, 10, or 100 individuals. We found significant increases in the residual variance (Vr), a proxy for environmental variance, and mutational variance (Vm), the rate increase of variance in gene expression due to mutations in each generation in the N = 1 lines evolving under strong genetic drift. Interestingly, this correlation was reversed for mutational heritability (h_m^2), which was determined to possess a positive correlation with population size. This was owing to a disproportionate increase in residual variance, Vr, in the N = 1 lines relative to the N > 1 MA lines. This suggests that mutations resulting in increased sensitivity to microenvironmental and transcriptional noise due to dysregulation or decanalization of gene expression are accumulating to the greatest degree under conditions of extreme genetic drift, and to a lesser degree in MA lines with larger population bottlenecks (and greater efficiency of selection). Decanalization is often portrayed as increasing evolvability by revealing cryptic genetic variation. Conversely, decanalization may also decouple genotype and phenotype by increasing microenvironmental sensitivity and noise in gene expression to such a degree that natural selection becomes less effective in the short-term. Significant increases in expression variability were identified within all five broadly defined chromatin domains in *C. elegans* experimental lines with the most extreme increases found for the N = 1 lines. The greatest and smallest increase in the Coefficient of Variation for gene expression was in domains associated with broad gene silencing and active transcription, respectively. Analysis of differentially expressed genes elucidated a pattern of underexpressed genes in all experimental populations; however the ratio of overexpressed to underexpressed genes was significantly greater in lines governed by genetic drift. Furthermore, the preponderance of overexpressed genes was especially pronounced in genes involved in mitochondrial respiration, stress response, and immune system pathways. These results indicate that selection plays an active role in maintaining genetic networks, even in the face of considerable variation brought on by spontaneously arising mutations.

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Graduate Student
Poster #: 14
Poster Session: 1
Breakout Room: 3
Award Competition: Yes

Identifying the role of BRCA1 in transcriptional regulation using *Caenorhabditis elegans*

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Breast cancer susceptibility gene 1 (BRCA1) and its heterodimeric partner BARD1 play an essential role in genomic stability by regulating DNA damage repair, cell-cycle checkpoints, and transcription regulation. Germline mutation in either of these genes exposes individuals to a higher risk of developing breast and ovarian cancer. The *Caenorhabditis elegans* orthologs, brc-1 and brd-1, also regulate DNA damage repair and cell cycle checkpoints; however, their role in regulating gene transcription is still unknown. Here, we show the transcriptional regulation function of brc-1 and brd-1 is conserved in worms using the cyp-13A subfamily of genes, which are the homolog of a human estrogen metabolizing gene CYP3A4. Using gene expression analysis, we found that knocking out brc-1 resulted in significant upregulation of four cyp-13A subfamily of genes, and loss of brd-1 function led to upregulation of six cyp-13A subfamily of genes. Our finding provides insights into how brc-1/brd-1 transcriptional regulation function is conserved in worms and further validates using *C. elegans* as a model system to investigate BRCA1 functions.

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Graduate Student
Poster #: 15
Poster Session: 1
Breakout Room: 4
Award Competition: Yes

Astrocyte-mediated glycolytic preconditioning via electron chain deficits mitigates trauma-induced neurodegeneration

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Selective vulnerability of different neuronal subtypes to dysfunction, degeneration and death underlies all neurological diseases. Differential interplay between genetic and environmental factors may contribute to this neuronal selectivity. Environmental stimuli have the capacity to trigger pathogenic cascades contingent on genetic factors. Such is the case for traumatic brain injury (TBI) and concussion, in which the initial biomechanical insult precipitates numerous cellular and systemic alterations with the potential to promote neurological dysfunction and progressive neurodegeneration. Yet the molecular mechanisms and genetic targets promoting the selective vulnerability of different neural subtypes to dysfunction and degeneration remain unclear in part due to the stochastic nature of the mechanical insult. Immediately following injury, rodent brains display signs of decreased oxidative phosphorylation that can persist for weeks. Conversely, the injured brain transiently elevates normoxic glycolysis in humans and animal models. The astrocyte-neuron lactate shuttle (ANLS) is a responsive process that entails astrocytic production and transport of the glycolytic by-product, lactate, to neurons which preferentially oxidize it rather than glucose to meet their energetic demands. This metabolic shift is similar to a phenomena observed in cancer cells and is termed the Warburg effect, in which aerobic glycolysis is favored over oxidative phosphorylation. Thus, the injured brain initially undergoes a Warburg-like response, but how this metabolic shift occurs within the astrocyte-neuron axis and its ability to impact neurological function and degeneration after concussion is not well understood. By translating experimental models of blunt force trauma in *C. elegans* to concussion in mice, we identify a conserved neuroprotective mechanism in which reduction of mitochondrial electron flux through complex IV suppresses trauma-induced degeneration of the highly vulnerable dopaminergic neurons. Reducing cytochrome C oxidase function elevates mitochondrial-derived reactive oxygen species, which signal through the cytosolic hypoxia inducing transcription factor, Hif1α, to promote hyperphosphorylation and inactivation of the pyruvate dehydrogenase, PDHE1α. This critical enzyme initiates the Warburg shunt, which drives energetic reallocation from mitochondrial respiration to astrocyte-mediated glycolysis in a neuroprotective manner. These studies demonstrate a conserved process in which glycolytic preconditioning suppresses Parkinson-like hypersensitivity of dopaminergic neurons to trauma-induced degeneration via redox signaling and the Warburg effect.

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Postdoc
Poster #: 16
Poster Session: 1
Breakout Room: 4
Award Competition: Yes

Dissecting the Sequential Evolution of a Selfish Mitochondrial Genome in *Caenorhabditis elegans*

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Despite 1.4 billion years of endosymbiosis, mitochondria maintain many copies of their own genomes and replicate independently of the cell cycle. This independence permits selfishly-acting mitochondrial genomes to arise, which outcompete other mitotypes and increase their own intracellular frequency regardless of neutral or deleterious effects on their host cell. While there are many theories about what factors contribute to the advantage of one mitotype over another, mitochondrial genomes are challenging to engineer, thereby preventing experiments aimed at distinguishing among the various theoretical predictions. Previously, we discovered a selfishly acting mitochondrial genome that arose spontaneously in a line of *Caenorhabditis elegans* following 346 consecutive generations of experimental evolution, and comprising a large deletion, two indels, and a missense mutation. The regular cryopreservation of the experimental line at several time intervals during its evolution provided an opportunity to dissect the sequential origin and fitness effects of the various mutations comprising this selfish mitochondrial genome. First, we sought to investigate whether subsequent mitochondrial DNA mutations compensate for the deleterious effects of preceding ones, as has been seen with mutations associated with dosage compensation and the nuclear electron transport chain mutations. Second, we investigated whether a particular class of mutations confers selfish behavior in mitochondria. We assayed fitness across four life-history traits in backcrossed ancestral lines with wild-type nuclear genomes and heteroplasmic mutation-bearing mitochondrial genomes. The addition of each subsequent mitochondrial mutation reduced overall fitness in the backcrossed lines. We then propagated ancestral lines containing each combination of mutations by bottlenecking to reduce interindividual competition in order to track the intraindividual frequency of heteroplasmy over evolutionary time and to determine if selfish drive could be detected. Without interindividual competition acting upon these lines, a mean increase in heteroplasmic frequency across replicates could be attributed to an intraindividual selfish drive. We found evidence of selfish drive for three of the four mutations. Interestingly the only line that did not show clear evidence of selfish drive was a frameshift insertion in nd5, but a subsequent insertion in nd5 at the same location did confer selfish drive. We found that selfish drive can be seen in a variety of different classes of mitochondrial mutations, but we found no evidence for compensatory effects of each subsequent mutation. We were unable to disentangle the contribution of the original deletion from the selfish behavior of each subsequent mutation. It is therefore possible that the subsequent mutations lack a selfish drive of their own, but serve to enhance that of the original deletion.

