53rd Annual
Texas Genetics Society Meeting

February 23-24, 2026

Holiday Inn San Antonio-Riverwalk

217 N St Mary's St, San Antonio, TX 78205

Texas Genetics Society
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Questions?
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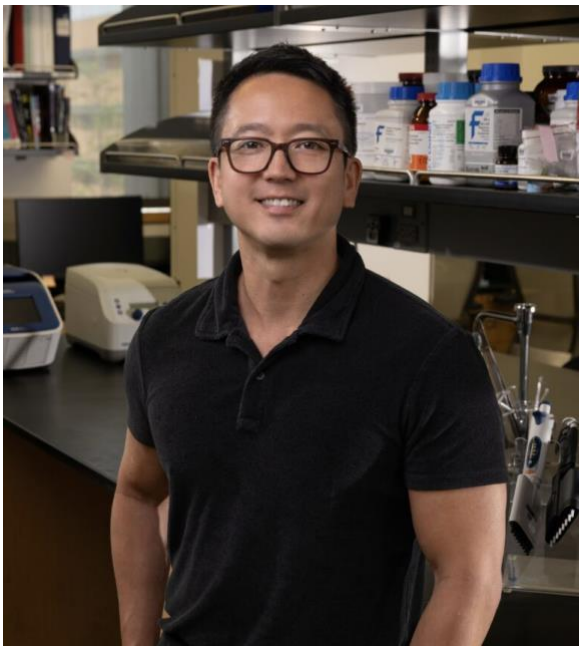
We welcome self-nominations!

Invited Speakers

2026 TGS Barbara Bowman Distinguished Geneticist



Matthew Fujita, University of Texas - Arlington

Talk Title: Genomic insights into Texas natural history: Insights from reptiles and amphibians

Dr. Fujita arrived in Texas in 2012 at The University of Texas at Arlington, where he and his lab started to catch whiptails, frogs, and other reptiles and amphibians under the scorching summer sun! They use genomics approaches to help us understand the processes that have generated the incredible and unique herpetological diversity in Texas.

Keynote Speaker

Jolene Ramsey, Texas A&M University

Talk Title: How bacteriophages make bacterial cells explode at just the right time

Dr. Jolene Ramsey has broad training in prokaryotic and eukaryotic virology. In her lab at Texas A&M University, their focus is on how bacterial viruses successfully manipulate their hosts to replicate and escape the cell. As a molecular virologist with broad interests, she also co-hosts on the popular “This Week in Virology” podcast.

Previous Texas Genetics Society Meetings, 1974–2025

No.	Year	Location	Organizer
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

			President	TGS Distinguished Geneticist Award	TGS Service Award
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	Penny Riggs
45	2018	College Station	Sarah Canterberry	David Threadgill	Erika Abel
46	2019	College Station	Jonathan Rios	Brendan Lee	David Nelson
47	2020	canceled	Caleb Phillips	--	--
48	2021	virtual	David Aiello	Mark Kirkpatrick	Tina L. Gumienny
49	2022	Bryan	Deborah Threadgill	--	Kelli Kochan
50	2023	Austin	Tina L. Gumienny	Nancy Moran	Heath Blackmon
51	2024	College Station	Heath Blackmon	Deborah Bell-Pedersen	David Aiello
52	2025	College Station	Megan Keniry	Loren Skow	Devon Boland

General Information

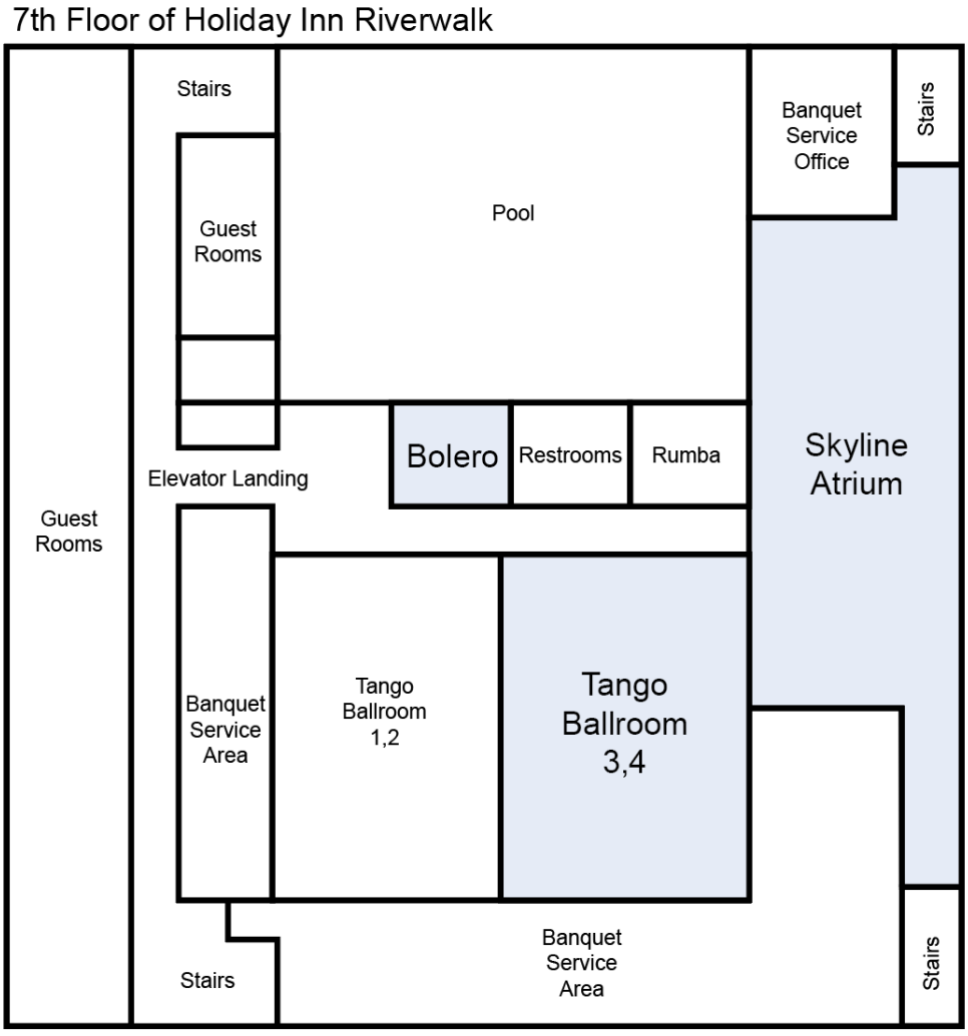
Conduct: TGS 2026 welcomes all individuals to our conference. We are dedicated to providing an environment that is collaborative, supportive, and engaging for everyone involved, and that is free of discrimination or harassment. It is imperative that everyone conduct themselves professionally and engage in courteous and respectful interactions.

Social Media Policy: Attendees are permitted to share info from presentations on social media, unless the speaker explicitly opts out by stating so at the start of their talk. Taking or sharing photos, videos, or reproductions of posters is not permitted unless you have the presenter's consent.

Drinks: Coffee, tea, soft drinks, and water will be available in the Bolero Room from 8:45 am to 5:30 pm daily.

Food: There will be a banquet on both Monday (Feb. 23) and Tuesday (Feb. 24). All other meals will be on your own outside the meeting space. There are dozens of restaurants within a few blocks of the venue.

Meeting Room Information:



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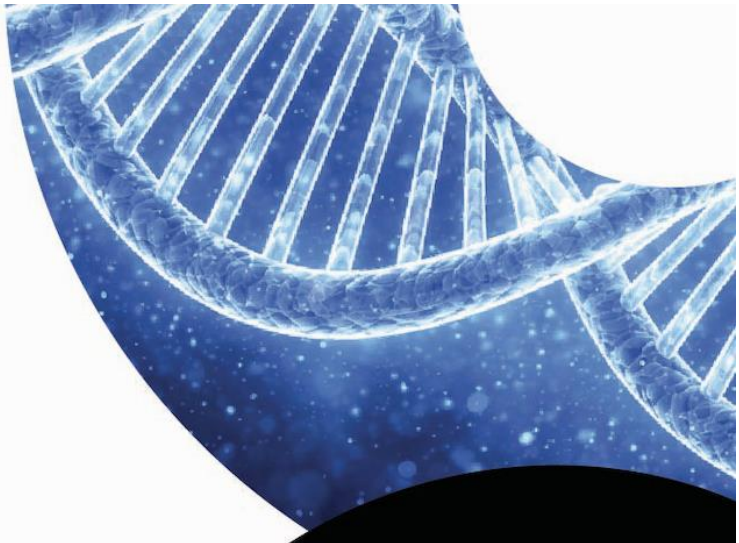

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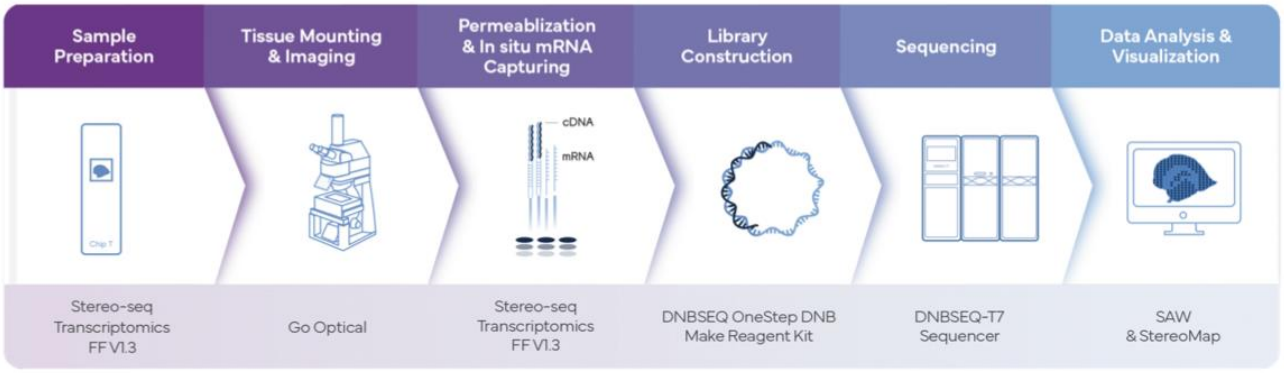

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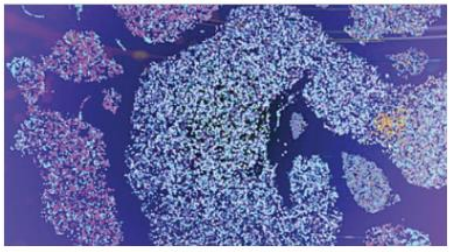

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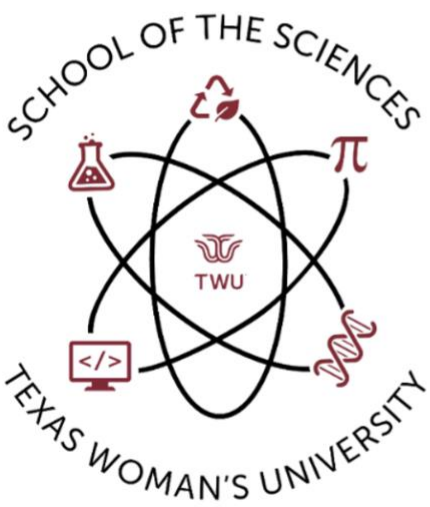

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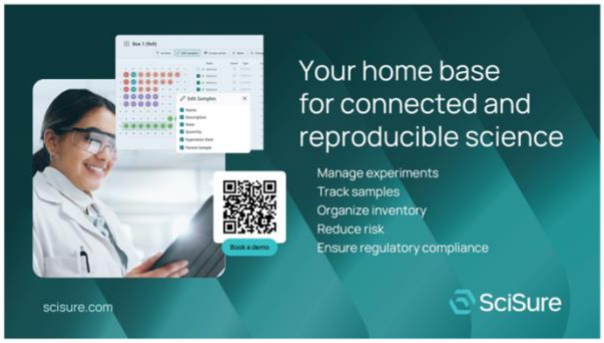

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Schedule

Monday - February 23

08:00 AM–11:00 AM	Registration Setup posters Talk File Submissions Vendor Setup	Pre-function space (hallway) Skyline Atrium Tango Ballroom 3, 4 Bolero Room
10:00 AM–11:00 AM	TGS Board of Directors Meeting	Location TBA
11:00 AM–11:15 AM	Welcoming Remarks	Tango 3, 4
11:15 AM–12:30 PM	Oral Presentations Moderator: Joe Manthey	Tango 3, 4
11:15 AM–11:30 AM	T1: **Genetic regulation of diet-induced thermogenesis in mice** Presenter: Emanuele Baldassarri, Texas A&M University	
11:30 AM–11:45 AM	T2: **Identification of a missense mutation in the NR0B1 gene in a Standardbred horse family with disorders of sex development** Presenter: Hailey Anderson, Texas A&M University	
11:45 AM–12:00 PM	T3: **Why did the cat cross the road? Exploring the genomic link underlying decision-making in wild cats** Presenter: Jack Towson, Texas A&M University - Kingsville	
12:00 PM–12:15 PM	T4: **When calories do not become reserves: Neprilysin-like 15 controls insulin–mTOR signaling and nutrient partitioning in Drosophila** Presenter: Shahira Arzoo, Texas Tech University	
12:15 PM–12:30 PM	T5: **Background-dependent response to ERBB3 inhibition in colorectal cancer** Presenter: Kaitlyn Carter, Texas A&M University	
12:30 PM–02:00 PM	Lunch Break (networking in San Antonio area restaurants)	
02:00 PM–03:00 PM	Oral Presentations Moderator: Alex Tice	Tango 3, 4
02:00 PM–02:15 PM	T6: **Characterizing green plant (Viridiplantae) assemblages in two Texas rivers by eDNA metabarcoding** Presenter: Luis Hurtado, Texas A&M University	
02:15 PM–02:30 PM	T7: **Cellular stress responses are sensitive to NADPH supply and demand in *Saccharomyces cerevisiae*** Presenter: Lily Ordoñez, Austin College	
02:30 PM–02:45 PM	T8: **Generating the transgenic stable fly lines with application for genetic biocontrol** Presenter: Tyler Chan, Texas A&M University	

02:45 PM–03:00 PM	T9: **Loss of Erbb2 reveals EGFR-dependent tumor progression in a mouse model of colorectal cancer** Presenter: William Leach, Texas A&M University	
03:00 PM–03:30 PM	Coffee Break and Networking	Bolero Room
03:30 PM–04:30 PM	Poster Session 1 (odd numbers)	Skyline Atrium
04:30 PM–06:00 PM	Oral Presentations Moderator: Devon Boland	Tango 3, 4
04:30 PM–04:45 PM	T10: **Widespread hybridization in neotropical cichlids is predicted by shared ecology** Presenter: Shannon Whitney, Texas State University	
04:45 PM–05:00 PM	T11: **Dietary determinants of hepatic retinoid variation in genetically diverse mice: foundations for a predictive model** Presenter: Brighton Garrett, Texas A&M University	
05:00 PM–05:15 PM	T12: **Single-cell transcriptomic profiling of BRAF V595E mutations in canine invasive urothelial carcinoma** Presenter: Victoria Gatlin, Texas A&M University	
05:15 PM–05:30 PM	T13: **PEX16 directs peroxisomal membrane protein trafficking in *Arabidopsis*** Presenter: Isabella Kreko, Rice University	
05:30 PM–05:45 PM	T14: **Patterns of selection and introgression among hybridizing warblers in the Davis Mountains, plus genome-wide association studies** Presenter: Ari Rice, Texas Tech University	
05:45 PM–06:00 PM	T15: **Germline-specific chromosomal aneuploidy in cattle detected by sperm sorting and sperm-FISH** Presenter: Mayra N. Mendoza Cerna, Texas A&M University	
06:00 PM–06:30 PM	Break	
06:30 PM–08:00 PM	Banquet and Plenary Talk **How bacteriophages make bacterial cells explode at just the right time** Presenter: Jolene Ramsey, Texas A&M University	Tango 3, 4

Tuesday - February 24

09:00 AM–10:30 AM	Oral Presentations Moderator: Jon Hibshman	Tango 3, 4
09:00 AM–09:15 AM	T16: **Reversal of epigenetic memory of an American diet exposure in C57BL/6J male mice through dietary intervention** Presenter: Alexandra Naron, Texas A&M University	

09:15 AM–09:30 AM	T17: **Progressive mitochondrial dysfunction in a *Drosophila melanogaster* model of PLA2G6-associated neurodegeneration** Presenter: Rubaia Tasmin, Texas Tech University	
09:30 AM–09:45 AM	T18: **From a one-horse town to full stable: recovering, refining, and revealing equine structural variation** Presenter: Sam Stroupe, Texas A&M University	
09:45 AM–10:00 AM	T19: **G6PD deficiency: a 'hidden' diagnosis beyond pharmacogenomics** Presenter: Ramiah Vickers, Baylor College of Medicine	
10:00 AM–10:15 AM	T20: **PhyloFisher v2: advancing accuracy and reproducibility in deep phylogenomics** Presenter: Robert Jones, Texas Tech University	
10:15 AM–10:30 AM	T21: **Defining mitochondrial protein functions using deep neural networks** Presenter: Abhinav Swaminathan, Texas A&M University	
10:30 AM–11:00 AM	Coffee Break and Networking	Bolero Room
11:00 AM–12:00 PM	Poster Session 2 (even numbers)	Skyline Atrium
12:00 PM–01:30 PM	Lunch Break (networking in San Antonio area restaurants)	
01:30 PM–03:00 PM	Oral Presentations Moderator: Daniela Palmer	Tango 3, 4
01:30 PM–01:45 PM	T22: **Spatially explicit population genetic simulations of avian island hybrid zones reveal importance of multiple isolating mechanisms in speciation** Presenter: Ethan Gyllenhaal, Texas Tech University	
01:45 PM–02:00 PM	T23: **Comparison of short-read and long-read RNA-Seq** Presenter: Jun Fan, Texas A&M Institute for Genome Sciences and Society	
02:00 PM–02:30 PM	T24: **Small RNA interactions with transgenes in genetically modified mosquito lines** Presenter: Vanessa Macias, University of North Texas	
02:30 PM–03:00 PM	T25: **The transcriptional landscape of adaptive thermal plasticity in cactophilic Drosophila** Presenter: Fernando Diaz, West Texas A&M University	
03:00 PM–03:30 PM	Coffee Break and Networking	Bolero Room
03:30 PM–05:00 PM	Oral Presentations Moderator: Brian Teague	Tango 3, 4
03:30 PM–04:00 PM	T26: **Identifying the role of the tri-carboxylate transporter, Indy2, in male fertility using the Drosophila model** Presenter: Surya Banerjee, Texas Tech University	

04:00 PM–04:30 PM	T27: **Incipient species and spatial covariance bias diversification analyses** Presenter: Samuel Borstein, Texas State University	
04:30 PM–05:00 PM	T28: **Understanding the neural basis of aggression across sexes using *Drosophila*** Presenter: Catherine Schretter, Southern Methodist University	
05:00 PM–05:15 PM	Presentation Judges Meeting	Tango 3, 4
05:15 PM–06:00 PM	Business Meeting + Awards	Tango 3, 4
06:00 PM–06:30 PM	Break	
06:30 PM–08:00 PM	Banquet and Plenary Talk **Genomic insights into Texas natural history: insights from reptiles and amphibians** Presenter: Matthew Fujita, University of Texas - Arlington	Tango 3, 4

Poster Sessions

Please see the online program + abstract book for poster titles, abstracts, and full author lists.