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Graduate Student
Poster #: 17
Poster Session: 1
Breakout Room: 4
Award Competition: Yes

Exploring neuron death in a *C. elegans* model of Alzheimer's disease

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Alzheimer's disease (AD) is the leading cause of dementia worldwide and has been characterized pathologically as gross loss of neurons and neuronal connections in areas of the brain that control memory and executive function. Genetic variations in a number of genes, including amyloid precursor protein (APP) and apolipoprotein E (APOE), have been shown to influence both age of onset and severity of AD. Both an additional copy and gain-of-function mutants in APP can cause earlier onset of AD, while the e4 allele of APOE (APOE4) hastens and exacerbates AD compared to its other alleles (APOE2 and APOE3). How these genes influence neuronal health in AD has yet to be determined. Our lab has developed a *C. elegans* model of AD that expresses both APP and APOE4. These worms exhibit specific, age-dependent neurodegeneration and cell death that can be readily observed using fluorescent microscopy. Combined with behavioral observations, we determined that expression of APOE4, but not APOE3, leads to death of the command egg laying neuron, HSN, by middle age. Further, genetic manipulation and time-lapse microscopy suggests neurons are not dying via apoptosis. We are currently exploring additional cell death pathways, as discovering the underlying neurodegeneration mechanisms may help identify novel therapeutic targets.

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Postdoc
Poster #: 19
Poster Session: 1
Breakout Room: 4
Award Competition: Yes

The perils and promise of models of chromosome evolution

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A surge of new and more complex models is allowing rapid testing of evolutionary hypotheses. However, new approaches are often only tested on simulated data that fit the inference model's assumptions. However, biological datasets often violate model assumptions in unexpected ways. This has led to a cyclic pattern where a new model is developed and widely applied, and only after it is in widespread use do we realize that biological datasets may often suffer from inflated false-positive rates. We have discovered just such an issue in a chromosome evolution model that our lab has developed. This model allows rates of chromosome number evolution to vary depending on the state of a binary trait. In an analysis of chromosome number evolution rates in Lepidoptera, we find that neutrally evolving simulated traits will be inferred to impact chromosome evolution rates 98% of the time. We show how to use simulated datasets like these to provide a valid p-value for the impact of empirical traits that will have a false positive rate of approximately 5%. Finally, we develop a new data visualization method that will allow users to agnostically interrogate their datasets to understand the degree of rate variation present across a phylogeny for any trait fit with a Markov model.

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Graduate Student
Poster #: 20
Poster Session: 1
Breakout Room: 5
Award Competition: Yes

Characterization of Flight-Muscle Specific Promoters in *Aedes aegypti*

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The *Aedes aegypti* mosquito is the primary vector of chikungunya, Zika, and dengue viruses. Current attempts to limit the spread of these viruses have been insufficient and novel methods based on genetics are being considered as an additional method for controlling these vector populations. Two flight genes predominately expressed in males, myo-sex and AeAct-3, and one flight gene expressed in both sexes, Aeflightin, are candidates for restoring male flight in a hypothetical male driven gene drive construct that knocks out flight in both sexes. Here, we cloned potential regulatory regions of these genes to direct the expression of a fluorescent protein, and incorporated each gene cassette into a transposable element vector. We hypothesized that once integrated into the mosquito genome, reporter gene expression would occur in the thoracic flight muscles during the pupal stage of development in males for myo-sex and AeAct-3, and in both sexes for Aeflightin. Following embryonic microinjections, we obtained multiple independent germline integration events for each construct. As predicted, fluorescence was observed in the thorax of AeAct-3 transgenic males pupae, but was unexpectedly observed in females as well. Future work will aim to characterize the expression of myo-sex, AeAct-3, and Aeflightin using RT-qPCR and more detailed fluorescent imaging to establish whether reporter matches the expression pattern of each endogenous gene. In addition, we will confirm insertion of the transgene with inverse PCR. These promoters could be used in future synthetic gene constructs to manipulate gene expression in *Ae. aegypti*.

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Undergraduate Student
Poster #: 21
Poster Session: 1
Breakout Room: 5
Award Competition: Yes

The Evolution of Achiasmatic Meiosis

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Crossing over of chromosomes during meiosis is a crucial process that serves the dual purpose to allow for recombination between homologous chromosomes and ensures proper segregation of chromosomes into daughter cells. Each is essential for the fitness of an organism. Despite the importance of crossing over, many species (including eutherian mammals) with chromosomal sex determination exhibit restricted recombination between the X and Y chromosome by either achiasmatic or asynaptic meiosis. In achiasmatic meiosis, all chromosomes in the homogametic sex segregate normally with crossing over but all chromosomes segregate in the absence of crossing over in the heterogametic sex. In asynaptic meiosis all chromosomes in the homogametic sex segregate normally with crossing over and all autosomes segregate with crossing over in the heterogametic sex as well. But, the sex chromosomes in the heterogametic sex segregate in the absence of crossing over. The mutations that lead to the transition from chiasmatic meiosis to achiasmatic meiosis are unknown and the selective force that has led to its fixation is unclear. We are using a forward-time, 100 locus population genetic model to investigate where in the genome (autosome, X, or Y) mutations that lead to achiasmatic meiosis are most likely to fix. The key factors that have been hypothesized to lead to this transition to achiasmatic meiosis are sexual antagonism and to reduce aneuploidy rates in males and both are tested. This model will also allow us to determine whether the dynamics of such a mutation is different when the selective force is a reduction in recombination versus improved segregation of chromosomes.

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Undergraduate Student
Poster #: 22
Poster Session: 1
Breakout Room: 5
Award Competition: No

How large inversions contribute to ecological adaptation in Atlantic herring

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Inversions contribute to genome evolution. They act by suppressing the recombination in heterozygous individuals, thus maintaining haplotype blocks that for instance may be responsible for adaptation. They are often surrounded by multiple structural rearrangements such as repetitive regions and segmental duplications. These factors preclude the detection of inversions using short reads and make the analysis difficult. We used short read whole genome sequencing data from 53 populations to detect four putative inversions in Atlantic herring on chromosomes 6, 12, 17, and 23, with corresponding sizes of 2.7 Mb, 7.8 Mb, 2.2 Mb, and 1.2 Mb, respectively. Each of these regions show large block of strong genetic differentiation among populations. Furthermore, we used long read PacBio sequencing data from one individual to confirm that these blocks of SNPs showing very strong linkage disequilibrium are caused by inversions and find proximal and distal breakpoints for inversions on Chr6 and Chr17. Atlantic herring is a pelagic fish and one of the most abundant vertebrates in the world. Natural selection has allowed Atlantic herring to adapt to waters with varying salinity, temperature, light conditions and food resources. We found a strong correlation between the frequencies of inversion haplotypes and water temperatures at spawning, indicating that these inversions are important for the adaptation to the spawning water temperatures. The inversion alleles showed strong genetic differentiation between populations spawning in relatively warm waters surrounding Ireland and Britain, compared with other parts of Atlantic Ocean, further emphasizing their role in local adaptation. Estimates of nucleotide diversity within inversion haplotypes range from 0.11% to 0.31%, which is lower than the genome average (0.3%), which may reflect strong selection as well as a smaller effective population size as recombination is suppressed between haplotypes. Overall, our study demonstrates how whole genome sequencing by short and long reads can be combined to study chromosomal inversions and their importance in adaptation.

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Graduate Student
Poster #: 23
Poster Session: 1
Breakout Room: 5
Award Competition: Yes

Media composition and fitness costs of antimalarial drug resistance mutations in *Plasmodium*

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Drug resistance mutations tend to disrupt key physiological processes, and therefore carry a fitness cost. The size of these fitness costs are a key determinant of rate of spread of these mutations in natural populations so are important to quantify. Head-to-head competition assays provide a standard approach to measuring differential fitness of microbial populations, and have been used extensively for malaria parasites. These assays typically use standardized culture media, containing Albumax and RPMI 1640, which has a 1.4-5.5 fold (mean: 2.6-fold) higher concentration of amino acids than human blood. In this rich media we predict that fitness costs will be underestimated because resource competition is weak. We tested this prediction using an artemisinin sensitive parasite edited to contain kelch-C580Y or R561H mutations conferring resistance to artemisinin or synonymous control mutations. We examined the impact of these single amino acid mutations on fitness, using replicated head-to head competition experiments conducted in media containing (i) normal RPMI, (ii) modified RPMI with reduced amino acid concentration, (iii) RPMI containing only isoleucine, or (iv) 3-fold diluted RPMI. We found a significant 1.3 – 1.4 fold increase in fitness costs measured in modified and isoleucine-only media relative to normal media, while fitness costs were 2.5-fold higher in diluted media. We conclude that fitness costs are strongly affected by media composition and are likely to be significantly underestimated in normal RPMI. Elevated fitness costs in nature will limit spread of ART-resistance but will also promote evolution of compensatory mutations that restore fitness.

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Technician
Poster #: 24
Poster Session: 2
Breakout Room: 6
Award Competition: Yes

Adaptive divergence of meiotic recombination rate in ecological speciation

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Theories predict that directional selection during adaptation to a novel habitat results in elevated meiotic recombination rate. Yet the lack of population-level recombination rate data leaves this hypothesis untested in natural populations. Here, we examined the population-level recombination rate variation in two incipient ecological species, the microcrustacean *Daphnia pulex* (an ephemeral-pond species) and *Daphnia pulicaria* (a permanent-lake species). The divergence of *D. pulicaria* from *D. pulex* involved habitat shifts from pond to lake habitats as well as strong local adaptation due to directional selection. Using a novel single-sperm genotyping approach, we estimated the male-specific recombination rate of two linkage groups in multiple populations of each species in common garden experiments and identified a significantly elevated recombination rate in *D. pulicaria*. Most importantly, population genetic analyses show that the divergence in recombination rate between these two species is most likely due to divergent selection in distinct ecological habitats rather than neutral evolution.

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Graduate Student
Poster #: 25
Poster Session: 2
Breakout Room: 6
Award Competition: Yes

The role of proprotein convertase subtilisin/kexin type 6 in placental development under gestational diabetes

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Abnormal placenta development has been indicated in preeclampsia and gestational diabetes (GDM), which are both common yet serious complications in approximately 10% of pregnancies. Proprotein convertase subtilisin/kexin-6 (PCSK6) is a protease that processes precursor proteins into active forms. Based on previous reports of PCSK6 expression in the placenta, involvement in embryonic development and vascular remodeling, we hypothesized that PCSK6 plays an important role in placenta development. Our WT placentas had a dynamic expression of PCSK6 in glycogen trophoblast cells, and its expression reached the strongest at E12.5. In mouse hyperglycemic (HG) pregnancies, PCSK6 protein levels were decreased which led us to further pursue the interaction of PCSK6 and HG. The current study applied a PCSK6 transgenic mouse model consisting of four groups: WT and PCSK6 knockout (KO) placentas from normoglycemic (NG) and HG pregnancies. Histological examination of placenta disclosed that spiral arteries (SpAs) in PCSK6 KO placentas, under NG and HG conditions, had decreased inner to outer diameter ratios and reduced trophoblast giant cell association compared to the WT placentas. Consistently, PCSK6 KO placentas over-phosphorylate β-catenin, a key protein to regulate transcription of migratory proteins. In the labyrinth, PCSK6 KO affected fetal capillary area (FCA) while HG affected maternal lacunae area (MLA). PCSK6 KO-HG placentas had increased interhaemal membrane (IHM) thickness. From these factors, the calculated diffusion capacity was increased in PCSK6 KO under NG but decreased under HG. In summary, our study demonstrated that PCSK6 is involved in SpA remodeling and glucose-dependent angiogenesis. Our study indicated a potential role of PCSK6 in preeclampsia and GDM related placenta dysfunctions. Future study will focus on understanding the molecular mechanisms underlying how PCSK6 deletion disrupted normal placenta development and function.