Session 1–Feb. 23–3:30pm

P1	Grace Alcocer, Trinity University
P3	Elena Alvarado, Rice University
P5	Swapnil Boyane, Texas Tech University
P7	Isabel Castro, TTU Center for Biotech & Genomics
P9	Karsyn Clouse, Austin College
P11	Catherine Fox, Texas A&M University
P13	Hector Gonzalez, Trinity University
P15	Omma Honey, Texas Woman's University
P17	Timothy Jobe, Texas Tech University
P19	Tahree Ladell, Texas Woman's University
P21	Bella Lawlar, Texas A&M University
P23	Satabdi Mandal, UT Arlington
P25	Lluis Mercade Goma, Texas Tech University
P27	Lily Ordoñez, Austin College
P29	Olivia Reed, Rice University
P31	Hazel Ruibal, Trinity University
P33	Mark Shklovskiy, Trinity University
P35	Miranda Sifuentes, UT Rio Grande Valley
P37	Ricardo Umanzor, Texas Woman's University
P39	Natalie Wideman, Texas A&M University

Session 2–Feb. 24–11am

P2	Ashley Allison, Texas Tech University
P4	Larissa Barroso, UT Rio Grande Valley
P6	Alyssa Briggs, UT Dallas
P8	Suparna Chatterjee, New Mexico State U.
P10	Eva Myers, Austin College
P12	Sadie Gaskins, Rice University
P14	Delainey Hinson, Southern Methodist U.
P16	Mahima Jetty, Austin College
P18	Dimitris Kalafatis, Texas A&M University
P20	Abagail Larkin, Austin College
P22	Madison Lester, Southern Methodist U.
P24	Amanda Mortensen, UT Austin
P26	Oluwatoyin Ogunbi, UT Austin
P28	Wyatt Porter, Texas A&M University
P30	Lauren Rosta, UT Austin
P32	Sukanya Samaddar, UT Arlington
P34	Biraj Shrestha, UT Arlington
P36	Shreya Tantry, UT Arlington
P38	Kailey Vick, Austin College
P40	Maryam Zeeshan, Austin College

Abstracts

T1

Title: Genetic regulation of diet-induced thermogenesis in mice

Presenter: Emanuele Baldassarri

Author List: Emanuele Baldassarri, Anna C. Salvador, Alexandra Naron, Ahmed Elsaadi, Aaron Vanwettering, Thomas Wong, Ryan McGovern, David W. Threadgill

Abstract: The prevalence of obesity and other metabolic diseases has increased, despite advancements in precision medicine in recent decades. There are many reasons why individuals who eat the same foods, consume the same calories, or exercise at the same level may experience different results. One key factor is thermogenesis, the production of heat through metabolic processes. Thermogenesis, and consequently body composition, is influenced by diet, exercise, genetics, or a combination of these factors. This aspect is not unique to humans and can also be observed in mice, which makes them a good model to study how genetically distinct individuals respond to the same diet. We previously demonstrated that A/J, C57BL/6J (B6), NOD/ShiLtJ, and FVB/NJ mice have different metabolic responses when exposed to a ketogenic diet (high fat, no carbohydrate) or an American diet (high fat, high carbohydrate). While a significant increase in heat expenditure was observed across the 4 strains exposed to the ketogenic diet, the magnitude of this response varied between strains. A/J had the steepest increase in heat expenditure in response to the ketogenic diet relative to American diet, whereas B6 showed a more modest response. The differences in heat expenditure could not be attributed to rates of activity or food consumption. Interestingly, B6 mice had the sharpest decrease in body fat percentage in response to ketogenic diet relative to the American diet. In contrast, A/J mice had a more modest, but still significant, decrease in body fat percentage in response to ketogenic diet relative to the American diet. To identify the genetic loci regulating thermogenesis during carbohydrate restriction, we generated F2 populations from B6 and A/J crosses and exposed them to a ketogenic diet for 3 months. Mice were phenotyped for body composition and metabolic rate, then genotyped using the Mouse Universal Genotyping Array (MUGA) with 7854 markers. Quantitative trait loci (QTL) mapping was performed to identify genomic regions driving heat expenditure, body fat gain, and activity. For heat expenditure, linkage analysis revealed QTLs on chromosomes 1 and 7 (Heatq1, Heatq2). Interestingly, the model for activity revealed nearly identical QTL on Chr1 (Actq1). Similarly, the most prominent QTL for percentage of body fat gain was observed on chromosomes 7 (Bfgq3), being in a nearly identical location to Heatq2. Two independent Chromosome Substitution Strains (CSS) lines were obtained to validate the effects of the AJ Chr1 (CSS1) and AJ Chr7 (CSS7) on the B6 background. By better understanding the interpersonal differences in genetic background, which is responsible for variation in rates of thermogenesis, there could be an opportunity to develop precision nutrition approaches to better understand metabolic disorders and address the obesity epidemic.

T2

Title: Identification of a missense mutation in the NR0B1 gene in a Standardbred horse family with disorders of sex development

Presenter: Hailey Anderson

Author List: Hailey Anderson, Sam Stroupe, Rytis Juras, Brian Davis, Terje Raudsepp

Abstract: Disorders of sex development (DSD) are conditions with discrepancies between chromosomal sex and gonadal and/or phenotypic sex and have long been reported in horses and other domestic species. The Nuclear Receptor Subfamily 0 Group B Member 1 (NR0B1), also known as DAX1, is an X-linked gene that regulates gene transcription and is essential for normal gonadal and adrenal development and steroidogenesis. However, to date, no NR0B1 mutations have been reported in horses as potentially causative of DSD phenotypes. Previous work compared a cohort of 91 DSD horses

to a control population of approximately 200 horses using short-read sequencing to identify potentially causative variants associated with DSD phenotypes. Variants in candidate genes were filtered to retain only those with predicted high to moderate effect, homozygosity for the alternate allele, and a population frequency of less than 2%. Subsequently, we identified a homozygous missense mutation (c. 674A>G) in exon 1 of NR0B1 that is present in two related Standardbred females, one with an intersex phenotype and the other with gonadal dysgenesis. The variant was absent in the larger population. Pedigree analysis with available data confirmed four additional relatives with reported DSD phenotypes, reduced fertility, and/or recurrent early pregnancy loss, as well as two relatives with no reported abnormalities. Short-read sequencing data was generated for these additional horses, and analysis is ongoing, including investigation of the functional impacts of this mutation.

T3

Title: Why Did the Cat Cross the Road? Exploring the Genomic Link Underlying Decision-Making in Wild Cats

Presenter: Jack Towson

Author List: Jack Towson, John Young Jr., Emma Brookover, Brian Davis, Jan Janečka, Lucas Spetic da Selva, Michael Tewes

Abstract: Roads serve as critical infrastructures of the global economy, but they raise alarming concerns about wildlife-vehicle collisions and habitat fragmentation. Wildlife will modify their behavior in response to road-related threats, exhibiting road avoidance and altered movement patterns. In South Texas, bobcats (Lynx rufus) and ocelots (Leopardus pardalis) are prime roadkill candidates, vulnerable to anthropogenic, environmental, and genomic difficulties exacerbated by roads. Adding to the urgency, the endangered status of the ocelot in the United States amplifies the need to mitigate road mortalities to sustain its genomic diversity and develop effective conservation initiatives. Despite progress towards identifying wildlife hotspots and implementing wildlife crossing structures (WCS) around major roads in South Texas, limited genomic research has been conducted on felid behavior to understand the behavioral decision-making process felids undergo when faced with barriers, specifically crossing or avoiding roads. In this study, bobcats and ocelots were captured at Laguna Atascosa National Wildlife Refuge, East Foundation Ranch, and other private ranches around roads including U.S. Highway 77 and Farm-to-Market 1847. Blood samples were collected dating back to 1985, and felids were fitted with GPS or VHF collars for approximately six months to monitor and assess movement patterns across the fragmented landscape. Genomic DNA from nearly 450 individuals was extracted, and whole genome sequences (WGSs) were developed using the Illumina short-read sequencing platform. Single nucleotide variants (SNVs) were called against bobcat and ocelot reference genomes, respectively, for each individual cat, and then jointly across all cats. Additionally, SNVs were called in a subset of ten behavioral genes hypothesized a priori to influence felid decision-making. Genomic variation in these targeted genes of interest will be analyzed in the 450 individuals across multiple phenotypic traits most likely related to road crossing probability, including aggression, exploratory behavior, and stress response. A gene ontology (GO)-esque functional categorization of the bobcat genome was performed, followed by an extensive literature review in carnivore ecology, to select the ten candidate genes. Population relatedness will be used to assign ‘genomic IDs’ and construct pedigree relationships to ultimately determine the origin of roadkill individuals. The kinship analyses will allow us to identify current individuals at a high risk of becoming a road mortality (e.g., individuals with roadkill parents or siblings) and pinpoint future generation individuals who may be inherently predisposed to high road mortality probability. In essence, the kinship patterns from genetic data can measure the ‘heredity of roadkill vulnerability.’ Movement patterns of genetically identified individuals will be coupled with behavioral gene expression to explain variables that contribute to felid road-crossing behavior and use of wildlife crossing structures, such as home range size, sociality, and fecundity. Collectively, these genomic dynamics will help illuminate the functional adaptability of these wild cats that has shaped their persistence in an expanding urban environment. By coupling behavioral and spatial movement patterns of bobcats and ocelots with functional genomics, informed decision-making regarding wildlife crossing structures will be implemented to maximize the fitness of these species, despite the notable challenges roads present to their survival and future in South Texas.

T4

Title: When Calories Do Not Become Reserves: Neprilysin-Like 15 Controls Insulin–mTOR Signaling and Nutrient Partitioning in Drosophila

Presenter: Shahira Arzoo

Author List: Shahira Helal Arzoo, Chase Drucker, Dr. Surya Jyoti Banerjee

Abstract: Obesity and type 2 diabetes arise from dysregulation of nutrient partitioning, in which ingested calories fail to be appropriately stored or utilized due to impaired metabolic signaling. Neprilysin has been implicated in metabolic disease through modulation of insulin sensitivity, yet the roles of catalytically inactive neprilysin-like proteins remain poorly understood. Here, we investigate Neprilysin-like 15 (Nepl15), a non-enzymatic member of the neprilysin family in *Drosophila melanogaster*, as a regulator of systemic glucose and lipid metabolism. Loss-of-function of Nepl15 results in a pronounced reduction in triglyceride and glycogen storage in male flies, despite normal viability and feeding behavior. This phenotype prompted a central metabolic question: when nutrient intake, digestion, and circulating nutrient availability are comparable, how is energy storage selectively impaired? We hypothesized that Nepl15 governs metabolic pathway flux, and that its loss compromises glucose–lipid interconversion, thereby reducing the availability of anabolic intermediates required for glycogen and triglyceride accumulation. Nepl15 mutants exhibited coordinated downregulation of key metabolic enzymes involved in lipid handling (Lpp, Mdy, Bmm), glycogen metabolism (GlyS, GlyP), and de novo lipogenesis (Acc, Fasn1, Fasn2). These enzymes are under the regulatory control of the nutrient-sensing mTOR pathway, which was significantly reduced in Nepl15 mutants, indicating suppression of anabolic signaling. Previous research had shown that adipokinetic hormone signaling did not change, directing attention to insulin signaling as the primary driver of this metabolic state. Insulin pathway activity was markedly attenuated in Nepl15 mutants, as evidenced by significant upregulation of the translational repressor 4EBP, reduced Akt phosphorylation, altered Foxo and phosphorylated Foxo levels, and decreased expression of the glucose transporter Glut1. Together, these findings demonstrate that Nepl15 loss impairs insulin–mTOR signaling, leading to reduced anabolic flux into glucose and lipid storage pathways. Despite reduced mTOR signaling, Nepl15 male mutants did not exhibit increased lifespan. Further analysis revealed significant downregulation of the longevity-associated factor Sirt6, suggesting that potential lifespan benefits of mTOR suppression may be counteracted by loss of Sirt6-mediated protective effects. Reactive oxygen species levels were unchanged, indicating that oxidative stress is not a primary contributor to the phenotype. Notably, Nepl15 mutants displayed increased locomotor activity despite diminished energy reserves, revealing a decoupling between energy storage and behavioral output. Collectively, these results establish Nepl15 as a critical regulator of nutrient partitioning in Drosophila. Rather than affecting food intake or digestion, Nepl15 modulates insulin–mTOR signaling to control metabolic pathway flux and the conversion of circulating nutrients into stored energy. This work provides mechanistic insight into how impaired anabolic routing of nutrients can generate a low-storage, high-activity metabolic state, with implications for understanding metabolic dysfunction in obesity and diabetes.

T5

Title: Background-dependent response to ERBB3 inhibition in colorectal cancer

Presenter: Kaitlyn Carter

Author List: Kaitlyn E. Carter, David W. Threadgill

Abstract: Colorectal cancer (CRC) is the second leading cause of cancer-related deaths in the United States. Advances in precision medicine have contributed significantly to early detection, diagnosis, and treatment, helping to reduce this substantial health burden. Increased understanding of molecular mechanisms which govern CRC progression has further supported this progress, as mortality rates have trended downward over the last five decades. However, death rates for individuals under fifty are increasing, highlighting the need for more effective targeted therapeutic approaches. This

preclinical research seeks to explore how genetic differences in the host influence tumor molecular profiles and treatment response, aiming to address variable outcomes observed in diverse patient populations in clinical trials. The ERBB receptor tyrosine kinase (RTK) family regulates numerous complex biological processes and has been linked to aberrant cell growth and survival, making these receptors key therapeutic targets and promising candidates for preclinical therapies. ERBB3 is a pseudo-kinase that lacks intrinsic kinase activity and depends on transactivation by other ERBB receptors. In homogenous preclinical mouse models, ERBB3 deletion in gastrointestinal epithelia significantly reduces polyp number in the intestine and colon. In contrast, clinical trials testing ERBB3 inhibitors have shown little to no efficacy and have been associated with poor patient outcomes. Building on this understanding, we investigated whether patient heterogeneity contributes to the failure of translating preclinical findings into clinical success. Using heterogenous preclinical mouse models, we discovered that the effect of ERBB3 inhibition on tumor growth is genetic-context dependent. ERBB3 inhibition on the 129S1/SvlmJ (129) background reduces tumor growth, whereas on the C57BL/6J (B6) background, tumor growth is promoted- offering a potential explanation for translational failures. This study aims to leverage mouse models to identify genetic modifiers linked to loss of ERBB3, with the goal of improving outcomes in failed clinical trials. We generated an F2 population from a 129 and B6 cross to identify regions associated with polyp count distribution through quantitative trait loci analysis (QTL). The distribution of the F2 population encompasses the range observed in both parental backgrounds. The F2 mice have been genotyped and we have identified significant regions of interest on Chromosomes 2 and 11 using R/qtl2. Candidate genes include Stk39, which has been shown to contribute to cancer progression through the MAPK/ERK pathway; Hmmr, recently identified as regulator of proliferation and CRC tumor progression through mTOR signaling (PI3K/AKT); and Gabrg2, which is involved in tumor growth through the MEK/ERK axis. Analysis leveraging human transcriptomic databases indicates that expression patterns of all candidate genes, when combined with low ERBB3 expression, affect patient prognosis. Further work will involve investigating the mechanisms underlying these modifiers to better understand their roles in CRC progression and identify potential therapeutic targets.

T6

Title: Characterizing Green Plant (Viridiplantae) Assemblages in Two Texas Rivers by eDNA Metabarcoding

Presenter: Luis Hurtado

Author List: Luis A. Hurtado, Katie K. Sanbonmatsu, Dale Kruse, Daniel Spalink

Abstract: Characterizing biodiversity at regional scales is crucial to the development and implementation of effective conservation action and policy, and to inform our understanding of macroecological patterns and processes. However, generating regional-level biodiversity data through observation-based search methods can be costly, laborious, and logistically challenging. Environmental DNA (eDNA) metabarcoding has emerged as an alternative tool to capture broad biodiversity signals from ecosystems, often requiring lower sampling effort and providing comparable or superior sensitivity than traditional search methods. In aquatic environments, where eDNA has become highly popular, it may facilitate the simultaneous characterization and monitoring of both aquatic and terrestrial biodiversity without the need for dedicated terrestrial sampling, as water may contain DNA from nearby terrestrial organisms. Lotic systems are particularly suitable for regional biodiversity characterization and monitoring via eDNA because they can transport it for long distances, permitting the detection of signals several kilometers from their source. Waterborne eDNA metabarcoding in lotic systems has been widely employed for the characterization of animal assemblages, but comparatively few studies of this kind have been performed with the goal of broadly characterizing plant assemblages. To our knowledge, research with this method and scope has not been conducted in the Southwestern United States, a region projected to experience increased drought risks in the near future. Thus, we were motivated to broadly characterize green plant (Viridiplantae) assemblages in two west-central Texas rivers through metabarcoding of water samples, representing the first effort of this kind in the American Southwest. We recovered taxa from 48 green plant families. While most taxa had already been documented near the sites where we detected them, some were not previously known to inhabit that rivers(s) where they were recovered. A substantial proportion of sequences corresponded to terrestrial embryophytes, highlighting the versatility of waterborne eDNA for capturing signals from aquatic and terrestrial organisms alike. Relative abundance of the two major green plant groups, embryophytes and green algae, exhibited considerable variation between samples, but we did not find strong evidence for a positive relationship between proportion of total reads and observed alpha diversity

for either group. We also explored spatial patterns of beta diversity, revealing contrasting trends for embryophytes and green algae. Collectively, our findings demonstrate the utility of eDNA metabarcoding for broad-scale biodiversity characterization and provide new insights into the floristic composition of Texas streams.

T7

Title: Cellular Stress Responses Are Sensitive to NADPH Supply and Demand in Saccharomyces cerevisiae

Presenter: Lily Ordoñez

Author List: Lily I. Ordoñez, David P. Aiello

Abstract: Cells constantly balance how much NADPH they synthesize and how much they consume. NADPH sits at the intersection of metabolism and stress resistance because it fuels biosynthesis, antioxidant defenses, and detoxification of reactive molecules. However, surprisingly little is known about how disrupting that balance at specific points in metabolism affects the ability of cells to survive complex stress environments. Saccharomyces cerevisiae grown on galactose was used as a model to better understand this balance. This study focused on three key enzymes: Pgm2, Gre3, and Glr1. Pgm2 is a phosphoglucomutase that channels galactose-derived carbon into the oxidative pentose phosphate pathway (PPP), thereby feeding the major source of cytosolic NADPH. Gre3 is an NADPH-dependent aldose reductase that can reduce galactose and reactive carbonyls. Glr1 is the NADPH-dependent glutathione reductase that regenerates reduced glutathione (GSH) from its oxidized form (GSSG) and helps maintain a reduced intracellular redox state. Together, Gre3 and Glr1 represent major NADPH-consuming "sinks" during redox and carbonyl stress. To evaluate what happens when NADPH supply and demand are deliberately unbalanced, we combined PGM2 deletion (*pgm2*Δ) with overexpression of GRE3 or GLR1. We then compared the growth of wild-type and *pgm2Δ* strains carrying either the empty vector pRS316 or the overexpression plasmids pGRE3 and pGLR1 across environmental stress conditions designed to test the limits of NADPH-dependent cellular defense capacity. Across these conditions, a consistent growth pattern emerged. Wild-type cells with intact PGM2 and no extra NADPH-consuming enzymes are the most resistant. Strains in which either NADPH supply was reduced, predicted by loss of PGM2, or NADPH demand was increased, predicted by GRE3 or GLR1 overexpression, showed intermediate sensitivity. The most sensitive strains were those that combined *pgm2Δ* with GRE3 or GLR1 overexpression, which frequently showed severely reduced or no growth when exposed to extracellular stressors. These results suggest that when Pgm2-dependent carbon flux into the PPP is limited, adding additional NADPH sinks like Gre3 or Glr1 pushes the system past a threshold where redox and detoxification pathways can no longer be maintained. Ultimately, these findings suggest a working model in which Pgm2 defines the capacity of the NADPH supply line from galactose, while NADPH-dependent enzymes such as Gre3 and Glr1 act as major sinks within a shared NADPH pool that must be divided among many competing stress-response and metabolic pathways. When supply and demand are reasonably matched, cells can tolerate environmental stresses using NADPH-dependent defense systems. In contrast, when they are mismatched, those same stresses become much more damaging because the cellular NADPH "budget" is insufficient to sustain key antioxidant and detoxification pathways. More broadly, this work illustrates how considering both how much NADPH can be generated and how it is allocated among pathways can help explain why particular metabolic changes leave cells especially vulnerable under specific stress conditions.

T8

Title: Generating the transgenic stable fly lines with application for genetic biocontrol

Presenter: Tyler Chan

Author List: Tyler Chan, Zach Adelman

Abstract: The stable fly (Stomoxys calcitrans, Linnaeus) is a cosmopolitan pest of livestock and disease vector which costs the US meat and dairy industry an estimated 2 billion worth of lost production annually, primarily due to the stable fly's painful bite. Traditional integrated pest management strategies to control stable flies relies heavily on pyrethroid insecticides (permethrin), but certain tested populations of stable fly are developing resistance to the common insecticides. Other methods to control stable flies have been explored included the irradiation and release of sterilized males, but low sexual dimorphism during development hampers the high-throughput sexing of males and females required for this technique, resulting in the release of both sexes which limits its effectiveness. These approaches are used in conjunction with other methods to limit the economic damage of stable flies, but given the low economic threshold of fly infestation (approximately 10 flies per animal), new methods are needed for safe and efficacious control of stable fly populations. For this reason, we present research on genetic engineering of stable flies for improved sterile insect technique (SIT), a newly generated transgenic strain of stable fly with diverse downstream applications, and an improved microinjection protocol for generating transgenic muscid lines. New promoter sequences and fluorescent proteins have resulted in a strain of stable flies that express bright, full-body green fluorescence through all life stages and contain a site-specific recombination sequence which allows for predictable integration of future genetic cargo via site-specific integrases. Additionally, a new formulation of solid agar plates innoculated with monogenic bacterial growth were used to feed the recovering injected embryos with reduced chances of contamination and improved survival rates, which were used to generate this new transgenic line.

T9

Title: Loss of Erbb2 reveals EGFR-dependent tumor progression in a mouse model of colorectal cancer

Presenter: William Leach

Author List: William R. Leach, Michael P. McGill, Kaitlyn E. Carter, Wyatt W. Porter, Megan Si, Megan Thomas, David W. Threadgill

Abstract: Colorectal cancer (CRC) is the second leading cause of cancer related deaths in the United States and third in incidence globally. While there has been substantial progress in decreasing CRC rates in older populations, rates in younger populations continue to rise. Further genetic and molecular characterization of CRC has identified several targets for precision therapy. One such target, ERBB2, is mutated or amplified in a subset of CRC and may be a marker for resistance to anti-EGFR therapy. This subtype is independent of EGFR and can be attributed to upregulated Erbb2 which suggests a potential therapeutic target for patients with resistance to anti-EGFR therapy. To further investigate the role of ERBB2 in this EGFR-independent subtype and elucidate the potential mechanism of resistance in vivo, we generated a population of Erbb2-deficient $Apc^{Min/+}$ C57BL/6J mice ($Apc^{Min/+}$, Erbb2f/f, Tg(Vil1-Cre)). We found that Erbb2-deficiency alters CRC initiation but enhances tumor progression in the $Apc^{Min/+}$ mouse model. A comprehensive molecular analysis predicted activation of EGFR, indicating a compensatory mechanism of EGFR to enhance progression in the absence of ERBB2. Differential gene expression showed amplification of Mapk and Ctnnb1 in Erbb2-deficient tumors suggesting the activation of the RAS-MAPK pathway. This was further confirmed through Western blots which confirmed activation of EGFR and subsequent activation of the MAPK pathway through MEK/ERK. Overall, these results show the involvement of Erbb2 in CRC progression and will be utilized to inform future precision therapeutics, especially for patients with resistance to anti-EGFR therapy.

T10

Title: Widespread Hybridization in Neotropical Cichlids Is Predicted by Shared Ecology

Presenter: Shannon Whitney

Author List: Shannon Whitney, Matthew McGee, Samuel Borstein

Abstract: Understanding the generators of biodiversity has been a central theme in biology. While once thought of as an evolutionary dead end, hybridization is now seen as a potential promoter of biodiversity as it facilitates the movement of genetic material across species boundaries, potentially generating variation in functional traits. Cichlid fishes (Family: Cichlidae), a family of over 1,700 species, are a model system to investigate the ecological and evolutionary drivers of biodiversity. Hybridization has been shown to play a key role in the evolutionary history of cichlid fishes, with high levels of hybridization being uncovered in the adaptive radiation of cichlids from the East African Great Lakes. However, cichlids are also diverse elsewhere, including the Neotropics. Among the most species rich and ecologically diverse clades from the Neotropics are the Heroine cichlids (Tribe: Heroini). Heroine cichlids likely diversified in the presence of ecological opportunity as they invaded Central America from South America. While heroine cichlids are substantially older than their well-studied East African Great Lake counterparts, the role of hybridization in the evolutionary history of heroines has received significantly less attention. Thus, heroine cichlids provide a unique system to study ecological speciation over deeper evolutionary timescales and broader spatial scales. Here, we leverage a publicly available restriction-site associated DNA (RAD-seq) dataset to investigate the extent of hybridization across Heroine cichlids. Using 356,969 SNPs from 104 species (214 individuals) we reconstructed the evolutionary history for heroines using maximum likelihood and multispecies coalescent frameworks, the latter of which are specifically designed to handle gene flow and incomplete lineage sorting. Additionally, we calculated Patterson's D-statistic to detect signals of hybridization between species. Finally, we used Bayesian regressions to test if shared ecological traits, such as diet or parental care, predict increased signals of hybridization. While the maximum likelihood phylogeny yielded high bootstrap support (median BS: 100), low site concordance factor revealed substantial underlying genomic discordance (median sCF: 46.3). Furthermore, the multispecies coalescent framework provided an alternate evolutionary history, further highlighting the difficulty in inferring an accurate phylogeny for this clade. Our findings reveal that hybridization is widespread both within and between the major heroine clades. Furthermore, we find that non-tree like evolution can be explained by shared ecological traits among taxa. Our results suggest that historical hybridization shapes biodiversity across deep macroevolutionary timescales.

T11

Title: Dietary Determinants of Hepatic Retinoid Variation in Genetically Diverse Mice: Foundations for a Predictive Model

Presenter: Brighton Garrett

Author List: Brighton Garrett, Ayyappa Sista, Alexandra Naron , Marianny Alvarado, Sujith Taridalu, Yoshinori Seki, David Threadgill, Masako Suzuki

Abstract: Hidden hunger–adequate caloric intake but insufficient micronutrient levels–affects many Americans, particularly those living in poverty. This is especially detrimental for pregnant women. During pregnancy, deficiencies in essential nutrients like vitamin A (vitA) can lead to adverse maternal and fetal outcomes. However, excessive vitA intake can also be teratogenic, making precise monitoring and treatment critical. VitA levels are most reliably measured in liver tissues which can be obtained through biopsy, but this method is invasive. To improve precision nutrition strategies, we aim to develop a machine-learning model using simplified diversity outbred (SDO) mice to accurately predict hepatic retinoid levels from plasma metabolomic profiles, a noninvasive sample. This is in support of the hypothesis that genetic

variation defines vitA bioavailability in the body and its influence on vitA levels responds to fluctuations in the diet. The SDO mice reflect the genetic diversity seen in human populations. We have observed significant variation in serum and hepatic retinoid levels among the three wild-derived founder strains (CAST/EiJ, PWK/PhJ, and WSB/EiJ), highlighting the impact of genetic diversity on vitA metabolism. Previously, we successfully built a support vector regression model using liver transcriptome data to predict liver retinyl ester levels on inbred mice ($R^2 = 0.657$), and we will extend this approach to genetically heterogeneous mice. Additionally, high-fat diets have been correlated with elevated hepatic retinoid levels. To further investigate dietary influences, we will subject SDO mice to either an American (high-fat, high-cholesterol) or Japanese (low-fat, low-cholesterol) diet, encompassing a broad range of vitA levels. Currently we are assessing the phenotypic variation in between diet groups in a genetically diverse mouse model. The cohort includes 168 mice (split evenly into males and females and diet groups). Mice are weighed weekly, and body composition is assessed using EchoMRI at baseline (before diet), 6 weeks, and 12 weeks. Analysis of the initial body composition reveals considerable variation among mice on the same diet, highlighting the influence of genetic background on dietary response. These results exemplify the importance of using outbred models to better capture real-world biological variability and inform more personalized approaches to nutrition and metabolic health. This project is still ongoing. In subsequent aims, tissue and serum analyses will support us to develop a hepatic prediction model. This noninvasive approach could provide a valuable tool for assessing vitA status in the liver and guiding dietary recommendations in at-risk human populations in the future.

T12

Title: Single-cell transcriptomic profiling of BRAF V595E mutations in canine invasive urothelial carcinoma

Presenter: Victoria Gatlin

Author List: Victoria Gatlin, Brian Davis

Abstract: Canine invasive urothelial carcinoma (iUC) is an aggressive bladder cancer that serves as a valuable comparative model for human muscle-invasive disease. To define the cellular context of recurrent BRAF V595E mutations, we performed single-cell RNA sequencing on tumor and matched normal bladder tissues from three dogs with iUC. BRAF V595E mutations localized predominantly to urothelial clusters, indicating that this alteration arises within the neoplastic epithelial compartment rather than stromal or immune cells. However, BRAF transcripts were low or undetectable in many mutation-positive urothelial cells, with more variable expression observed in non-urothelial populations. This disconnect between mutation status and transcript abundance suggests that BRAF V595E is not associated with a uniform or dominant BRAF-driven transcriptional program at single-cell resolution in this cohort. These findings, together with the observed epithelial, stromal, and immune heterogeneity, motivate larger integrative studies to clarify the functional role of BRAF V595E and its relevance for BRAF-targeted diagnostics and therapies.

T13

Title: PEX16 directs peroxisomal membrane protein trafficking in Arabidopsis

Presenter: Isabella Kreko

Author List: Isabella Kreko, Bonnie Bartel

Abstract: Peroxisomes are conserved organelles involved in fatty acid beta-oxidation and reactive oxygen species metabolism; proteins called peroxins (PEX proteins) guide organelle formation and function. Mutations in peroxins often result in deleterious phenotypes, such as Zellweger spectrum disorder in humans and developmental defects in plants. Early-acting peroxins (PEX3, PEX16, and PEX19) work together to insert other peroxisomal membrane proteins (PMPs) during peroxisomal biogenesis. However, our understanding of this process and the exact functions of these peroxins, in

particular PEX16, remains limited. We used CRISPR-Cas9 to develop pex16 mutant alleles disrupting various predicted domains to investigate the roles of these domains. We found that PEX16 is essential for life in Arabidopsis, as an early frameshift mutation confers embryo lethality. In viable mutants, we found multiple striking phenotypes. First, we found partial mitochondrial localization of a peroxisomal membrane reporter using confocal microscopy and lower levels of the reporter and some endogenous PMPs using immunoblotting, indicating decreased insertion of PMPs. Second, we found that pex16 peroxisomes are large and mostly devoid of intralumenal vesicles. Lastly, we found that pex16 seedlings display slowed lipid droplet mobilization and beta-oxidation defects in growth assays without notable defects in lumenal protein import. Our findings reveal peroxisomal defects in pex16, and we plan to learn more about PMP insertion when PEX16 is dysfunctional by assessing the localization of various PMP-fluorescent protein reporters. Our goal is to expand our understanding of PEX16 roles in peroxisomal biogenesis, including its localization, effect on PMP insertion, and influence on peroxisome morphology.

T14

Title: Patterns of selection and introgression among hybridizing warblers in the Davis Mountains, plus genome-wide association studies.