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Graduate Student
Poster #: 26
Poster Session: 2
Breakout Room: 6
Award Competition: Yes

Caenorhabditis elegans PIEZO Channel Coordinates Multiple Reproductive Tissues to Govern Ovulation

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PIEZO1/2 are newly identified excitatory mechanosensitive proteins; they are non-selective ion channels that exhibit a preference for calcium in response to mechanical stimuli. Dysfunction of PIEZOs cause a variety of genetic diseases, including the dysplasia in cardiovascular, respiration, and connective tissues. However, the cellular and molecular mechanisms of PIEZOs in these diseases are less understood. To further understand the function of these proteins, we investigated the roles of pezo-1, the sole PIEZO ortholog in *C. elegans*. pezo-1 is expressed throughout development in *C. elegans*, with strong expression in reproductive tissues. A number of deletion alleles as well as a putative gain-of-function mutant caused severe defects in reproduction. A reduced brood size was observed in the strains depleted of PEZO-1. In vivo observations show that oocytes undergo a variety of transit defects as they enter and exit the spermatheca during ovulation. Post ovulation oocytes were frequently damaged during spermathecal contraction. Given that PIEZO is an ion channel and may regulate spermathecal contractility through Ca^{2+} signaling pathways, we tested the genetic interactions between pezo-1 mutants and several cytosolic Ca^{2+} regulators with RNA interference (RNAi). Indeed, pezo-1 mutants are affected upon depletion of known cytosolic Ca^{2+} regulators. We also observed that loss of PEZO-1 revealed an inability of self-sperm to properly navigate back to the spermatheca after being pushed out of the spermatheca during ovulation. Mating with males rescued these reproductive deficiencies in our pezo-1 mutants. Reduced brood sizes were observed in each auxin-inducible tissue-specific degradation strain, suggesting PEZO-1 may act in different reproductive tissues to coordinate the reproduction. Using CRISPR/Cas9, we generated the patient-specific PIEZO2 allele (p.R2718P) in *C. elegans*, named pezo-1(R2405P). Homozygous animals carrying the pezo-1(R2405P) mutation displayed reproductive defects similar to the pezo-1ko mutants, including reduced ovulation rates, ooplasmic uterine masses, and reduced brood sizes. Overall, these observations support the idea that *C. elegans* is an appropriate model system to study PIEZO diseases. An undergoing EMS-mediated suppressor screen with this pezo-1 patient-specific allele should help identify other genetic interactors.

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Postdoc
Poster #: 27
Poster Session: 2
Breakout Room: 6
Award Competition: Yes

ocrl-1, sac-1, and sac-2 Coordinate to Sequentially Remove PtdIns(4,5)P2 and PtdIns(4)P from Nascent Phagosomes During Apoptotic Cell Clearance

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Apoptotic cells are removed and recycled in a process known as apoptotic cell clearance, consisting of phagocytosis and phagosome maturation. The initiation of phagosome maturation depends on the depletion of the phospholipid PtdIns(4,5)P2, found on the plasma membrane and the nascent phagosome, and the simultaneous production of PtdIns(3)P, whose effectors drive the physiological changes necessary for cell corpse degradation. This PtdIns(4,5)P2 to PtdIns(3)P switch is indirectly driven by the small GTPase rab-35 and the phagocytic receptor ced-1. We found considerable evidence that, in *Caenorhabditis elegans*, PtdIns(4,5)P2 is converted into PtdIns(4)P by the 5-phosphatase OCRL-1, and that PtdIns(4)P is in turn rapidly dephosphorylated by the 4-phosphatases SAC-1 and SAC-2/W09C5.7. Loss of function of ocrl-1, sac-1, and/or sac-2 inhibit proper apoptotic cell clearance. Additionally, after testing all known phosphodiesterases in *C. elegans*, none were found to affect PtdIns(4,5)P2 dynamics during apoptotic cell clearance, strongly suggesting that dephosphorylation -- and not cleavage of the inositol head -- is the predominant mode of PtdIns(4,5)P2 depletion in phagosomes. Finally, we establish that the PtdIns(4,5)P2 to PtdIns(3)P switch and actin coat disassembly occur simultaneously, and that rab-35 and ced-1 promote both actin coat disassembly and the removal of the Rac1 ortholog CED-10 and CDC-42, two small GTPases found to promote phagocytosis and the formation of the actin coat. Collectively, our work provides additional evidence that the PtdIns(4,5)P2 to PtdIns(3)P switch -- regulated by ocrl-1, sac-1, and sac-2 -- controls the transition between phagocytosis and the initiation of phagosome maturation, demonstrating that tight regulation of phosphatidylinositol dynamics is a necessary, although previously underappreciated, aspect of apoptotic cell clearance.

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Postdoc
Poster #: 28
Poster Session: 2
Breakout Room: 6
Award Competition: Yes

Genetic mapping of natural variation in telomere length in *Arabidopsis thaliana*

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Telomeres are nucleoprotein complexes at chromosome termini that protect the chromosomes from deterioration during cell division. Mean telomere length in many eukaryotic species often shows considerable and heritable intraspecific variation between individuals or populations, but few factors establishing natural variation in telomere length set point have been uncovered. Because of the availability of hundreds of genetically distinct natural populations and recombinant inbred lines, the model plant *Arabidopsis thaliana* offers a unique opportunity for the analysis of mechanisms that control natural telomere length. Here, we describe results of different approaches for genetic mapping of factors that affect telomere length establishment in *A. thaliana*. Using Quantitative Trait Loci (QTL) analysis of 480 *Arabidopsis* Multi-parent Advanced Generation Inter-Cross (MAGIC) lines, we identified a QTL accounting for 47.1% of the total mean telomere length. We identified the evolutionarily conserved NOP2a/OLI2 gene encoding an rRNA methyltransferase important for ribosome maturation as a candidate causal gene behind this telomere length QTL. Using Genome-wide association study (GWAS) mapping in a total of 795 wild accessions of A. thaliana, we identified 13 regions with GWAS-significant associations underlying telomere length variation, including a region that harbors the telomerase reverse transcriptase (TERT) gene. Ongoing experiments are designed to elucidate the molecular mechanism for how these natural alleles of NOP2a and TERT affect telomere length establishment. Additional mapping populations are also being developed to uncover more genes involved in telomere length establishment.

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Postdoc
Poster #: 29
Poster Session: 2
Breakout Room: 7
Award Competition: Yes

Do ecological associations mediate diversification histories across biogeographic barriers? An evaluation of a Central American assemblage of pine-oak birds

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The field of comparative phylogeography has recently emphasized the importance of incorporating species-specific traits when testing hypotheses about how taxa diversify in response to shared biogeographic barriers. However, most comparative phylogeographic studies have focused on a narrow subset of species-specific traits, largely assessing the impact of morphology, often used as a proxy for dispersal capacity, on patterns of diversification. Here, we incorporate a rarely used phenotype, ecological association on a habitat type, and ask whether the degree of specificity to this habitat predicts patterns of demography and genetic diversity. Specifically, we evaluate the historical demography and genetic diversity of populations of two pine-oak specialists, the Azure-crowned Hummingbird and the Acorn Woodpecker, and two pine-oak generalists, the Gray-crowned Yellowthroat and House Wren, all of which span an ecological and biogeographic barrier in northern Central America. We predicted that pine-oak specialists, in accordance with the specialist-generalist variance hypothesis, will show more genetic structure and have demographic histories that are concordant. We used whole-genome re-sequence data from 20 individuals (5 per species) of populations spanning this barrier and evaluated patterns of genetic diversity (FST, heterozygosity), population structure, and demographic and divergence history. Population structure was evaluated using the model-based clustering program STRUCTURE, historical demography was evaluated using the sequentially markovian coalescent program MSMC2 and divergence history was assessed using the full-likelihood Bayesian program ecoevolity. What we find is that taxa that are ecologically specific (specialists) on pine-oak are more likely to have greater population structure and genetic diversity across the barrier. Populations of specialists across the barrier are also more likely to have similar demographic trajectories and divergence histories, whereas populations of generalists are not.