Presenter: Ari Rice

Author List: Ari Rice, Joseph D. Manthey

Abstract: New world warblers (family: Parulidae) comprise 115 small, colorful songbirds of which many species are known to hybridize in the wild. This makes warblers a popular subject on how reproductive barriers evolve between species, how hybridization shapes genomic patterns, and whether exchanged genetic material confers advantages among its recipients via adaptive introgression. However, one particularly unique example of warbler hybridization exists in the Davis Mountains of west Texas, where Virginia's warblers (L. virginiae) and Colima warblers (L. crissalis) have produced a very small, yet apparently self-sustaining population of hybrids. Here, we present an update on our genomic work with this population and address several key questions about it, such as how long it has existed for, whether admixture extends into neighboring warbler populations, and whether selection affects localized patterns of ancestry among hybrids. In addition, we present our findings on a genome-wide association study to determine SNPs associated with plumage and morphometric features. Thus far, we have discovered admixed individuals in the Guadalupe Mountains, which was previously thought to contain only Virginia's warblers. Through windowed admixture and Bayesian genomic cline analyses, we also found reduced admixture on the Z chromosome and biased ancestry towards Colima warblers on a large section of a particular autosome. Interestingly, this section contained multiple SNPs associated with chest color in birds, and one of these SNPs occurs in a cancer-related MC3 gene. However, more work is required to determine whether hybridization truly benefits this bird population or if it simply happens due a lack of conspecific mates in the Davis Mountains.

T15

Title: Germline-Specific Chromosomal Aneuploidy in Cattle Detected by Sperm Sorting and Sperm-FISH

Presenter: Mayra N. Mendoza Cerna

Author List: Mayra N. Mendoza Cerna, Yuri Tani Utsunomiya, Vinicius Henrique da Silva, Adam Taiti Harth Utsunomiya, Haniel Cedraz, Gabriela Canabrava, Clara Gonzalez-Marin, Nader Deeb, Pablo Ross, Terje Raudsepp

Abstract: Balanced and unbalanced chromosomal abnormalities can persist undetected in breeding bulls when confined to the germline, escaping routine cytogenetic screening based on somatic cells. The ability to identify germline-specific chromosomal abnormalities is therefore critical for understanding unexplained subfertility and for improving genetic

screening in cattle breeding programs. Here, we report the detection and validation of a germline-restricted chromosomal abnormality using sperm sorting technology combined with molecular cytogenetic analyses. Proprietary sperm sorting technology from STgenetics, Karyoflow®, revealed abnormal fluorescence distributions inconsistent with the expected X- and Y-bearing sperm populations, indicating the presence of a distinct subpopulation of genetically unbalanced spermatozoa. Whole-genome sequencing of flow-sorted sperm populations (tail sequencing) suggested the presence of excess chromosomal material originating from Chr22. To validate these findings at the cellular level, fluorescence in situ hybridization (FISH) was performed on spermatozoa usingBAC probes specific for Chr22. Sperm-FISH demonstrated a subpopulation of spermatozoa exhibiting disomy for Chr22. In total, 1017 spermatozoa from unsexed semen were analyzed, of which 120 (11.79%) displayed the disomic pattern. In X-sorted semen, 2035 spermatozoa were examined, with 335 cells (16.45%) showing chr22 disomy. Importantly, cytogenetic analysis of blood lymphocytes of the same individual revealed a normal chromosomal complement, indicating that the aneuploidy was restricted to the germline and not detectable through conventional karyotyping. Together, these results reinforce the high sensitivity and specificity of the KaryoFlow® plus tail sequencing approach for detecting chromosomal abnormalities in male germline. The integration of sperm sorting, sequencing, and sperm-FISH provides a powerful framework for uncovering cryptic germline aneuploidies that directly impact fertility but remain invisible in somatic analyses. This approach has important implications for reproductive management and genetic quality control in cattle breeding programs.

T16

Title: Reversal of epigenetic memory of an American diet exposure in C57BL/6J male mice through dietary intervention

Presenter: Alexandra Naron

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Abstract: Metabolic disorders such as obesity and diabetes are rising in prevalence worldwide highlighting a need for more effective interventions. The American (Western) diet is high in carbohydrates and fats and supports obesity and metabolic dysregulation. Carbohydrate restriction through ketogenic dietary interventions has emerged as a promising treatment strategy for obesity and metabolic dysfunction. We previously investigated the efficacy of ketogenic dietary interventions in genetically distinct mice. Our group demonstrated that when C57BL/6J (B6) males are exposed to the American diet, their fat percentage is 1.77-fold higher than B6 males consuming a ketogenic diet, suggesting these mice are responsive to carbohydrate restriction in the form of a ketogenic diet. B6 mice exposed to an American diet for as short as two weeks no longer responded to a ketogenic diet intervention. To test whether B6 mice exposed to the American diet gained an epigenetic memory preventing their response to the ketogenic intervention, mice on the American diet for three months were provided a diet that is deficient of methyl donors for two weeks before going onto the ketogenic diet intervention. Body composition measurements were collected at each stage of the dietary treatment and up to 6 months of the ketogenic intervention. After the methyl intervention diet, mice on the methyl donor deficient diet had a significant loss of total fat mass with little impact on lean mass. One and six months post intervention, we observed that exposure to the methyl donor deficient diet resulted in mice gaining only 3.9 +- 2.2 gm and 12.48 +- 4.68 gm of fat mass, respectively, compared to mice who received a diet supplemented in methyl donors. Adipocytes from gonadal fat pads were isolated and sequenced from. Numerous differentially methylated regions (DMRs) induced by the American diet were reversed by the methyl donor deficient treatment. Based on these responses, we speculate that the exposure to a methyl donor deficient diet can reverse memory of the prior American diet exposure.

T17

Title: Progressive Mitochondrial Dysfunction in a *Drosophila melanogaster* Model of PLA2G6-Associated Neurodegeneration

Presenter: Rubaia Tasmin

Author List: Rubaia Tasmin, Devisri Pranvi Matam, Ruchika Theagarajan and Dr. Surya Jyoti Banerjee

Abstract: PLA2G6-associated neurodegeneration (PLAN) is a rare and progressive neurological disorder caused by mutations in the PLA2G6 gene, which encodes a calcium-independent phospholipase A2 enzyme essential for phospholipid remodeling and membrane homeostasis. Disruption of this enzyme leads to widespread cellular dysfunction. *Drosophila melanogaster* models with loss-of-function mutations in the homologous iPLA2-VIA gene display age-dependent locomotor decline, reduced lifespan, and female-specific fertility defects. Previous studies have shown that iPLA2-VIA localizes to mitochondria, and its loss results in mitochondrial aggregation, impaired mitochondrial dynamics, and apoptosis in female germ cells. These findings suggest that mitochondrial dysfunction may represent a central pathological mechanism in PLAN. Accordingly, we hypothesized that loss of iPLA2-VIA leads to systemic mitochondrial dysfunction and disruption of cellular homeostasis. In this study, we examine mitochondrial ultrastructural abnormalities, functional impairments, and associated cellular consequences in iPLA2-VIA mutant flies to better understand the contribution of mitochondrial dysfunction to PLAN pathogenesis. Transmission electron microscopy (TEM) of heads, thoraxes, and ovaries of young (<1 week) and aged (3-week-old) iPLA2-VIA mutant and control flies revealed mitochondrial abnormalities in iPLA2-VIA mutant flies, including disrupted and fragmented cristae, mitochondrial membrane damage, elongated mitochondria, and altered morphology across tissues and ages. To investigate the molecular basis of these defects, quantitative PCR analysis was performed on key genes regulating mitochondrial biogenesis, and mitochondrial dynamics. This analysis revealed the downregulation of biogenesis and fission–fusion regulators, including mTOR, PGC1α, MFN1, MFN2, OPA1, DRP1, and FIS1, indicating impaired mitochondrial homeostasis and dysregulated mitochondrial dynamics. Consistent with the structural defects, ATP assays of head, thorax and ovary showed reduced mitochondrial activity in iPLA2-VIA mutant flies, and ROS assays of head, thorax and ovary indicated disrupted oxidative stress in iPLA2-VIA mutant flies compared to control flies. To further assess systemic consequences of iPLA2-VIA loss, metabolomic and lipidomic profiling of young (<1 week) and aged (3-week-old) mutant and control flies was performed using mass spectrometry, revealing significant alterations in metabolic and lipid pathways. Smurf assays were performed to evaluate intestinal barrier integrity by monitoring the leakage of blue dye from the gut into surrounding tissues, which serves as an indicator of epithelial breakdown and systemic physiological decline. The iPLA2-VIA mutant flies showed extensive dye spread beyond the intestinal tract, indicating a significant loss of gut barrier function compared to controls. In parallel, hematoxylin and eosin (H&E) staining of fly head sections revealed prominent vacuolar lesions and widespread tissue degeneration in mutant brains, consistent with neurodegenerative pathology. Metabolomic and lipidomic data were analyzed using SIMCA, MetaboAnalyst 6.0, and Ingenuity Pathway Analysis (IPA) to identify significantly dysregulated pathways associated with mitochondrial dysfunction, aging, and neurodegeneration. This work provides mechanistic insight into how mitochondrial dysfunction contributes to disease progression in PLA2G6-associated neurodegeneration and establishes *Drosophila melanogaster* as a powerful model for identifying and evaluating therapeutic targets for PLAN.

T18

Title: From A One-Horse Town to Full Stable: Recovering, Refining, and Revealing Equine Structural Variation

Presenter: Sam Stroupe

Author List: Sam Stroupe, Jonah Cullen, Leif Andersson, Terje Raudsepp, Sian Durward-Akhurst, Jessica Petersen, Molly McCue, Ted Kalbfleisch, Brian W Davis

Abstract: Genomic research is often hindered by large-scale structural variation (SV). When using a singular haploid reference genome and short-read sequencing, SVs are largely ignored which can obscure biology by excluding causative variants. Existing routine genetic tests often rely on assays designed to genotype associated single nucleotide variants or small indels, and in some cases have not identified causative variants. These indirect methods of genotyping have shown to be inaccurate in cases when linkage does not hold true across breeds. Currently, there are no efficient and reliable methods to directly genotype large-scale causative structural variation in equids such as a 43mb inversion that causes the tobiano coat pattern or a tandem duplication of a 4.6kb region that causes greying and predisposition to melanoma. Long-read sequencing technology and pangenomics overcome downfalls of previous methods by spanning and comparing complex genome structure across many representative haplotypes. This powerful comparative genomic tool retains variation across all included haplotypes to allow multiple populations, breeds, and species to be represented simultaneously. Which in turn, will facilitate diagnostic accuracy across diverse breeds. To understand the impact of structural variation on equine phenotypes, we integrated cutting-edge long-read sequencing and pangenome tools and developed containerized workflows to facilitate the discovery, analyses, and interpretation of equine genomic diversity. Using this methodology, we were able to accurately recover and refine previously documented examples of causative SV as well as reveal novel SVs associated with iconic equine phenotypes. Additionally, numerous novel structural variants with potential phenotypic effects were identified including over 32,000 structural variants predicted to have a large impact on protein structure. Pangenomics offer a powerful and practical path forward for genomic research by integrating diverse genomes into a comprehensive graph structure, facilitating structural variant discovery and characterization.

T19

Title: G6PD Deficiency: A 'Hidden' Diagnosis Beyond Pharmacogenomics

Presenter: Ramiah Vickers

Author List: Ramiah Vickers, Sarah H. Elsea

Abstract: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzymopathy, affecting ~500 million people worldwide. G6PD is essential for protecting red blood cells (RBCs) from oxidative damage as it is the only enzyme within RBCs to provide a steady supply of NADPH, known for combating oxidative stress. Deficiency of G6PD therefore results in RBCs being vulnerable to oxidative challenges that can result in several clinical manifestations including acute hemolytic anemia, neonatal jaundice, and in more severe cases, chronic non-spherocytic hemolytic anemia. The prevalence of G6PD deficiency differs considerably among various racial groups with individuals of African and Asian ancestry being more highly affected. Additionally, G6PD is located on the X chromosome making males more highly affected, although heterozygous females are epigenetic mosaics due to X-inactivation; thus, the potential severity for hemolysis in female carriers is highly variable. Chronically, G6PD deficient individuals have exhibited increased risks for diabetes, atherosclerotic cardiovascular disease, chronic kidney disease, and infection. Moreover, these individuals have been found to exhibit more severe disease-specific complications. Despite these risks, many genetic diagnostic laboratories do not report G6PD deficiency outside of pharmacogenetic findings, unless hemolytic anemia is indicated, suggesting a lack of consideration of this deficiency on overall health status. We hypothesized that G6PD deficiency might be under-reported and could therefore result in increased health disparities due to a lack of knowledge regarding medically actionable risks, as well as conferring risk for additional complications amongst individuals with various comorbidities. To investigate, we performed a retrospective study to evaluate the prevalence and reporting of G6PD deficiency in clinical exome sequencing. Analysis of ~20,000 clinical exomes reported between 2013-2021 identified 2,341 individuals with 111 documented G6PD variants. Of these, 224 subjects (9.6%) had an unreported pathogenic or likely pathogenic variant in G6PD, including 81 males (36.2%) and 143 females (63.8%; 11 homozygotes, 132 heterozygotes–some of whom also had biallelic G6PD variants). Additionally, 19 (8.5%) of these individuals had anemia listed as a clinical manifestation. Of interest, 48 (21.9%) subjects had at least one additional confirmed genetic diagnosis, including other hemoglobinopathies and various metabolic disorders, among other conditions. These findings highlight the potential for increased risk for additional comorbidities and more complicated medical sequelae due to an increased risk for oxidative stress and medication-related trigger responses resulting in hemolytic anemia. Hemoglobinopathies are not included in the American College of Medical Genetics and Genomics recommendations for reporting of secondary

findings; thus, both affected individuals and 'trait' carriers may not be reported. Furthermore, these data demonstrate that G6PD deficiency should be considered medically actionable beyond pharmacogenomics, with consideration of the clinical implications and associated risks for individuals with co-existing genetic and non-genetic diagnoses. The broader impact of under-reporting G6PD deficiency on overall health, particularly in the presence of other genetic diagnoses, remains to be determined, warranting further investigation. We anticipate that the findings of this study have the ability to enhance quality improvement protocols and policies across clinical diagnostic laboratories to support in-depth precision medicine and to reduce health disparities across at-risk and underserved populations.

T20

Title: PhyloFisher v2: Advancing Accuracy and Reproducibility in Deep Phylogenomics

Presenter: Robert Jones

Author List: Robert E. Jones, Alfredo L. Porfirio-Sousa, Fabien Burki, Marek Eliáš, Laura Eme, Ryo Harada, Elisabeth Hehenberger, Martin Kolísko, Serafim Nenarokov, Tomáš Pánek, Andrew J. Roger, Antonis Rokas, Eric D. Salomaki, Xing-Xing Shen, David Žihala, Matthew W. Brown, Alexander K. Tice

Abstract: Resolving ancient (>100 million years) splits in the Tree of Life is inherently difficult. Over deep timescales, phylogenetic signal erodes due to substantial saturation, while systematic artifacts such as compositional bias, heterotachy, and long-branch attraction can produce strongly supported but misleading relationships. A common strategy is to concatenate many genes into supermatrices, which dilutes bias of individual genes, improves taxon sampling, and accommodates missing data well. However, most phylogenomic matrix construction is done using ad hoc tools and datasets, raising concerns about reproducibility and transparency. To address this, in 2021 we developed PhyloFisher v1, a software package that integrates a manually curated database of orthologous protein-coding genes spanning diverse eukaryotes with a suite of state-of-the-art phylogenetic tools. These include a novel algorithm for ortholog selection prediction of alternative genetic codes, removal of genes or taxa based on completeness, prediction of amino acid compositional heterogeneity, removal of heterotachious or fast-evolving sites and taxa, and flexible supermatrix construction from resampled proteins or nucleotide sequences. PhyloFisher emphasizes transparency and semi-automation, with expert curation, focusing on a moderate set of high-quality protein coding genes with robust phylogenetic signal. This balance provides sufficient phylogenetic signal to resolve deep, recalcitrant nodes, while limiting noise from paralogs, contamination, and overly large, automated datasets, thereby enabling the use of the most sophisticated evolutionary models and methods. In PhyloFisher v2, the curated database has been expanded from 240 to ~400 protein-coding genes, with broader representation of understudied lineages, and has been migrated into an SQLite format for faster and flexible querying, as well as increased reproducibility and improved integration with automated workflows. In PhyloFisher v2 we improve on ortholog assignment, by incorporating recent advances in software, algorithms, and models currently available for deep phylogenomics which improve on accuracy, as well as speed. We also increase available utility scripts to allow users to easily employ the latest dataset validation strategies and ease the user burden of publication quality figure construction. Together, these advances strengthen PhyloFisher as a reproducible and transparent framework for deep phylogenomics of eukaryotes.

T21

Title: Defining mitochondrial protein functions using deep neural networks

Presenter: Abhinav Swaminathan

Author List: Abhinav B. Swaminathan, Mohammad Zulkifli, Rachel M. Guerra, Sofia M. Calabrese, Dimitris T. Kalafatis, Amy N. Spelbring, David P. Barondeau, David J. Pagliarini, Vishal M. Gohil

Abstract: Despite the fundamental importance of mitochondria in cellular metabolism, the molecular function of many mitochondrial proteins remains unknown. Since protein function can be inferred from their interacting partners, we repurposed the protein structure prediction algorithm - AlphaFold Multimer (AFM) - into a classifier model to predict protein-protein interactions of the entire human mitochondrial proteome. By screening 630,003 protein pairs, we created a compendium of 2895 known and novel interactions, which included interacting partner(s) of 85 uncharacterized mitochondrial proteins, linking them to a known biochemical pathway. Extending the AFM-based analysis to 11 diverse eukaryotes identified evolutionarily conserved interactions among human hits, including previously unknown regulators of core bioenergetic pathways. Guided by these predictions, we experimentally defined the mitochondrial copper delivery pathway to cytochrome c oxidase and discovered an uncharacterized protein, ARM1, as a new 2Fe-2S cluster-containing protein that repairs oxidatively damaged mitochondrial aconitase. Our results resolve a decades-old biochemical mystery surrounding endogenous repair mechanisms of aconitase and, more generally, illustrate how our compendium can be used for systematic structure-based functionalization of the human mitochondrial proteome.