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Graduate Student
Poster #: 30
Poster Session: 2
Breakout Room: 7
Award Competition: Yes

Integrative role of the DBL-1/BMP signaling pathway with BLMP-1/BLIMP1 in *Caenorhabditis elegans* development

Mohammed Farhan Lakdawala, Pamela Joseph, and Tina Gumienny

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Animals use multiple signaling pathways for cell-to-cell communication for proper development. One signaling pathway is defined by its ligand family of bone morphogenetic proteins (BMP). In the roundworm *C. elegans*, BMP member DBL-1 has a well-defined, conserved pathway. The DBL-1 signaling pathway is involved in a spectrum of traits, including body size, brood size, and others. How does this BMP pathway control target gene expression? Previous studies in *C. elegans* show that transcriptional regulator BLMP-1 affects a similar array of traits as DBL-1. However, the relationship between DBL-1 and BLMP-1 is not studied. We discovered that DBL-1 and DBL-1 signaling are affected by loss of BLMP-1. We also found that DBL-1 negatively regulates blmp-1 expression in a stage-specific manner. Additionally, ChIP-seq, RNA-seq, and co-immunoprecipitation analyses suggest that the DBL-1 pathway and BLMP-1 act together to control expression of some common target genes, further linking these conserved molecular mechanisms during development.

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Graduate Student
Poster #: 31
Poster Session: 2
Breakout Room: 7
Award Competition: Yes

Targeted Capture Of Pathogen DNA From Museum Collections To Understand The Natural History Of Zoonotic Pathogens

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Some of the deadliest pathogens in human-history result from zoonotic transmission. SARS-CoV-2 is a topical example, but is just one of many. Many of the known zoonoses are directly transmitted by small mammals such as rodents, which are linked to over 60 zoonoses, including diseases caused by viruses, bacteria, fungi, helminth and protozoans. Natural history museums serve as a repository for archiving samples of flora and fauna for research purposes. Each sample provides a data point in time and space that can be used to document the presence and prevalence of zoonotic pathogens. We aim to exploit underutilized museum collections of rodents by using a panel of biotinylated-capture probes for targeted capture and sequencing of pathogen DNA. The targeted capture and sequencing methodology involves hybridization of pre-designed biotinylated oligonucleotide probes which target specific loci from different pathogen groups within a reaction containing whole DNA extracts from host tissues. Non-target DNA does not bind to the probes and is discarded while the DNA of interest is captured using streptavidin-coated magnetic bead separation and enriched prior to sequencing. We designed a capture array probe set containing 39,916, 80bp RNA oligos targeting pathogens from 28 groups, including bacteria, helminth, and protozoans. Our experiments with six pathogens species indicated up to 12,800-fold enrichment of the target pathogen sequences. We used the same probes to screen for pathogens in over three decade old archived rodent liver tissues from southern Africa stored in liquid nitrogen. Our probes detected several clades of *Bartonella* species bacteria, including undocumented *Bartonella spp*. Our approach demonstrates the utility of a single assay for the detection of multiple pathogens from different host species.

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Postdoc
Poster #: 33
Poster Session: 2
Breakout Room: 7
Award Competition: Yes

Gene Regulatory Networks in Development: Genetic Variation and Robustness of Anterior-Posterior (AP) Axis Formation in *Drosophila*

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Body plan patterning is a critical step in embryonic development that has health and viability consequences across the life of the organism. Anterior-posterior (AP) axis formation is an early event in body plan patterning and establishes the head-to-tail orientation for determining cell and tissue fates. In *Drosophila melanogaster*, Bicoid is a well-studied transcription factor that acts as a morphogen in AP axis patterning by influencing expression of the Gap genes in a concentration-dependent manner to create distinct expression profiles. The Gap genes influence additional target genes that also show distinct expression profiles. Though this system has been studied extensively and many of the relevant genes have been identified, the mechanisms that allow robustness of AP axis formation across genetic backgrounds are not well-characterized. We will address this gap using the natural variation of the *Drosophila melanogaster* Genetic Reference Panel (DGRP). By quantifying spatial expression patterns of AP genes across lines of the DGRP, we can identify genetic backgrounds that show significant changes in expression. We expect to identify genomic regions (QTLs) associated with these changes in expression, which we will interrogate for potential causal elements such as enhancers of AP genes. In parallel, we will be conducting RNA-seq to uncover potential undescribed regulatory interactions and downstream targets of AP genes. These data will be used to construct an improved model of the AP patterning network.

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Graduate Student
Poster #: 34
Poster Session: 2
Breakout Room: 8
Award Competition: Yes

Stress response and growth signaling coordinate intestinal regeneration in an emerging vertebrate model

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Several vertebrate lineages possess exceptional capacities for tissue regeneration, providing unique opportunities to understand mechanisms of regeneration in vertebrates. Among these, snakes exhibit extreme intestinal regeneration following months-long fasts that involve rapid increases in metabolism, intestinal function, and tissue mass. This process induces growth and stress response pathways, yet their specific roles in regeneration, and the degree to which these mechanisms are conserved across vertebrates, remain unknown. We integrate phenotypic, transcriptomic, proteomic, and phosphoproteomic data from diverse snake species to characterize the mechanisms that drive shifts in metabolism, nutrient uptake, and cellular stress underlying post-feeding regeneration. We identify key tradeoffs between mitigating cellular stress and promoting cell growth and proliferation, and our results provide new evidence for an integral, switch-like role of stress response as a key regulator of organ regenerative pathways in vertebrates.

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Graduate Student
Poster #: 35
Poster Session: 2
Breakout Room: 8
Award Competition: Yes

Inequality of Sex Chromosome to Autosome Fusions

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Chromosomal fusions play an integral role in the remodeling of genomes and in karyotype evolution. Fusions that join a sex chromosome to an autosome are particularly abundant across the tree of life, but previous models on the establishment of such fusions have not accounted for the physical structure of the chromosomes. Our preliminary analysis predicts that a fusion joining an autosome to the pseudoautosomal region (PAR) of a sex chromosome will not remain stable, and the fusion will switch from the X to the Y chromosome each generation due to recombination. We have produced a forward time population genetic simulation to explore the outcomes of fusions to both the pseudoautosomal and non-recombining regions of sex chromosomes. The model can simulate the fusion of an autosome containing a sexually antagonistic locus to either the PAR or non-PAR end of a sex chromosome. This model is diploid, two-locus and biallelic, and is able to run thousands of simulations under a variety of conditions.