T22

Title: Spatially explicit population genetic simulations of avian island hybrid zones reveal importance of multiple isolating mechanisms in speciation

Presenter: Ethan Gyllenhaal

Author List: Ethan F. Gyllenhaal, Lluis Mercade, Elsie H. Shogren, J. Albert C. Uy, Benjamin C. Haller, Joseph D. Manthey, Philipp W. Messer

Abstract: A fundamental goal in evolutionary biology is to understand what genetic, geographic, and ecological factors allow for the coexistence of new species. Isolated oceanic islands act as natural laboratories for understanding the origin of species but oftentimes rely on inferences based on occurrence patterns rather than more powerful tests that leverage hybridizing taxa experiencing secondary contact for the first time. If reproductive isolation is enough to avoid lineage fusion and ecological divergence is enough to pre-empt competition, secondary contact can result in coexistence and the build-up of local biodiversity. Two avian hybrid zones in the South Pacific exemplify the opposite ends of this spectrum, with the Myzomela honeyeaters of the Solomon Islands showing evidence of coexistence and Pachycephala whistlers of Fiji experiencing high gene flow. The two hybridizing pairs are starkly different in key factors relating to coexistence, with strong assortative mating, genetic incompatibilities, and ecological divergence in the honeyeaters, and only evidence for strong plumage divergence in the whistlers. To identify what factors are most important for secondary sympatry in different geographic contexts, we performed a series of geographically explicit population genetic simulations of these two contact zones. To be precise, we evaluated the relative role of four polygenic factors: assortative mating, sex chromosome-linked genetic incompatibilities, ecological divergence, and dispersal syndrome divergence. We found that all factors except the sex-linked incompatibility were important for long-term coexistence of alternate genome-wide ancestries, but alternate sex chromosome haplotypes persisted when incompatibilities were present. The outcomes were dramatically different based on geographic context, with the same parameters resulting in co-existence in the Solomon Islands and complete genetic swamping in Fiji. More generally, the relatively weak role of sex-linked incompatibilities in autosomal ancestry is striking, as sex chromosome divergence is often cited as a driver of speciation in birds.

T23

Title: Comparison of Short-Read and Long-Read RNA-Seq

Presenter: Jun Fan

Author List: Jun Fan, Rachel Lorraine Bruce, Jiaqian Qi, Zhenyu Li, Jeffrey D Cirillo

Abstract: Transcriptome analysis provides valuable insights into gene expression patterns under diverse physiological and pathological conditions. Currently, the majority of transcriptomic studies rely on Illumina short-read sequencing, which offers high throughput, enables simultaneous profiling of thousands of transcripts across many samples, and is very cost-effective. However, even with well-established bioinformatics pipelines, short-read RNA-seq libraries struggle to clearly resolve full-length transcripts. This limitation leads to difficulties in distinguishing between isoforms of the same gene and hinders accurate assembly of complex or repetitive regions in the transcriptome. We hypothesize that PacBio long-read Iso-Seq/Kinnex library sequencing can overcome these shortcomings and provide a richer, more comprehensive view of gene expression, particularly transcript isoforms, in biological samples. We examined this hypothesis by collaborating with Dr. Li's lab in the School of Pharmacy for studies analyzing platelet gene expression in mice. We performed both Illumina short-read and PacBio long-read RNA-seq library preparations. Our results demonstrate that PacBio Iso-Seq captured additional peaks across the transcriptome compared to Illumina short-read RNA-seq. Further analyses revealed some key platelet regulatory genes that were completely missed in the short-read RNA-seq libraries. We conclude that long-read RNA-seq is superior to short-read sequencing for detailed analysis of transcriptional regulatory pathways.

T24

Title: Small RNA interactions with transgenes in genetically modified mosquito lines

Presenter: Vanessa Macias

Author List: David Jordan, Aixa Brizuela, Matthew Carrel, Nelson Lau, Vanessa Macias

Abstract: Genetic engineering in research is a powerful tool to reveal gene function and typically involves encoding a new synthetic nucleotide sequence into a genome to generate or perturb a particular gene function. In mosquitoes, genetic engineering is also an important technology for developing transgenic mosquitoes for use in mosquito and disease control applications. The synthetic DNA constructs used in both basic and applied science to generate such transgenic organisms are almost always comprised of genes and gene parts that did not originate from the target organism, parts such as phage- or transposon- derived insertion sequences, virus-derived 3'UTRs that ensure plasmid delivery to the nucleus and fluorescent protein encoding sequences derived from sea animals that enable visual confirmation of transgene insertion. To approach the very broad question as to what extent these transgenes are treated by the organism as a foreign entities, we have surveyed the small RNAs used by two distinct RNAi mechanisms in wild and transgenic Aedes aegypti. These important vectors of human and animal viruses mosquitoes are specially equipped with a diversified ability to recognize genetic parasites using siRNAs and piRNAs. Our small RNA sequencing data reveal surprising homology patterns between small RNAs and transgene modules; based on these data, we propose several testable mechanisms of transgene recognition that will support the larger functional description of foreign DNA interactions in mosquitoes. This work has both basic and applied importance to the informed use of genetic engineering for hypothesis testing and disease control.

T25

Title: The Transcriptional Landscape of Adaptive Thermal Plasticity in Cactophilic Drosophila

Presenter: Fernando Diaz

Author List: Fernando Diaz, Luciano Matzkin

Abstract: Phenotypic and transcriptional plasticity allow organisms to adjust their traits and patterns of gene regulation in response to changing environments, helping populations cope with environmental stress. Understanding the genomic mechanisms underlying these responses is central to evolutionary and ecological genomics. Cactophilic Drosophila, which inhabit arid and climatically variable environments, provide a valuable system for examining how thermal environments shape plastic responses across life stages and generations. Here, I synthesize recent work showing that thermal

acclimation produces stage-specific effects on heat tolerance, with especially pronounced responses during larval development. Genome-wide analyses reveal that thermal plasticity is accompanied by extensive changes in gene regulation, including widespread differential gene expression and dynamic patterns of alternative splicing. These transcriptional responses involve genes associated with stress response, metabolism, and development, and differ across life stages and environmental contexts. Parental thermal environments can also influence offspring transcriptional profiles by modulating gene expression levels, although these effects do not involve alternative splicing. Parental and offspring acclimation generate opposing patterns of gene expression, suggesting compensatory regulatory responses that may help stabilize performance under fluctuating conditions. Together, these results highlight how environmental variation shapes transcriptional plasticity in cactophilic Drosophila, and demonstrate the value of integrating phenotypic assays and transcriptomic analyses to understand the mechanisms by which organisms respond to thermal variation across fluctuating environments.

T26

Title: Identifying the role of the tri-carboxylate transporter, Indy2, in male fertility using the Drosophila model

Presenter: Surya Banerjee

Author List: Mst Hasina Begum, Riddhi Patel, Ada Salazar, Surya Jyoti Banerjee

Abstract: In humans, male infertility contributes to nearly half of infertility cases worldwide, yet its underlying molecular mechanisms remain unclear for one-third of the cases. The mammalian sodium-dependent Solute Carrier (SLC) family members, SLC13A1 (NaS1), a sulfate symporter, and SLC5A1 (SGLT1), a glucose symporter, play critical roles in sulfate homeostasis in the kidney and in the transport of digested monosaccharides from enterocytes to blood, respectively. Interestingly, SLC13A1-deficient mice exhibit reduced fertility, whereas pharmacological activation of SLC5A1 enhances sperm quality in humans, underlying their roles in fertility. However, the mechanism remains unknown. Another member, SLC13A5 (568 amino acids), 12 transmembrane domains containing transporter for the cellular uptake of citrate (NaCT), is expressed in the human liver, brain, and testes. It has low dependency on Na+, and its mutation causes epileptic encephalopathy. However, its role in male reproductive physiology remains unexplored. Thus, SLC13 and SLC5 family members become paradoxical candidates for fertility defects. A Drosophila (fruit fly) ortholog of the SLC family members, Indy2 (I'm not dead yet 2), shows 29.61%, 15.14%, and 36.62% sequence homology with human SLC13A1, SLC5A1, and SLC13A5, respectively. Indy2 (562 amino acids) is predicted to be a Na+-independent citrate transporter with 13 transmembrane domains, a cytoplasmic N-terminus domain, and an extracellular C-terminus domain. Transcriptomics data show Indy2 is exclusively expressed in the fly testes. Previous research revealed that the presence of sufficient citrate in sperm was important for sperm capacitation. Thus, we hypothesized that Indy2 is important for maintaining male fertility by regulating citrate-dependent energy metabolism. First, we confirm that Indy2 expression is limited to the wild-type fly testes by RT-qPCR. Moreover, RNA in situ hybridization reveals that Indy2 mRNA expression is relatively higher in the middle (where spermatocytes develop) and basal (where spermatids are present) testis, and lower at the apical (germline stem cells are localized) testis, aligning with another transcriptomics study. Immunohistological staining confirms that the Indy2 protein colocalizes with E-cadherin and Beta-Catenin proteins on the testicular membrane. Interestingly, the homozygous Indy2 mutation drastically lowers fecundity and fertility of the mutant flies. The number of eggs, pupae, and adult progeny decreased drastically with the increase in age of the Indy2 mutant parent flies. This suggests that Indy2 plays a crucial role in fertility and reproduction. Next, we find that glycogen and lipid storage are noticeably reduced in the Indy2 mutant testes by PAS and Nile Red staining. Additionally, we reveal that Indy2 mutant testes contain less ATP by biochemical assay, and distorted filamentous actin in the sperm tails by phalloidin staining. As ATP binds with globular actin to polymerize them for forming the filamentous actin, thus, reduced ATP production in the mutant testes may cause this abnormality in the sperm tails of the mutant flies. Overall, Indy2 loss of function impairs testicular nutrient storage, energy production, and sperm tail integrity, which ultimately results in fertility defects in fruit flies. In the future, we will evaluate the ability of citrate transport by Indy2 and whether the human SLC13A5 plays similar roles in fertility.

T27

Title: Incipient Species and Spatial Covariance Bias Diversification Analyses

Presenter: Samuel Borstein

Author List: Eleanor M. Hay, Samuel R. Borstein, Matthew D. McGee

Abstract: Macroevolutionary analyses typically treat species as discrete units and account for shared evolutionary history. However, speciation is a continuous process. Gene flow may occur between species, and taxa are often spatially clustered, potentially biasing inferences of diversification. Here, we investigate how species delimitation and spatial non-independence influence speciation dynamics and drivers of diversification using cichlid fishes as a model system. Using a phylogeny and trait dataset of 1,712 species, we first generated a reduced dataset of 820 species by removing incipient species based on known breeding incompatibilities. We then fit phylogenetic and spatiophylogenetic models using an integrated nested Laplace approximation framework to jointly account for shared ancestry and spatial covariance. We find that the treatment of incipient species and spatial non-independence both alter speciation patterns and the predicted drivers of diversification. Analyses of the full dataset found strong trait associations and spatial hotspots driven by young adaptive radiations in Lake Victoria and Lake Malawi, whereas removing incipient species reduced extreme speciation rates, weakened or removed trait effects, and largely eliminated spatial hotspots. These results demonstrate that macroevolutionary inference is sensitive to species delimitation and spatial structure, highlighting the need to consider the influence of incipient species and spatial covariance in comparative analyses.

T28

Title: Understanding the neural basis of aggression across sexes using Drosophila

Presenter: Catherine Schretter

Author List: Catherine E. Schretter, Mei Shao, Gerald M. Rubin

Abstract: Regulating aggression is particularly critical for survival across species, as engagement alters group dynamics and can lead to injury or even death. While Drosophila display aggression across sexes, features of female and male aggression differ, including the environmental contexts and behavioral components. Previous work has implicated several isomorphic and sexually dimorphic cell types in guiding the approach, engagement, and continuation of female aggression (Palavicino-Maggio et al., 2019; Schretter et al., 2020; Deutsch et al., 2020; Chiu et al., 2021; Chiu et al., 2025; Schretter et al., 2025). However, little is known about the circuitry downregulating aggression across sexes due to the lack of a complete map of the central nervous system, or connectome, in a male fly. In the present study, we used the previously generated female connectomes alongside the recently completed male dataset to map a sexually dimorphic aggression-promoting cell type, aIPg, across sexes. We uncovered a novel glutamatergic cell type that preferentially targets the aIPg circuit. Further quantitative behavioral analysis uncovered that this cell type downregulates components of aggressive behavior across sexes. In addition to divergent inputs, common visual and auditory pathways innervating this isomorphic cell type suggest specific circuit mechanisms for both conserved and variable regulation of aggression. Beyond aggression, our study has important implications for how the brain flexibly regulates social behaviors across sexes and in changing environmental contexts.

P1

Title: Extracting Microbial DNA from Oak Tissue for Metagenomic Analysis

Presenter: Grace Alcocer

Author List: Bethany Bass, Grace Alcocer, Brain Teague

Abstract: Microbial ecology is the study of how fungi, bacteria, and viruses interact with each other and their environment. Current studies extract, amplify, and sequence DNA to determine the species present in a sample, but still fail to detect some microbes. An improved protocol for microbiome DNA extraction would isolate and purify microbial DNA to a quality sufficient for direct single-molecule sequencing without amplification. An initial protocol from Suda et. al. had unconvincing validation data, steps that decreased DNA yield, and only worked for leaf tissue. In this study, we developed a method that improves yield and purity, and expanded it to work for fresh leaves and bark. Our protocol uses sonication to separate microbes from plant tissue before microbial cell lysis, which allows the use of smaller volumes of dangerous solvents during extraction. We also used polyvinylpyrrolidone and phenol-chloroform extractions to remove phenolic compounds. Finally, we replaced isopropanol DNA precipitation steps with spin column cleanups. The resulting samples are of sufficient quality to amplify with PCR and subsequently sequence to identify the species present in a sample. Recent efforts are focused on purifying DNA that can be directly sequenced, even without PCR.

P2

Title: Concordant phylogeographic patterns shape codiversification in Camponotus texanus and its endosymbiont Blochmaniella

Presenter: Ashley Allison

Author List: Ashley Allison, Kole Menendez, Swapnil S. Boyane, Joseph D. Manthey

Abstract: Obligate endosymbionts often reflect the evolutionary history of their host and provide an opportunity to study codiversification across landscapes. Here, we studied a native carpenter ant of Texas, Camponotus texanus, and its endosymbiont Blochmaniella. This species is primarily found and associated with Ashe juniper and oak habitats in central and western Texas. Using whole-genomic data of 16 individuals, we investigated the phylogeographic patterns of host-endosymbiont codiversification, gene flow, and host genetic diversity. We performed population structure analyses like principal component analysis (PCA), ADMIXTURE to infer genetic structure. We then performed a co-phylogeny analysis of host and endosymbionts to observe the phylogenetic congruence. Next, we assessed the genetic diversity of hosts by calculating observed heterozygosity, and patterns of gene flow were inferred using estimation of effective migration surfaces (EEMS) analysis. PCA revealed two genomic clusters, hereafter referred to as central and western populations, with the western population further subdivided into two groups along the second principal component (PC2). In contrast, ADMIXTURE assigned individuals into two clusters. Observed heterozygosity was similar across both populations; however, a positive correlation with latitude was observed within the central population. Cophylogenetic analyses displayed largely congruent clades between hosts and their endosymbionts, indicating codiversification. Finally, EEMS analyses of both host and endosymbiont highlighted higher corridors for gene flow within central populations and reduced migration rates among western populations. Together, these results demonstrate that geographic isolation and restricted gene flow among the populations can shape the codiversification of Camponotus texanus and its obligate endosymbiont Blochmaniella.

P3

Title: Elucidating the Role of Peroxin PEX3 in Arabidopsis thaliana

Presenter: Elena Alvarado

Author List: Elena Alvarado, Isabella Kreko, Bonnie Bartel

Abstract: Peroxisomes are organelles that break down fatty acids and reactive oxygen species. The protein PEX3 aids in peroxisome biogenesis by inserting membrane proteins. PEX3 has escaped forward-genetic analysis in Arabidopsis because it is encoded in two genes, PEX3A and PEX3B, and null mutants of both PEX3 isoforms are inviable. Therefore, I am using various strategies to produce partial loss-of-function alleles that are viable. First, I am using CRISPR-Cas9 to develop in-frame mutations of PEX3A in plants with a null copy of PEX3B. PEX3 is predicted to contain an FFAT-like (FFAT) motif, which could interact with proteins that tether organelles. Therefore, I am seeking to produce a pex3ΔFFAT mutant. Second, I am also generating constructs encoding PEX3B with no functional FFAT motif and introducing this construct into plants with null mutations in both PEX3 isoforms. Finally, to understand the role of the C-terminus of PEX3, I am generating C-terminal truncation mutants. After generating these mutants, I will use physiological and molecular assays of peroxisome function to compare the mutants to wild type. Understanding the impacts of in-frame and truncation mutations will help elucidate the roles of PEX3 in peroxisome biology.

P4

Title: Investigating the role of the Conserved Notch Pathway in Glioblastoma cells.