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Undergraduate Student
Poster #: 36
Poster Session: 2
Breakout Room: 8
Award Competition: Yes

The Phenotypic and Genomic Consequences of Transposable Elements in *C. elegans* Bergerac strains

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One of the earliest samples of *Caenorhabditis elegans* was isolated in Bergerac, France. Early estimates indicated that a Bergerac strain had a high copy-number of the transposable element *Tc1* and also displayed diminished fitness compared to lower *Tc1* bearing counterparts. In order clarify the extent of phenotypic disruption caused by high TE copy number, four fitness traits (developmental rate, longevity, survivorship, and productivity) were analyzed in three Bergerac strains (RW7000, RW6999, and CB4851) and compared to a wildtype N2 control. All three Bergerac strains were shown to have significantly reduced fitness compared to the control for all traits measured with specific traits displaying significant differences between Bergerac strains. To understand the molecular basis for these differences, whole genome sequencing was completed on each Bergerac strain. The *Tc1* copy number for each strain was estimated using the McClintock meta-pipeline. Here we show that *Tc1* copy number is negatively correlated with fitness scores. Additionally, lines CB4851 and RW6999 exhibited a significantly higher accumulation of *Tc1* insertions on the X chromosome relative to the autosomes. Our genomic analysis of the location of *Tc1* insertions and the genes disrupted by *Tc1* has revealed the probable cause of low fitness in these strains, while also revealing the target site preferences of *Tc1*. Future endeavors using these strains include a search for mutations associated with *Tc1* proliferation, a comparison of the relative amount of RNA transcripts in the Bergerac strains, and long-term experimental evolution at high population sizes, which will reveal the causes and consequences of *Tc1* proliferation and the genetic basis for adaptive, compensatory evolution.

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Undergraduate Students
Poster #: 37
Poster Session: 2
Breakout Room: 8
Award Competition: Yes

Examining growth phenotypes of the pgm2Δ mutation in *Saccharomyces cerevisiae* lacking or overexpressing *TPS1*, *NTH1*, and *ATH1*

Katherine E. McBroom, Zoya Waheed, Shruti Raghavan, Shreya Uppala, Sarah Smith, Akshaya Selvamani, Rebecca McDonald, Pranavya Manickavelu, Krishna Patel, Katrina Ngo, Ashley Charales, Vana Bahram, David P. Aiello

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Phosphoglucomutase (PGM) is the enzyme that is responsible for interconverting glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P) in *Saccharomyces cerevisiae* carbohydrate metabolism. Yeast lacking *PGM2* (*pgm2*Δ), the major isoform of PGM, exhibit slow growth, calcium homeostasis defects, and an accumulation of glycogen when metabolizing galactose as a carbon source. The overexpression of a glycogen breakdown gene, *GPH1*, partially rescued *pgm2*Δ mutants. We hypothesized that the partial rescue was due to an increase in trehalose levels, a carbohydrate source that is often produced in the absence of glucose. To further investigate these findings, trehalose-6-phosphate synthase 1 (*TPS1*) was knocked out and overexpressed in *pgm2*Δ mutants. The overexpression of *TPS1* was shown to successfully rescue *pgm2*Δ growth sensitivities; concomitantly, the *tps1*Δ mutation exacerbates *pgm2*Δ mutant phenotypes. These results reveal a potential link between trehalose synthesis and glycogen accumulation. There are two enzymes that hydrolyze trehalose to free glucose: an acid trehalase encoded by *ATH1* and a neutral trehalase encoded by *NTH1*. To further examine the link between carbohydrate metabolism and calcium homeostasis, we examined the hypothesis that *nth1*Δ and *ath1*Δ mutations would exacerbate *pgm2*Δ growth defects due to the lack of free glucose generated from the breakdown of trehalose, whereas the overexpression of these genes would suppress growth defects by mobilizing more free glucose.

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Undergraduate Student
Poster #: 38
Poster Session: 2
Breakout Room: 8
Award Competition: No

Examining the effects of extracellular Ca^{2+} and Methylglyoxal on HACS and LACS mutants in *S. cerevisiae* mutants lacking *PGM2*

Harsha Tamtam, Paul Mpunga, Michael A. Selby, Aarthi Kannan, David P. Aiello

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The major isoform of phosphoglucomutase, *PGM2*, interconverts glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P) in carbohydrate metabolism. *S. cerevisiae* mutants lacking *PGM2* exhibit an increase in the G1P:G6P ratio from an inability to interconvert between the two metabolites. Furthermore, the *pgm2*Δ mutant displays slow growth on galactose-containing media, sensitivity to CsA, increased induction of the unfolded protein response, and higher levels of intracellular Ca^{2+}. Calcium influx into the plasma membrane is mediated by two systems: LACS (Low-Affinity Ca^{2+} Influx System), comprised of only one known protein encoded by FIG1, and HACS (High-Affinity Ca^{2+} Uptake System) consisting of a complex of integral membrane proteins encoded by *MID1*, *CCH1*, and *ECM7*. Loss of individual HACS genes exacerbated the slow growth phenotype of *pgm2*Δ on galactose with combinations of HACS gene deletions being near lethal. However, addition of extracellular Ca^{2+} rescued galactose-specific growth defects of HACS mutant strains in the context of *pgm2*Δ. Paradoxically, we observed an increase in total cell Ca^{2+} in *mid1*Δ and *cch1*Δ mutants, further amplified in *pgm2*Δ strains. Previous work with methylglyoxal (MG) demonstrates a rescue of *pgm2*Δ growth defects by increasing cytosolic Ca^{2+} levels through vacuole- and ER-independent mechanisms. Addition of MG rescued growth of HACS mutant strains in the context of *pgm2*Δ. The data suggest there are additional Ca^{2+} influx mechanisms for yeast cells.

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Undergraduate Student
Poster #: 39
Poster Session: 2
Breakout Room: 9
Award Competition: No

The DBL-1/TGF-β signaling pathway regulates pathogen-specific innate immune responses in *C. elegans*

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The innate immune response coordinates several molecular activities, including a cell-cell signaling pathway called Transforming Growth Factor-β (TGF-β), conserved in species from the simplest animals to humans. In the roundworm *C. elegans*, the DBL-1/TGF-β pathway is required for an effective innate immune response to fight some fungal and bacterial challenges. To determine if DBL-1 is specifically required to mount an effective innate immune response against a broad range of bacteria, we challenged wild-type and dbl-1(nk3) mutant roundworms with a selected panel of bacteria that are opportunistic human pathogens. We compared avoidance behavior, nematode survival, nematode tissue integrity, pharyngeal pumping, and gene expression changes of wild-type and dbl-1(nk3) animals on our bacterial panel. Loss of DBL-1 function has a strong, specific effect on some of these *C. elegans* innate immunity-associated traits. Finally, the DBL-1 pathway is itself differentially affected by the bacteria exposure. Collectively, these findings demonstrate bacteria-specific host immune responses regulated by the DBL-1/TGF-β signaling pathway.