Presenter: Larissa Barroso

Author List: Larissa Barroso, Megan Keniry

Abstract: Glioblastoma (GBM) remains the most aggressive primary brain cancer, representing nearly 50% of all brain malignancies, with a median survival rate of less than 15 months. Our lab has identified a role for the FOXO4 transcription factor in maintaining a stem-like phenotype in GBM. To further investigate this novel role, we performed RNA sequencing on FOXO4 knockout on U87MG cells that underwent CRISPR-Cas9 mutagenesis generated in the lab. Through this, we found that the loss of FOXO4 in the U87MG cell line altered the expression of NOTCH3 and its downstream targets, like CCND1. Furthermore, exogenous NOTCH3 induced CCND1 expression in U87MG cells. Furthermore, this preliminary evidence suggests that FOXO4 transcriptional activity may influence glioblastoma aggressiveness, at least in part, by modulating NOTCH3 activation. Current studies aim to confirm whether the FOXO4-NOTCH3 axis is essential for maintaining stem-like states in GBM cells. Elucidating this interaction could uncover novel molecular targets for therapeutic intervention and advance our understanding of GBM pathogenesis.

P5

Title: Genomic Evidence for Introgression and Hybridization among North American Camponotus Lineages

Presenter: Swapnil Boyane

Author List: Swapnil S. Boyane, Joseph D. Manthey

Abstract: Hybridization and introgression are common among closely related species with overlapping distributions and are increasingly recognized for their role in shaping evolution. However, these processes are not well studied in Carpenter ants. In this study, we analyzed whole-genome short-read sequencing data from 194 North American carpenter ants in the genus Camponotus, including C. modoc, C. herculeanus, and C. novaeboracensis, to examine gene flow and introgression among these related lineages. We first used ADMIXTURE analysis to determine population genetic structure, which assigned individuals into six genetic clusters. Notably, C. modoc individuals are subdivided into three distinct clusters, along with several admixed individuals from various parental backgrounds and a separate cluster representing a putative hybrid lineage, hereafter referred to as C. hybrid. We also estimated gene trees for 50-kbp non-overlapping windows to infer phylogenetic relationships among sampled individuals, which recovered six major clades, consistent with the population structure observed in ADMIXTURE. Next, we employed f-branch statistics with the Dsuite program to detect introgression, revealing strong gene flow involving the C. hybrid lineage and both C. herculeanus and C. novaeboracensis To explore heterogeneity in phylogenetic relationships across the genome, we used topology weighting with TWISST. Our results indicated that species-level relationships were predominant across most of the genome; however, some topologies grouped C. hybrid with C. novaeboracensis, suggesting localized introgression on scaffold 4. Lastly, we constructed phylogenetic networks using SNaQ, which indicated a hybridization event with gene flow from the C. hybrid lineage into both C. herculeanus and C. novaeboracensis, corroborating patterns observed in earlier analyses.

P6

Title: The Functionality of Transposable Element Derived Enhancers and eRNAs

Presenter: Alyssa Briggs

Author List: Alyssa Briggs, Tae Hoon Kim

Abstract: Transposable elements (TEs) are a subset of repetitive elements that have long been considered non-functional remnants within the genome, i.e., junk DNA. However, many emerging studies are uncovering their critical roles in genome structure, genomic plasticity, and transcriptional regulation. Of particular note, regulatory regions such as enhancers have been found to derive from TE sequences. While the majority of TEs are transcriptionally silenced, processes such as immune activation can lift this repression. TEs within enhancer regions may then undergo transcription into enhancer RNAs (eRNAs) and serve further regulatory purposes that are yet to be explored. The transcription of any TE is a mutagenic risk, holding the potential for indels, duplications, structural inversions, or translocation. However, this risk seems to be acceptable for genome management and plasticity, otherwise there would be little reason for any TE to be transcriptionally active. To fully understand regulation in both healthy and diseased cells, we must explore how historically ignored repetitive elements such as TEs contribute to this landscape. Here we seek to efficiently leverage stores of existing data to uncover discoveries that were previously discarded along with difficult to map TEs. We have created a simple, flexible, and reproducible bioinformatics pipeline capable of identifying and quantifying any repetitive genomic elements from existing repositories of sequencing data utilizing a combination of chimeric read alignment-based localization and alignment-free taxonomic quantification. The pipeline was benchmarked using simulated reads, then used to analyze a GRO-seq viral infection time course data set. We focused our study on LINE-1 (L1), the only TE family with fully intact instances still capable of retrotransposition in humans. Full-length L1s (fl-L1s) offer rich material for the potential creation of new genes and regulatory elements while also carrying the risk of unchecked retrotransposition if transcribed. Over the infection time course, an oscillatory pattern was observed in fl-L1 transcription, starting low and spiking at 6 and 72 hours post infection. This behavior shows similarities to that of NF-κB, a transcription factor that induces cytokine expression during the immune response, suggesting that transcription of fl-L1 behaves akin to regulatory factors that influence cellular response to infection. Furthermore, fl-L1s were mapped to regions overlapping known immune enhancers and showed evidence of bidirectional eRNA transcription. This suggests that some immune enhancers may derive their sequences from TEs and allow those TEs to be transcribed during infection, their contributions outweighing the risk of transposition. We hope that this evidence will inspire further studies into the contribution a diverse range of repetitive elements make to the genome's regulatory machinery and that our pipeline will address a software need within the community to aid in the identification and quantification of repetitive elements - the dark matter of the genome.

P7

Title: Implementation of Visium HD for Single-Cell–Scale Spatial Transcriptomics in FFPE Samples

Presenter: Isabel Castro

Author List: Timothy Jobe, Rozina Vafa, Ana Tobias Porras, Dongming Jiang, Zheyun Niu, Luis Brandi , Manisha Tripathi, Isabel Castro-Piedras

Abstract: Recent advances in spatial transcriptomic technologies have enabled transcriptome-wide profiling at increasingly fine spatial resolution while preserving tissue morphology. However, successful application of these methods to formalin-fixed, paraffin-embedded (FFPE) specimens requires careful optimization of library preparation, sequencing, and data quality assessment to ensure robust and reproducible results. In this study, we describe the implementation and performance of the Visium HD Spatial Gene Expression workflow for FFPE human prostate adenocarcinoma tissue. Visium HD replaces discrete multi-cell capture spots with a high-density, continuous array of 2 × 2 µm barcoded capture features, providing near single-cell spatial resolution across entire tissue sections. This platform enables comprehensive transcript capture without gaps in tissue coverage and supports precise spatial localization of gene expression within complex tumor regions. To maximize data quality from FFPE-derived RNA, Visium HD libraries were sequenced using Aviti™ high-accuracy sequencing technology, which offers improved base-calling accuracy, higher-quality reads, and enhanced transcript detection. We evaluate library complexity, spatial signal integrity, and transcript recovery, demonstrating the feasibility and robustness of this integrated workflow for high-resolution spatial transcriptomic analysis. This study shows that the integration of Visium HD spatial transcriptomics with Aviti sequencing facilitates the generation of high-resolution, high-fidelity spatial gene-expression profiling of prostate cancer tissues. This approach provides single-cell scale resolution, precise transcript localization, and comprehensive transcriptomic coverage.

P8

Title: Rethinking Assessment in Microbiology through Specifications Grading

Presenter: Suparna Chatterjee

Author List: Suparna Chatterjee

Abstract: Assessment plays a crucial role in gauging learner achievement. A specifications-based grading approach was introduced in a microbiology class to measure student performance through a more inclusive framework. This assessment method centers on demonstrating proficiency in clearly defined learning goals, which collectively determine a student's course grade. For the undergraduate Health Sciences Microbiology course, primarily serving pre-nursing students fulfilling a program requirement was designed to assess students' performance around 16 distinct learning objectives (LOs). The approach incorporated two foundational elements: (i) comprehensive feedback on submitted work highlighting strengths and identifying areas requiring further practice or content review, and (ii) multiple opportunities to develop competencies that prove difficult through assignment resubmission. Students' lecture grades reflected the total number of LOs they successfully demonstrated mastery in, while their overall course grade combined their mastered LOs with final examination performance. This assessment strategy yielded encouraging results, with students demonstrating more favorable dispositions toward microbiology content and a notable decrease in unsuccessful course completion rates (grades of D or F, and course withdrawals).

P9

Title: Evaluating the potential role of casein kinase II in *pgm2Δ* galactose-dependent growth defects

Presenter: Karsyn Clouse

Author List: Karsyn R. Clouse, David P. Aiello

Abstract: In *Saccharomyces cerevisiae*, phosphoglucomutase (PGM) is responsible for the interconversion of glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P), with the Pgm2 isoform accounting for approximately 90% of total PGM activity. When the *pgm2Δ* mutant is grown on galactose, it exhibits hyperaccumulation of G1P and altered G1P:G6P ratios relative to wild type. Accordingly, the *pgm2Δ* mutant displays various galactose-related growth defects. Among these defects is impaired calcium homeostasis, but the molecular mechanism underlying this calcium homeostasis deficiency remains unknown. Notably, the calcium homeostasis phenotype is rescued in the *pgm2Δspt4Δ* double mutant. Together with Spt5, Spt4 regulates transcriptional elongation in *S. cerevisiae*, suggesting that transcriptional regulation may be implicated in *pgm2Δ* galactose-dependent growth defects, including calcium homeostasis. One complex potentially related to both calcium homeostasis and the Spt4-Spt5 complex is casein kinase II (CKII). Casein kinase II is a serine-threonine kinase composed of two catalytic subunits, Cka1 and Cka2, and two regulatory subunits. In *S. cerevisiae*, CKII has been implicated in diverse cellular processes, including cell survival, cell cycle regulation, cell polarity, stress responses, transcription, translation, and chromatin maintenance. Although CKII has many substrates and a direct physical interaction with the Spt4-Spt5 complex has not been firmly established in vivo, CKII has been shown to coprecipitate with Spt4-Spt5. Interestingly, Hrr25, a yeast homolog of casein kinase I, inhibits calcineurin signaling through phosphorylation of Crz1; however, it remains unclear whether Crz1 is a shared substrate of both casein kinase I and casein kinase II in *S. cerevisiae*. Additionally, because cka1Δ exhibits sensitivity to multiple environmental stressors, assessing whether these phenotypes are amplified or abated in *pgm2Δcka1Δ* relative to *cka1Δ* may provide insight into the molecular pathways underlying *pgm2Δ* galactose-dependent growth defects. To characterize the role of casein kinase II in the *pgm2Δ* background, the following strains were constructed and spot-plated on various media types, including calcium homeostasis stressors: *cka1Δ*, *pgm2Δcka1Δ*, *spt4Δcka1Δ*, and *pgm2Δspt4Δcka1Δ*. This study characterizes the growth phenotypes of these strains under diverse stress conditions as a preliminary evaluation of the potential role of casein kinase II in *pgm2Δ* galactose-related growth defects.

P10

Title: Breast Cancer-Specific Enhancer RNAs Identified by Integrative GRO-Seq and ChIP-Seq Bioinformatics Analysis

Presenter: Eva Myers

Author List: Eva Myers, Erin Lam, Tanner Steubing, Shayne Easterwood

Abstract: Breast cancer is the most commonly diagnosed malignancy in women. Current therapies lack cell-type specificity, resulting in treatment-associated toxicity and persistent adverse effects. Although breast cancer progression is driven by extensive transcriptional reprogramming, the noncoding regulatory elements responsible for cancer-specific gene activation remain incompletely defined. Active enhancers generate enhancer RNAs (eRNAs), cell-type-specific noncoding transcripts that regulate gene expression. Aberrant eRNA activity has been implicated in cancer-specific transcriptional programs; however, its role in oncogenic regulation in breast cancer remains poorly characterized. Integrative bioinformatics analyses of Global Run-On sequencing (GRO-Seq) and Chromatin Immunoprecipitation sequencing (ChIP-Seq) data identify eRNAs selectively transcribed in breast cancer cells. Breast cancer-specific eRNAs were defined by overlap with active enhancer-associated histone modifications and genomic proximity to cancer-

associated genes. Approximately 45% of the identified eRNAs transcribed in the MCF7 breast cancer cell line are not transcribed in the non-tumorigenic MCF10A breast cell line, providing ample targets for cancer-specific gene regulation. These cancer-specific eRNAs are associated with altered expression in proteins involved in oncogenic processes such as metastasis, immune escape, and angiogenesis. The identification of cancer-specific noncoding RNAs that regulate oncogenic proteins provide an avenue for developing more precise therapeutic strategies for breast cancer with less off-target effects.

P11

Title: Whole Genome Analysis of Rare Equine Congenital and Reproductive Disorders

Presenter: Catherine Fox

Author List: C. R. Fox*, M. N. Mendoza, H. C. Anderson, S. C. Stroupe, B. W. Davis, T. Raudsepp

Abstract: Congenital and reproductive disorders in equines are uncommon but can have significant clinical implications. Through our cytogenetic services, we identified three cases involving rare developmental and reproductive abnormalities in equines: a stillborn foal with no forelimbs, an infertile stallion with complete azoospermia and an infertile stallion with morphologically normal but immotile sperm and chronic respiratory disorders. These cases have been clinically described in detail, and the stallions were previously analyzed using traditional cytogenetic techniques, which are limited in their ability to detect molecular changes. Both infertile stallions were chromosomally normal; however, due to the rare nature of these conditions, we hypothesize that there is an underlying genetic component that remains unidentified. This study aims to utilize whole genome sequencing to identify mutations in key genes associated with reproductive disorders that are undetectable by conventional cytogenetic analysis. Short-read sequencing data was generated for the three cases and compared to a control group of approximately 200 horses. Both candidate gene and hypothesis-free genome-wide approaches were used, filtering by variants of high impact and homozygosity for alternate alleles with <1% frequency. Preliminary analysis began with the stillborn foal and revealed several high-impact mutations in key developmental genes. Investigation of the functional impact of these mutations and analysis of the remaining horses are in progress. This research highlights the power of whole genome sequence analysis for the study of rare congenital conditions and the development of genomics-based personalized veterinary medicine.

P12

Title: Uncovering Key Regulators for the Mitochondrial Ethanol and Stress Response Element and elucidating their role in host defense

Presenter: Sadie Gaskins

Author List: Sadie Gaskins, Yvette Acevedo, Alicia Chan, Lois Armendariz, Elissa Tjahjono, Armando Moreno, Alexey Revtovich, Natalia V. Kirienko

Abstract: Mitochondria play key roles in maintenance of cellular homeostasis through regulation of several biochemical processes. Mitochondrial dysfunction contributes to several pathologies including metabolic disorders, cancer, aging, and neurodegenerative disease. Surveillance pathways recognize mitochondrial stress by monitoring important mitochondrial functions. These pathways activate transcription of genes that restore cellular function. Previous studies in *Caenorhabditis elegans* showed that the evolutionarily conserved mitochondrial Ethanol and Stress Response Element (ESRE) surveillance network, which acts through an 11-nucleotide motif in the promoter region of target genes, is activated in response to reactive oxygen species (ROS) and mitochondrial damage and is required for defense against Pseudomonas aeruginosa liquid-based pathogenesis. Despite ongoing efforts, regulation of this network remains largely unknown. We identified potential regulators of the ESRE network by screening an RNAi library consisting of 1152 transcription factors

and kinases using a transgenic worm strain (3XESRE::GFP) that carries 3 tandem repeats of the ESRE motif. We treated worms with rotenone, which activates ESRE via ROS generation. We identified 8 repressors and 13 activators involved in ESRE regulation. Bioinformatic analyses of these hits revealed connections between ESRE, ER proteostasis, mitophagy, and worm longevity. To assess if our hits were specific to ESRE regulation, we tested their effect on mitochondrial unfolded protein response (UPRmt) and mitogen-activated protein kinase cascade (MAPKmt) under stressed (spg-7 RNAi) and basal conditions. Only 4 of our 21 hits affected activation of these two surveillance pathways, indicating an overall specificity of these hits to ESRE. We also measured the involvement of hits in UPRER activation by tunicamycin using a hsp-4::GFP reporter. Only 2 of our 21 hits affected UPRER activity. Further studies will use chromatin immunoprecipitation (ChIP) to assess if our hits influence ESRE regulation by binding to the ESRE motif. Previous studies in the Kirienko lab showed that disruption of ESRE activity decreased host defense against P. aeruginosa in a liquid-based pathogenesis model. To determine whether our identified ESRE regulators affect host survival under these conditions, we tested the effect of RNAi-mediated gene expression knockdown of each of the 21 hits during liquid-based pathogenesis by P. aeruginosa, S. aureus, and E. faecalis. Preliminary results suggest a connection between ESRE repressors and innate immune pathways involved in host defense. Future work will continue liquid-based pathogenesis experiments to characterize this response. This work aims to elucidate a regulatory network for the ESRE pathway to better understand its role in mitochondrial surveillance and host defense, and implement these tools to modulate mitochondrial dysfunction and response to bacterial pathogenesis.

P13

Title: Effect of Gene Expression Rate on Lactose Metabolism in Genetically Engineered *Saccharomyces cerevisiae*

Presenter: Hector Gonzalez

Author List: Hector Gonzalez, Luka Pravica, Brian Teague

Abstract: Over 150 million metric tons of cheese whey, a byproduct of cheese production, are produced annually with 47% of it being dumped into the environment without prior treatment. Up to 70% of dissolved material in whey is lactose making cheese whey a viable substrate for bioethanol production through fermentation. *Saccharomyces cerevisiae* is commonly used in other industrial fermentations, however, S. cerevisiae lacks the genes required for lactose metabolism. Scientists have attempted to genetically engineer *S. cerevisiae* to ferment lactose through a plasmid containing both a β-galactosidase (LAC4) and a lactose permease (LAC12) gene. Previous work has shown proof of concept, however, the process is yet to be scaled to the industrial level. We hypothesize the rate of gene expression plays a significant role in the efficiency of lactose fermentation, thus understanding this relationship may provide a path to its industrialization. Our approach included designing strains with differing gene expression rates and then measuring their kinetics in lactose and whey based media. We developed 18 strains of *S. cerevisiae*, with the LAC4 and LAC12 genes integrated at the HO locus, using GoldenGate cloning and CRISPR genetic engineering. We were able to show that in the strains containing K.lactis genes the rate of lactose assimilation was dependent on the rate of expression of the recombinant genes and thus supported our hypothesis. We plan to continue our characterization of our K. lactis and K. marxianus derived strains by measuring the kinetics of ethanol production in fermentations.

P14

Title: Investigating Local Tardigrade Species Diversity

Presenter: Delainey Hinson

Author List: Delainey Hinson, Jonathan D. Hibshman

Abstract: Tardigrades are known for their ability to survive extreme stresses, including desiccation, high pressure, radiation, and high temperatures1–4. Although tardigrades are often generally considered to be tolerant of extreme stress, there is considerable variation in resilience across species. By understanding the evolutionary relationships within the Tardigrada phylum, we can gain insight into the evolutionary trajectory for extremotolerance. However, there are several gaps in knowledge regarding these evolutionary relationships, as there is limited information regarding tardigrade species diversity and characterization, particularly when focusing on specific regions. Here we investigate tardigrade evolutionary relationships using an integrative taxonomic approach that combines genetic sequencing and morphological characterization of wild tardigrades, allowing us to document and characterize species diversity on the campus of Southern Methodist University, with the ultimate goal of studying extremotolerance across different species. After sequencing two common molecular markers, 18S rRNA and ITS2, we used neighbor-joining analysis to determine phylogenetic relationships between sixteen wild tardigrades and several control sequences from GenBank. We found that most wild specimens cluster within expected genus-level groupings, validating the molecular identification approach. Through this, we have identified tardigrades from campus that are likely representatives of several genuses, including Macrobiotus, Milnesium, and Mesobiotus. We found that there are several samples within the Macrobiotus clade that form distinct subclades lacking close matches in GenBank, suggesting either the existence of previously undescribed species or inaccurate genus boundaries. This possibility will continue to be explored through our research, using morphological distinctions to aid species identification and characterization, as we had previously focused mainly on genus-level identification. These findings improve our understanding of tardigrade phylogeny and lay the foundation for future investigations into the molecular mechanisms of extremotolerance across tardigrade species.

P15

Title: Determining the role of ADAMTS proteases in TGF-β signaling in *C. elegans*

Presenter: Omma Honey

Author List: Omma Honey, Tina L. Gumienny

Abstract: Abstract The transforming growth factor-β (TGF-β) signaling pathway plays critical roles in regulating animal development, tissue homeostasis, and disease progression. However, how this pathway is regulated between the sending cell and the receiving cell in a time- and tissue-specific manner is not well understood. Some ADAMTS proteases cleave extracellular matrix components (ECM) that control TGF-β ligand activity. It is difficult to study TGF-β signaling in mammalian systems because they have complex, critical, and sometimes redundant functional roles. It is ideal to study TGF-β signaling in the roundworm *C. elegans* system because TGF-β, ADAMTS, and ECM genes are conserved, the mutants are nonlethal, and the system offers practical advantages. In *C. elegans*, DBL-1 is a conserved TGF-β superfamily member and is essential for regulating body size, tissue morphogenesis, and other processes. DBL-1 signaling is modulated by an ECM component, LON-2, which sequesters DBL-1 from its receptors. *C. elegans* has only five ADAMTS proteases, four of which affect a trait that DBL-1 also affects. We will explore the potential of ADAMTS proteases to cleave LON-2 in a regulated manner, facilitating proper DBL-1 signaling during *C. elegans* development in specific times and places. We will determine if ADAMTS proteins cut LON-2 by testing mKate-tagged LON-2 cleavage in ADAMTS mutant backgrounds by western blotting. We will also determine if LON-2 localization and protein levels are altered by loss of ADAMTS gene function using confocal microscopy. Further studies are needed to define the molecular interactions between ADAMTS enzymes, LON-2, and DBL-1, as these insights could enhance our understanding of TGF-β pathway modulation.

P16

Title: Investigating Metabolic Stress Responses in *pgm2*Δ *Saccharomyces cerevisiae* regulated by GAS1 and GRX1

Presenter: Mahima Jetty

Author List: Mahima Jetty, Carrie F. Curtis, David P. Aiello

Abstract: Glucose metabolism is a critical component in maintaining cellular function, and if there are disruptions to carbohydrate metabolism they can cause stress to be imposed on the cell. In *S.cerevisiae*, the PGM2 gene encodes phosphoglucomutase, an enzyme that is involved in glucose metabolism and the production of UDP-glucose, which is a precursor for glycogen biosynthesis. If PGM2 is deleted, metabolic defects are seen as well as broader cellular defects. For example, loss of PGM2 impacts glycogen storage and cell wall homeostasis, creating metabolic and structural stress in the cell. This suggests that losing PGM2 may have an impact on other stress-responsive pathways. To have a better understanding of how the loss of PGM2 causes cellular defects, the roles of GAS1 and GRX1 genes were examined, as these genes β directly function in cell wall maintenance and redox regulation respectively. GAS1 encodes a 1,3 glucanosyltransferase that is required for proper cell wall biosynthesis and remodeling. In stressful conditions, it is possible that disruptions in glucose metabolism may directly affect GAS1-dependent processes because of altered cell wall precursor availabilities and stress responses. On the other hand, GRX1 encodes a glutaredoxin which plays a role in maintaining redox balance through the glutathione system, and therefore is known to respond to both oxidative and metabolic stresses. It is possible that changes in metabolic state can therefore influence redox regulation through GRX1 influenced pathways. GAS1 and GRX1 function in distinct biological pathways, hence if both genes are examined in the context of the *pgm2*Δ mutant, it is possible to see how different stress-response systems respond to metabolic imbalances. Through focusing on cell-wall structure and redox-associated pathways, it is possible to understand how this study contributes to a broader understanding of how disruptions in glucose metabolism affect multiple cellular pathways and functions.

P17

Title: A multi-omics approach to understand the interaction between cotton roots and *Fusarium oxysporum* f. sp. *vasinfectum* (FOV)

Presenter: Timothy Jobe

Author List: Timothy O. Jobe, Mostafa Abdelrahman, Ilham Laadsi, Most Zakia Ferdous Ara Begum, Mauricio Ulloa, Mohamed Fokar, Yehia Mechref

Abstract: Fusarium wilt, a significant and widespread disease in cotton, is caused by the fungus *Fusarium oxysporum* f. sp. *vasinfectum* (FOV). This disease is transmitted through various mechanisms, including the dissemination of spores and mycelium in the soil. Initially, the fungus infects the roots and subsequently spreads throughout the plants via the vascular system, leading to wilting and eventual plant death. Despite the economic importance of FOV in cotton, there is limited knowledge about the molecular signals governing the interaction between cotton roots and the FOV pathogen. The objective of this study is to address this knowledge gap by characterizing the transcriptomic and proteomic profiles of cotton roots in response to FOV4 infection. To identify key genes and proteins involved in FOV4 infection, cotton roots from FOV4-resistant and sensitive genotypes were exposed to FOV4 and differentially expressed genes (DEGs) and proteins (DEPs) were identified at 24 hours, 10 days, and 30 days. In the resistant line, key markers involved in defense against FOV4 include the RNA-binding (RRM/RBD/RNP motifs) family protein and ENTH/VHS/GAT family protein, which exhibited significant upregulation compared to the control. In comparison, in the sensitive line, markers such as Haloacid dehalogenase-like hydrolase and Kelch repeat F-box proteins were the most highly upregulated. These results along with key similarities and differences in the proteomic and transcriptomic data will be presented.

P18

Title: Predicting protein-metal interactions using HoloFold

Presenter: Dimitris Kalafatis

Author List: Dimitris Kalafatis, Abhinav Swaminathan, Vishal Gohil

Abstract: With the advent of novel protein folding prediction software like AlphaFold3, it is becoming increasingly easier to guide experiments based on predicted protein domains, interacting partners, or ligands. However, predicted ligand binding has high false positive rates, and models have difficulty distinguishing different metal binding sites in vivo. To solve this problem, we developed HoloFold, a machine-learning model that incorporates evolutionary, structural, and ligand-specific information to accurately and quickly predict protein-ligand interactions. Holofold has been shown to have extremely high recall even with rare metals. For example, Holofold is able to recall about 20% of true copper-binding proteins at 100% precision in a dataset composed of 5% copper proteins, and 81% "bait" zinc-binding proteins. For reference, using AlphaFold3 on the same dataset and using a cutoff of 0.5 for the ipTM score results in a precision of less than 10% at 20% recall. By applying HoloFold to the human and yeast proteomes for all metals common in biology, we curated a high-confidence metalloproteome with important implications for disease and new biology, as well as providing similar predictions in a simpler model system to confirm evolutionarily conserved interactions. Specifically, by using HoloFold to predict mitochondrial protein interactions with copper, we have identified novel proteins that likely bind copper in vivo, changing what we know about mitochondrial biology.

P19

Title: Analyzing the effect of UV on the expression levels of P2RY6 and SSTR4 two possible regulators of a calcium-dependent chromatin compaction and DNA protection pathway in human melanocytes.

Presenter: Tahree Ladell

Author List: Tahree Ladell, Melanie Enrique, Dr. Michael Bergel

Abstract: Skin cancer is the most common cancer. Exposure to ultraviolet (UV) radiation can damage cellular DNA, leading to mutations and an increased skin cancer risk. Human cells, including melanocytes, the pigment-producing cells in the skin, have mechanisms to respond to UV radiation, including chromatin remodeling for DNA protection. Recent studies suggested that calcium plays a critical role in modulating cellular responses to UV radiation. This study investigates a calcium-dependent pathway involving P2RY6 and SSTR4 receptors that induces UV-dependent chromatin compaction and enhances DNA protection in melanocytes. We show that UV exposure leads to chromatin compaction and genomic stability, by increasing intracellular calcium, activating P2RY6 and SSTR4 signaling, and increasing their transcription levels (based on RNA-sequencing). Our goal is to corroborate these results by Western blotting and RT-qPCR. Our findings highlight the role of calcium-dependent pathways in maintaining DNA integrity, providing insights into potential skin cancer prevention strategies.

P20

Title: Identifying the Functional Role of RLM1 and PST1 Expression in *S. cerevisiae pgm2Δ* Mutants

Presenter: Abagail Larkin

Author List: Abagail Larkin, David P. Aiello

Abstract: Phosphoglucomutase (PGM) is responsible for interconverting glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P) in *Saccharomyces cerevisiae* (*S. cerevisiae*). Previous research has shown that yeast strains lacking PGM2, the gene encoding the major isoform of PGM, exhibit growth defects on a variety of media types. Likewise, disruption of calcium homeostasis in *pgm2Δ* mutants has been observed impacting overall cell health. Previously, RNA-

sequencing was conducted on *pgm2Δ* mutants to analyze genome wide changes in gene expression to explain observed growth defects. It was observed that a variety of genes involved in the HOG and CWI MAPK pathway were upregulated as a result of the loss of PGM2. This upregulation included PST1, a largely uncharacterized gene. PST1 is known to encode for a protein involved in cell wall biosynthesis, however its biological role is unknown. The expression of PST1 is regulated by RLM1. Rlm1p acts as a transcription factor for a variety of CWI genes, and it is regulated by Slt2p, a protein involved in both the HOG and CWI MAPK pathways which was likewise found to be upregulated in the initial *pgm2Δ* RNA-seq data. Previous research in the Aiello lab found that the removal of RLM1 in *pgm2Δ* mutants proved to be lethal on galactose and media types containing oxidative stressors. This suggests that RLM1 and PST1 could be necessary genes in *pgm2Δ* mutants, indicating an important functional role for both genes. This study examined the role of RLM1 and PST1 on *pgm2Δ* mutant growth, as well as investigated the possible cause of overexpression. It was observed that *pgm2Δrlm1Δ* mutants had a lethal phenotype as expected. Lethal phenotypes were likewise observed in *pgm2Δrlm1Δpst1Δ* strains on media containing galactose, cell wall stressors, and oxidative stressors. While the loss of RLM1 showed significant growth defects, removal of PST1 alone showed no significant change in growth to *pgm2Δ* mutants on similar media types. Likewise, overexpression of PST1 in *pgm2Δ* strains showed no growth effects, suggesting PST1 has a neutral impact on *pgm2Δ* growth. Interestingly, RLM1 overexpression had negative/lethal growth phenotypes in both wild type and *pgm2Δ* strains on galactose, cell wall inhibitors, and oxidative stressors. Collectively, our results suggest an important role for Rlm1p-mediated gene expression in adaptation to galactose and responses to cell stress.

P21

Title: Identifying Molecular Differences Between Flat and Polypoid Adenomas in Colorectal Cancer

Presenter: Bella Lawlar

Author List: Bella Lawlar, David Bautz, Joshua Uronis, David Threadgill

Abstract: Flat colorectal adenomas are clinically important precursors to carcinoma that can be easily missed during endoscopic screening, yet the biological basis for why some individuals preferentially develop flat lesions rather than polypoid polyps remains poorly understood. Our central question is: what genetic modifiers affect adenoma growth toward a flat versus polypoid morphology? We hypothesize that inherited variation differentiates the epithelium and the tumor microenvironment (TME) in ways that inhibit vertical growth and remodeling, favoring lateral, flat expansion. Previous work done in the lab identified the percentage of flat polyps and polypoid polyps found in nine mouse strains. Based on these findings, two strains with varying percentage of polyp morphologies were evaluated; KK/HIJ mice, which develop roughly 20% flat polyps, and I/LnJ mice, which develop around 90% flat polyps. Histological and mutational profiling analysis on tumor samples from each strain showed similar activation of nuclear beta-catenin in both polyp morphologies. Similar distributions of beta-catenin stabilizing mutations between both strain and adenoma grade (high and low) were also observed. Because of the histological and mutation similarities between polyps, supervised and unsupervised clustering of RNAseq data using flat and polypoid tumor samples from both KK/HIJ and I/LnJ was performed. Unsupervised clustering revealed that tumor samples were strain-dependent and tumor morphology-specific. Supervised clustering of the normal colon epithelium from both strains showed a wound healing signature in the I/LnJ strain, that has predominantly flat polyps, to be downregulated relative to KK/HIJ. This suggests that the molecular differences between the two polyp types did not lie within the tumor but within the adjacent tissue surrounding the polyp. Using this information, an F2 population was created with KK/HIJ and I/LnJ strains. Of the 275 F2 mice generated, 265 individuals developed polyps following chemical induction with azoxymethane (AOM). These mice were then used to map polyp shape QTLs. Results show that there is high statistical association between genetic markers and the flat morphology on Chromosomes 7 and 9. This data establishes that there is genetic control of the polyp shape independent of the tumorigenesis process. Based on this evidence, we are strengthening this QTL data previously found with a second cross using FVB/NJ and I/LnJ mouse strains to narrow the intervals on Chromosomes 7 and 9, with the FVB/NJ strain producing 10% flat polyps, 50% less than KK/HIJ mice. Future studies will identify candidate genetic modifiers responsible for flat polyp formation and elucidate mechanisms by which the TME modulates polyp morphology following tumor initiation. This work will provide insight for potential biomarkers that can identify populations at risk for flat polyp formation can be more thoroughly screened.

P22

Title: Characterizing desiccation response proteins in *C. elegans*

Presenter: Madison Lester

Author List: Madison Lester, Zhirong Wang, Bailey A. Brown, Jonathan D. Hibshman

Abstract: All organisms encounter stress in their environment. One type of stress is desiccation: the complete loss of water. Most cells and animals cannot survive loss of cellular water; however, some exceptional invertebrates can. We use the model nematode, *Caenorhabditis elegans*, as a system to study mechanisms of desiccation tolerance. There isn't a good in vivo understanding of how organisms like *C. elegans* survive desiccation stress. Lab-generated RNA-seq and proteomics data indicate that many *C. elegans* transcripts and proteins change in relative abundance during desiccation. However, we do not yet know if those proteins are functional or where they might be expressed to convey protection during stress. Therefore, we used reporter strains with fluorescently tagged proteins to confirm the expression changes and to provide more information about the localization of the response to particular cells or tissues. Reporters were crossed into daf-1(e1370) to obtain temperature-dependent constitutive dauer larvae for desiccation. Control and desiccated worms were imaged, and the difference in fluorescence intensity was quantified. This data provides further validation of the upregulation or downregulation of these genes and proteins during desiccation. Now, we will work to find the specific function of these genes for stress survival. We are also investigating the role of another upregulated protein, DUR-1, in stress survival, as well as the general health of *dur-1* worms. As a part of this, I am measuring the brood size of *dur-1* mutants to determine if this gene has an impact on reproduction. There is no significant difference in total brood size, so *dur-1* likely does not affect the reproductive health of *C. elegans*, although it is probable that it is involved in desiccation tolerance. This research provides further insight into specific genes and proteins that have roles during desiccation and opens new avenues for research about desiccation tolerance and mechanisms of stress survival.