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Faculty
Poster #: 40
Poster Session: 2
Breakout Room: 9
Award Competition: No

RNA binding proteins coordinately control lifespan in *C. elegans*

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Regulation of gene expression affects lifespan in *Caenorhabditis elegans*. While transcription factors have been extensively studied for their role in aging, less is known about how RNA binding proteins may contribute to the aging process. We recently performed a CRISPR/Cas-9 based Synthetic Genetic Interaction (CRISPR-SGI) screen in *C. elegans* focused on conserved neuronally-expressed RNA binding proteins, and identified many double mutants with fitness defects. In one notable interaction between the MBNL1/2 ortholog mbl-1 and the ELAVL ortholog exc-7, double mutants displayed a severely shortened lifespan (~70%). Both genes are required for regulating hundreds of transcripts and isoforms, and both play a critical role in lifespan extension through insulin signaling. Additional interactions between mbl-1 and fox-1, and exc-7 and fox-1 showed ~10% and ~20% lifespan shortening respectively, while the constituent single mutants had no effect on lifespan. We have therefore identified a trio of RNA binding proteins combinatorically required for proper lifespan in *C. elegans*. The exc-7; mbl-1 double mutant appears to develop into healthy young adults after which their health rapidly declines. We have used RNA seq data to investigate which RNAs may be uniquely dysregulated in the exc-7; mbl-1 double mutant. nhx-6, a predicted Na/H exchanger, which was identified from our RNA Seq data contributes to the phenotype and is expressed in the intestine. "SMURF" assays performed to establish intestinal barrier permeability shown early "leaky" guts in the exc-7; mbl-1 double mutants. We are currently investigating further genes of interest (GOI) identified through our RNA seq analysis, and testing whether they modulate the lifespan phenotype of exc-7;mbl-1 mutants. mbl-1, fox-1, and exc-7 are neuronally-enriched genes. Initial experiments have shown partial rescue of the lifespan phenotype with mbl-1 re-expression in the nervous or intestinal tissues of the double mutant but not muscle tissue. Shortly we will be conducting experiments to test whether exc-7 expression in the nervous system is the critical tissue affecting whole-worm lifespan seen in the exc-7; mbl-1 double mutant.

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Graduate Student
Poster #: 41
Poster Session: 2
Breakout Room: 9
Award Competition: No

The RAP-2 Small GTPase and MIG-15 MAP4 kinase promote tertiary fate in *C. elegans* VPC Patterning

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During *C. elegans* development, graded EGF signal from the anchor cell (AC) induces the six equipotent vulval precursor cells (VPCs) to assume a pattern of 3°-3°-2°-1°-2°-3° cell fates. The VPC closest to the AC is induced via the Ras-Raf-MEK-ERK MAP kinase cascade to assume 1° fate. Presumptive 1° cells generate DSL ligands to induce the two neighboring cells via the Notch receptor to assume 2° fate. 1° and 2° developmental programs have been shown to be mutually antagonistic. Our lab showed that lower EGF dose causes Ras to switch effectors, from Raf to RalGEF-Ral, which functions to promote 2° fate in support of Notch. We further showed that Ral signals through GCK-2, a member of the Ste20 family of mitogen-activated protein kinase kinase kinase kinases (MAP4Ks), to trigger a p38 MAP kinase cascade to promote 2° fate (Shin et al., 2018). 1° and 2° cells execute distinct and stereotyped division patterns to form the vulva. In contrast, 3° fate is typically referred to as the "ground" or "uninduced" cell fate; 3° cells divide once and fuse with the surrounding epithelium. We have found that a paralog of GCK-2, MIG-15, also plays a role in VPC patterning. Upon mutation or RNAi depletion of MIG-15 or RAP-2, we observed an increase in ectopic 1° as well as ectopic 2° cells. MIG-15 is also required for expression of a putative cell fate reporter in 3° cells. Both RAP-2 and mig-15 are necessary for full expression of the 3° biomarker. Thus, we hypothesize that, like 1°- and 2°-promoting signals, 3°-promoting signals antagonize other vulval cell fates. Using CRISPR-Cas9, we engineered an insert of fluorescent protein and epitope tag into the 5' end of the endogenous mig-15 gene, revealing ubiquitous expression in the animal, localized to the cytosol and cell-cell junctions. We also inserted auxin-inducible degron (AID), which mediates conditional degradation of tagged proteins. We will use complementary degradation experiments and tissue-specific transgenic rescue to test whether MIG-15 functions in the VPCs to repress 1°- and 2°- signals. We will also use CRISPR to mutationally activate MIG-15, as we did previously with the paralogous GCK-2. Preliminary data suggest that RAP-2 functions similarly to MIG-15, and RAP-2 has been shown to activate MIG-15 in other systems. We hypothesize that RAP-2-MIG-15 promotes 3° fate, counter to the notion of 3° fate as "uninduced." Our work presents positions us to explore signals that promote a "ground" developmental state and perhaps informs the relationship cancers and surrounding stromal cells.

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Graduate Student
Poster #: 42
Poster Session: 2
Breakout Room: 9
Award Competition: No

An ultrahigh-density platform for large-scale in vivo phenotypic screens using *C. elegans* models

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The escalating cost of the early drug screening and poor translatability of the in vitro research outcomes during the later stages of the drug development pipelines has been demanding the development of new model systems including in vivo small animal models such as *Caenorhabditis elegans*, Drosophila, and zebrafish. Among these models, *C. elegans* is the only model system that is amenable to ultra-high-throughput screening that recapitulates human disease complexity through drug absorption, distribution, metabolism, excretion, and toxicity. High-content phenotypic screening using *C. elegans* models is gaining importance for early hit identifications and toxicology assessments in vivo. To facilitate *C. elegans* based screening, we have developed a microfluidic technology to immobilize 96 populations in the past. Here we demonstrate an ultra-high-density platform using the 384-well format and 4.5 mm well-to-well spacings. The chip can immobilize up to 30 animals under each well to image 1,920 and 11,520 animals in 64-well and 384-well designs, respectively. *C. elegans* were immobilized in 3 min and with >98% trapping efficiency. One imaging session of 64-well chip provided 2,880 fluorescence images from all 1,920 channels in <8 min that was analyzed for cellular and subcellular phenotypes. We demonstrated the high-content screening capability using the polyglutamine aggregation models (polyQ24 and polyQ40) and testing the efficacy of dronedarone, one of the previously identified hits, in reducing the number of aggregates in a dose-dependent manner. The chemical showed higher efficacy when treated at L1 stage animals as compared to the L3 animals while analyzed at the young adult stage. The device was used to assess several *C. elegans* models including the health of the motor neurons in the laterally oriented Punc-25::GFP animals using high-resolution images.

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Postdoc
Poster #: 43
Poster Session: 2
Breakout Room: 10
Award Competition: No

An Investigation Using 16S rRNA Gene Sequencing To Characterize The Microbial Taxa Present In Different Body Compartments Of Carpenter Ants

Brandon Meadows, Johnathan Hruska, Joseph Manthey

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Microbiomes have been shown to provide fitness benefits to their hosts in a wide variety of organisms. Ants present an exciting opportunity to investigate microbiomes due to their eusocial nature as they engage in behaviors that could facilitate microbial transfer between colony mates. We sought to characterize the microbial taxa found in the three different body compartments of ants: the head, thorax, and gaster. We sampled 16 individuals from seven different species in the genus *Camponotus*. We then performed Illumina 16S rRNA gene sequencing and investigated if host species, phylogenetic distance, and geographic sampling site could explain the host microbial diversity in the different body compartments. We also investigated if the different body compartments possessed a core microbial community. We found the microbial communities of the three body sites differed significantly from each other with differences between host species in the gaster and thorax, but not the head. The head had the greatest amount of diversity while the gaster contained the lowest amount of diversity and was dominated by the obligate endosymbiont, *Blochmannia*. Lastly, our core microbiome analysis showed the head had the largest core community while the gaster only had two bacterial taxa in the core community, *Blochmannia* and *Wolbachia*.

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Graduate Student
Poster #: 44
Poster Session: 2
Breakout Room: 10
Award Competition: No

Using Genomic Strategies to Identify Serpentine Stress Tolerance QTL

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Soil is a complex mixture of organic and inorganic materials that greatly affects the growth and nutritional value of plants. Plants uptake nutrients and in some soils, toxic metals, then animals and humans eat them and adverse effects ensue over time. Serpentine soils are characterized by low calcium-to-magnesium ratios, increased heavy metal presence, minimal nutrients, and low water retention. The few amounts of flora that grow in these types of soils are evidence of the stress and adaptive capabilities needed to survive a serpentine environment. By exploring the genetic mechanisms needed to resist this harsh soil, we can advance resources for developing nutrient efficient uptake in agriculture and phytoremediation. The genomes of the serpentine endemic (*Caulanthus amplexicaulis* var. *barbarae*), its granite-living sister taxa (*Caulanthus amplexicaulis* var. *amplexicaulis*), and recombinant inbred lines have been sequenced to facilitate the identification of genomic regions involved in serpentine tolerance. Information collected from predictive software and genomic programs-genes, variants, recombination breakpoints, chromosome orientations, and markers-have allowed the construction of both physical (372 Mb) and genetic (3,530 cM) maps. These maps have been used in conjunction with Quantitative trait loci tools to further identify loci involved in serpentine stress tolerance and present options for further investigation.

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Graduate Student
Poster #: 45
Poster Session: 2
Breakout Room: 10
Award Competition: No

Identifying Novel Developmental Genes Using the Undiagnosed Disease Network

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Many individuals with rare disorders struggle to receive a diagnosis for their disorder or to find treatments that improve their quality of life. The Undiagnosed Disease Network, a nationwide collection of researchers and clinicians, aims to help these individuals by identifying predicted genetic causes for their disorders, leading them to a subsequent diagnosis. Many predicted disease-causing variants in genes from UDN participants have been identified, making them a potentially rich source of future study. We performed preliminary literature research on each of the genes predicted to cause disease in order to identify those that are potentially important in human development with the ultimate goal of studying these genes further in the lab. We paid particular attention to the genes that might have a role in the development of the nervous, cardiovascular, and musculoskeletal systems. The top three genes that we are interested in exploring are TANGO6, MRTF A/B, and CHD2. Future studies in the lab will aim to determine the role that TANGO6 and CHD2 have in the development of zebrafish through the examination of gene expression patterns and the creation of knockout (null) lines. We will also be comparing MRTF A/B functionality between zebrafish and mice to elucidate the conservation of the genes between organisms and their role in cardiac and skeletal muscle development.

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Undergraduate Student
Poster #: 46
Poster Session: 2
Breakout Room: 10
Award Competition: No

Previous Texas Genetics Society Meetings, 1974-2020

No.	Year	Location	Organizer	President	TGS Distinguished Geneticist Award	TGS Service Award
1	1974	Galveston	Barbara Bowman			
2	1975	Houston	Margery Shaw, Tom Caskey			
3	1976	Austin	Eldon Sutton			
4	1977	San Antonio	John Prince			
5	1978	Dallas	Raymond Lewandowski			
6	1979	Galveston	Lillian Lockhart			
7	1980	Houston		Eldon Sutton	--	--
8	1981	College Station		Barbara Bowman	--	--
9	1982	San Antonio		Robert Ferrell	C.P. Oliver	--
10	1983	Austin		Bob Sanders	Meta S. Brown	--
11	1984	Dallas		Lillian Lockhart	Bob Wagner	--
12	1985	Galveston		Arthur Beaudet	Rose Schneider	--
13	1986	Houston		Margery Shaw	T.C. Hsu	--
14	1987	College Station		Don Barnett	Margery Shaw	--
15	1988	Denton		Satish Srivastava	Eldon Sutton	--
16	1989	San Antonio		Frank Greenberg	Lillian Lockhart	--
17	1990	Austin		James Womack	Barbara Bowman	--
18	1991	Dallas		Charleen Moore	Dorothea Bennett	--
19	1992	College Station		Stephen Daiger	Bill Stone	--
20	1993	Galveston		Olivia White	Mike J. Siciliano	--
21	1994	Houston		John VandeBerg	Jack Schull	--
22	1995	San Antonio		Mary Jo Harrod	Frank Greenberg	--
23	1996	Austin		Fred Elder	James Womack	--
24	1997	Dallas		Bill Stone	Louise Strong	Don Barnett
25	1998	Austin		Sue Naylor	Tom Caskey	Eldon Sutton
26	1999	Austin		Ann Killary	Arthur Beaudet	Olivia White
27	2000	Houston		Mike Siciliano	Robert Ferrell	Fred Elder
28	2001	San Antonio		Paul Samollow	Sue Naylor	Charleen Moore
29	2002	South Padre		Ronald Walter	Alfred Knudson, Jr.	Andrew Dewees
30	2003	Austin		Jim Derr	Masatoshi Nei	Sue Ann Berend
31	2004	South Padre		Robert Baker	James Lupski	Sue Naylor
32	2005	Dallas		Christi Walter	Robert Baker	Paul Samollow
33	2006	Galveston		Rodney Nairn	Bert O'Malley	James Womack
34	2007	San Antonio		Sue Ann Berend	Jacqueline Hecht	Robert Baker
35	2008	College Station		Carol Wise	Larry Thompson	Christi Walters
36	2009	Austin		Laura Cox	Richard Gibbs	Michael J.Sicilliano
37	2010	Houston		Loren Skow	David Nelson	Rodney Nairn
38	2011	Dallas		Bhanu Chowdhary	David Russell	Carol Wise
39	2012	San Antonio		Ralf Krahe	Sen Pathak	Ann M. Killary
40	2013	College Station		Heather Conrad-Webb	Stephen Daiger	Joe Angel
41	2014	Waco		Penny Riggs	Gigi Lozano	Loren Skow
42	2015	Dallas		John (Trey) Fondon	Jonathan Cohen	Heather Conrad-Webb
43	2016	Houston		Clare Gill	Ralf Krahe	Stephen Daiger
44	2017	College Station		Erika Abel	Ann Killary	--
45	2018	College Station		Sarah Canterberry	David Threadgill	--
46	2019	College Station		Jonathan Rios	Brendan Lee	David Nelson
47	2020	cancelled		Caleb Phillips	--	--
48	2021	virtual		David Aiello	Mark Kirkpatrick	--