P23

Title: Understanding the Genomics of Ecological Speciation in the *Enchenopa binotata* species complex (Family: Membracidae)

Presenter: Satabdi Mandal

Author List: Satabdi Mandal, Daniela Palmer

Abstract: Ecological speciation is the process of new species forming through divergent natural selection in different ecological environments. In this process, populations adapt to niches with minor ecological variation and undergo selection for traits that are best suited to survive new ecological spaces. Under strong ecological selection, certain regions of the genome, involved in adaptation start diverging. Factors like spatial and temporal resource partitioning may cause assortative mating that maintain divergence in these genomic regions. Selection on these divergent genomic regions can keep assimilating until different populations speciate and either ecological or genetic differences make them reproductively incompatible. Within the insect order Hemiptera, phytophagous treehoppers in the *Enchenopa binotata* species complex are a remarkable example of ecological speciation, having adapted to over 20 host plant species belonging to 11 different orders. The *E. binotata* complex first diverged from other Enchenopa species around 17.7 million years ago, but the majority of their diversification has occurred within the last 29 to 120 thousand years ago. It has been posited that the species complex diverged mainly via host-shift adaptation, where they shifted to nearby available host plants and adapted to their phenology with time. Behavioral studies show difference in maturation timing between species, depending on plant phenology, and sexual communication. Genomic studies on phytophagous insects undergoing ecological selection in sympatry in the wild are rare. In this study, we analyze patterns of differentiation in trait loci

corresponding to host plant adaptation and infer patterns of speciation in this recently evolved species complex. First, we generated a de novo genome assembly for *E. binotata* on the Eastern Redbud (Cercis canadensis) using PacBio Hifi data. The final purged assembly was 1.1Gb in length, consisting of 70 contigs, with an N50 of 49.6Mb and 34.05% GC content. BUSCO analysis using the Hemipteran database (hemiptera_odb10) recovered 96.9% complete genes (94.7% single copy and 2.2% duplicated). Taxonomic evaluation using BlobTools2 confirmed that the majority of contigs were assigned to Arthropoda (80.4%), and a few to contaminants like, Bacteroidota (5.23%), Pseudomonadota (1.3%) and Cnidaria (1.96%). Upon running the file through RepeatModeler, a total of 2225 unique repeat families were found in the genome, of which, 134 were LTR (Long Terminal Repeat) families. This assembly serves as a contiguous and comprehensive reference genome for *E. binotata*, and will enable our ongoing studies on genome evolution, genomic differentiation between lineages in the species complex, and adaptation.

P24

Title: Immune cells activate in response to alcohol in the nematode *C. elegans*

Presenter: Amanda Mortensen

Author List: Chelsea J. Webber, Benjamin Clites, Zheng Wu, E. A. Cardona, Amanda C. Mortensen, Jon Pierce

Abstract: To search for novel genetic modifiers of intoxication, we performed a genome wide association for intoxication in *C. elegans*. This pointed to a deleterious variant in the putative nicotinic acetylcholine receptor, *lgc-24*, which may cause hypersensitivity to alcohol. We confirmed a role for *lgc-24* in alcohol responses with several models of intoxication in worm. Deletion of *lgc-24* in an N2 wildtype background caused hypersensitivity to suppression of egg laying which was rescued with expression of the *lgc-24* gene. The l*gc-24* receptor is almost exclusively expressed in coelomocytes, which is a cell type that resembles macrophages and microglia in its endocytic function, but has not been known to activate morphologically. Coelomocytes typically exist in three ovoid pairs distributed evenly along the body. We found that treatment with alcohol caused a dose-dependent activation of the coelomocytes in the N2 control strain. Coelomocyte activation was quantified as unpairing and the presence of ramifying processes. Intriguingly, we also found that wild strains carrying deleterious variants in *lgc-24* have chronically activated coelomocytes even in untreated worms. Finally, we compared the transcriptome of microglia to coelomocytes and found that orthologs of 52.1% microglial genes are expressed in coelomocytes. We conclude that a) *lgc-24* has a critical role in coelomocyte homoeostasis, b) disruption of this receptor leads to cell activation, and c) coelomocytes activate morphologically in response to alcohol exposure suggesting a previously unknown immune function. This research may further our understanding of alcohol-mediated inflammation.

P25

Title: The value of accounting for geomorphic processes in hybrid zones

Presenter: Lluis Mercade Goma

Author List: Lluis Mercade Goma, Joseph Manthey, Ethan Gyllenhaal

Abstract: Hybrid zones represent the moment of truth in the speciation process, where two potential species come together and show how gene flow, selection, and recombination shape their genetic futures and potential for co-occurrence. However, most hybrid zones occur on continents, where complex communities shaped by dynamic ecological niches complicate long-term modeling of their dynamics. Islands provide a good environment to study genetic processes; their closed communities and their strict boundaries make it easier to model the population over time. However, for dispersive species like birds, islands often don't have the space for the consistent parental input necessary for hybrid zones. This limits the scope of analyses that can be performed on incipient species in the natural laboratories that islands represent.

Additionally, although they are easier to model than continents, some islands undergo rapid geomorphic changes that shape the past and future states of the hybrid zones. To understand how geomorphic processes can impact island hybrid zones, we simulated 120,000 years of evolution to replicate the unique hybrid zone of the Fiji Whistler (Pachycephala vitiensis) endemic to Fiji, where two evolutionary lineages from different islands with distinct throat plumage phenotypes meet on a peninsula. We achieved this by generating different maps that try to mimic how sea-level and geomorphic uplift affect the available space of the island. We tested 4 different geological scenarios: static map, Pleistocene sea-level change, uniform uplift across all islands in addition to sea-level change, and island-specific uplift with sea-level change. Furthermore, we developed a method for quantifying the dynamics of this non-traditional hybrid zone. We found that these neutral simulations with a simple genetic basis of phenotypic divergence failed to replicate the observed hybrid zone across the full span of the simulation or to the present day, but in some cases, we did recover scenarios that maintained it for tens of thousands of years.

P26

Title: Genetic Suppressors of Freezing in Dopamine-Deficient *C. elegans*: Potential Therapeutic Targets for Parkinsonian Disorders

Presenter: Oluwatoyin Ogunbi

Author List: Oluwatoyin Ogunbi, Dawn Guzman, Jonathan Pierce

Abstract: The frustrating failure to initiate and maintain motion of body parts–freezing–represents a common problem in Parkinson's Disease (PD) and forms of atypical Parkinson's like Progressive Supranuclear Palsy (PSP) and Corticobasal Degeneration (CBD) that impacts quality of life. While much of the research on Parkinsonian disorders aims to prevent, halt or reverse the damage of dopaminergic neurons, it would be beneficial in the meantime to alleviate symptoms even in the presence of neurodegeneration. Our lab has modeled the freezing of gait symptom of many Parkinsonian diseases in *C. elegans*. Previously, we found that the *C. elegans* traverses its environment in two ways, swimming and crawling. Wild-type worms are able to switch between the two with little difficulty. In worms where dopamine signalling is disrupted, though, this change between motions is defective, causing them to freeze their posture temporarily when switching from swimming to crawling forward. By conducting an unbiased forward genetic screen in dopamine deficient worms, we isolated four strains with rescued motor ability. By genetic sequencing we identified eight candidate genes, seven of which were novel in the context of Parkinsonian disorders. The implications of our findings are that some of these candidate genes represent molecules that could be modulated to causally suppress the motor deficit. We are in the process of independently confirming the genes and their molecular pathways, which could potentially be used for development treatments for even late-stage PSP/CBD when dopamine neurons are mostly degenerated.

P27

Title: Balancing NADPH Supply and Demand: PGM2, GRE3, and GLR1 Cooperatively Shape Stress Responses on Galactose in *Saccharomyces cerevisiae*

Presenter: Lily Ordoñez

Author List: Lily I. Ordoñez, David P. Aiello

Abstract: Phosphoglucomutase 2 (Pgm2) links galactose metabolism to glucose-6-phosphate (Glc6-P) production in the Leloir pathway and thereby feeds the oxidative pentose phosphate pathway (PPP), the major source of cytosolic NADPH in *Saccharomyces cerevisiae*. Loss of PGM2 (*pgm2Δ*) causes slow growth on galactose, accumulation of glucose-1-phosphate $^{+}$ (Glc-1-P), and defects in Ca^{2} homeostasis, but the mechanisms connecting altered sugar phosphate metabolism to stress sensitivity remain unclear. Because many stress protective enzymes consume NADPH, we focused

on two NADPH-dependent systems that are upregulated or implicated in *pgm2Δ*: GRE3 and GLR1. Gre3 is an aldose reductase that can reduce galactose to galactitol and detoxify reactive carbonyls, while Glr1 is the glutathione reductase that regenerates reduced glutathione (GSH) from oxidized glutathione (GSSG) and helps maintain a highly reduced intracellular redox state. Together, these enzymes represent major NADPH sinks during carbonyl, oxidative, and general stress. To better understand how NADPH "supply" and "demand" interact, we constructed wild-type and *pgm2Δ* strains carrying an empty control plasmid (pRS316) or overexpressing GRE3 (pGRE3) or GLR1 (pGLR1). We then compared the growth of these six strains on glucose and on galactose media under a panel of stresses, including increased galactose, oxidizing and reducing agents, osmotic stress, the reactive dicarbonyl methylglyoxal (MG), and calcineurin inhibition by cyclosporin A (CsA), using spot plate assays. Across conditions, a consistent growth pattern emerged. On glucose, all strains grew similarly, indicating that neither *pgm2Δ* nor GRE3/GLR1 overexpression is intrinsically lethal. On galactose, however, the wild-type control strain was the most resilient, strains with either a hypothesized reduced NADPH supply (*pgm2Δ*) or increased NADPH demand (GRE3 or GLR1 overexpression) showed intermediate growth defects. Strains that combined *pgm2Δ* with GRE3 or GLR1 overexpression were the most sensitive to extracellular stressors. This three-tier pattern was observed as galactose concentration increased and under multiple stress conditions that are known or expected to be involved in NADPH- and glutathione-dependent detoxification pathways. This is consistent with the hypothesis that limiting Pgm2-dependent PPP flux or adding extra NADPH-consuming enzymes narrows the "NADPH budget" and reduces stress tolerance. In the presence of CsA, the wild-type control strain still grew, but all strains carrying either *pgm2Δ* or GRE3/GLR1 overexpression were strongly impaired. Wild-type strains overexpressing GRE3 or GLR1, as well as *pgm2Δ* + pRS316, failed to grow, whereas *pgm2Δ* strains overexpressing GRE3 or GLR1 showed only very poor but detectable growth. These results suggest that when calcineurin signaling is blocked, any major imbalance in NADPH supply or demand greatly increases sensitivity, and that Gre3or Glr1-dependent detoxification pathways can slightly modify, but not fully rescue, the $^{+}$ severe Ca^{2} - and redox-related stress present in *pgm2Δ* cells. Together, these findings support a working model in which Pgm2 impacts NADPH supply by regulating galactose derived Glc-6-P entry into the PPP, while Gre3 and Glr1 shape NADPH demand through carbonyl reduction and glutathione recycling. When supply and demand are balanced, cells tolerate galactose and diverse stressors; when supply is restricted and/or demand is elevated, cells become hypersensitive to oxidative, reductive, osmotic, carbonyl, and calcineurin-related challenges.

P28

Title: Modeling background dependent tumor growth in response to ERBB3 inhibition using mouse derived organoids

Presenter: Wyatt Porter

Author List: Wyatt W. Porter, Kaitlyn E. Carter, David W. Threadgill

Abstract: Colorectal cancer (CRC) is the second leading cause of cancer-related death worldwide, highlighting the urgent need for effective therapeutic strategies. This project investigates one such potential target, ERBB3, by examining differences in cell viability and proliferation in response to ERBB3 inhibition in C57BL/6J (B6) and 129S1/SvlmJ (129) $Apc^{Min/+}$ mice. Using mouse-derived organoids to model CRC, we aim to explore why preclinical studies suggested ERBB3 inhibition could reduce polyp number, whereas clinical trials have shown limited success, emphasizing the potential role of genetic context in therapeutic outcomes. Organoids from both strains were generated and subsequently treated with EGFR, ERBB3, and dual EGFR-ERBB3 inhibitors. Growth rate inhibition, proliferation, and cell viability assays are performed to measure and validate long-term kinetic assays using live cell imaging systems. We expect to see increased cell proliferation and increased cell viability in B6 $Apc^{min/+}$ mice and decreased cell proliferation and decreased cell viability in 129 $Apc^{min/+}$ mice, to support previous in vivo analyses. This research will provide increased evidence that background differences between strains can impact outcome. Furthermore, by conducting this experiment with organoids instead of a mouse model, we plan to demonstrate that organoids can add robustness to a study when combined with the mouse model.

P29

Title: Uncovering Mitophagy-Dependent Mechanisms of SSRI-Mediated Neuroprotection in Neurodegenerative Disease Models

Presenter: Olivia Reed

Author List: Olivia A. Reed, Daniel M. Hong, Alical M. Chan, Natasha V. Kirienko

Abstract: Selective serotonin reuptake inhibitors (SSRIs) are widely-prescribed antidepressants, including being frequently used by individuals with neurodegenerative diseases (NDDs). Although traditionally viewed as solely limiting ser-otonin reuptake, emerging evidence indicates that SSRIs also influence mitochondrial health, lipid metabolism, proteostasis, and neuroinflammation. Understanding these non-canonical roles is particularly important in NDDs, where mitochondrial dysfunction and impaired proteostasis are major pathogenic hallmarks. Clinical and preclini-cal studies report heterogeneous outcomes following SSRI treatment in NDD contexts, suggesting that these non-canonical effects from SSRIs may differentially modulate disease progression, likely depending on idiosyn-cratic genetic and metabolic backgrounds. Neuronal health depends on tightly-regulated mitochondrial function due to these cells' exceptionally high ener-getic demands. This mitochondrial quality control is maintained by mitophagy, a selective autophagic pathway canonically initiated by stabilization of PTEN-induced kinase 1 (PINK1) on damaged mitochondria, licensing their removal. Numerous studies showed that mild stimulation of PINK1-dependent mitophagy can be neuroprotective, whereas impaired mitophagy accelerates neurodegeneration. The Kirienko lab used the model nematode *Caenorhabditis elegans* to screen ~45,000 small molecules to identify those that increase level of PINK-1. Of eight hits, three increased motility in a *C. elegans* model of Alzheimer's disease, with the strongest hit being sertraline, an FDA-approved SSRI commonly prescribed to treat depression. Given this novel interaction between sertraline and PINK-1, and the conflicting literature on SSRI effects in NDDs, we seek to identify conserved pathways through which SSRIs alter neuronal mitochondrial quality control, metabolic state, and proteostasis, and to de-termine how these pathways contribute to neuroprotection or neurotoxicity. In our study, we discovered a PINK-1-dependent, SSRI-mediated A) reduction in protein aggregation and B) lipid depletion in *C. elegans*. Given that both phenotypes required PINK-1, we hypothesized that SSRI-induced lipid depletion and aggregation reduction are mechanistically linked. To test this, we used sertraline as an initial SSRI and screened for candidate pathways mediating these effects. Knock down of PINK-1-associated genes as well as components of the insulin/IGF-1 signaling pathway revealed a significant increase in fat accumulation in ser-traline-treated worms. In contrast, knock down of the β-oxidation pathway showed that fat stores were still strong-ly depleted in sertraline-treated worms, suggesting increased β-oxidation is not the cause of SSRI-induced fat loss. Furthermore, we found this effect to be largely independent of serotonin increases as its supplementation had little impact on fat content and polyQ aggregation. Sertraline treatment decreases basal levels of oxidative stress, oxygen consumption rate, and ATP in a PINK-1-dependent manner, consistent with altered mitochondrial quality control and metabolic output. Supplementation with oleic acid, a major substrate for triacylglycerol syn-thesis, fully rescued sertraline-induced reductions in oxidative stress, lipid stores, and protein aggregation. Ser-traline was found to decrease nuclear localization of MDT-15, a transcription factor that regulates oleic acid syn-thesis, suggesting sertraline-induced alterations in metabolism are due to disruptions in lipid synthesis. Our re-sults reveal a previously unrecognized, PINK-1-dependent mechanism of SSRI action in which impaired lipid synthesis and mitochondrial metabolic remodeling coordinately drive lipid depletion and protein aggregation re-duction, which will provide a framework for understanding the context-dependent outcomes of SSRIs in NDDs.

P30

Title: ASD risk-allele increases penetrance of uncommon individual behavioral trait in *C. elegans*

Presenter: Lauren Rosta

Author List: Rosta L, Wulffraat G, Hernández P, Iyer S, Wang L, Brumback A, Pierce J.

Abstract: Autism spectrum disorder (ASD) is a condition caused by genetic and environmental factors and characterized by atypical behaviors, including increased prevalence of motor stereotypies. DLG4 synaptopathy is a rare neurogenetic disorder where mutations in the gene DLG4, also known as PSD95 or SAP90, lead to global developmental delay, intellectual disability, and ASD in patients. We used the nematode *Caenorhabditis elegans* to model the T611I variant in the DLG4 ortholog *dlg-1*. During phenotyping, we found that some worms intersperse typical dorsoventral swimming bends with left-right bends, resembling an orchestra conductor's arm motions. This conducting behavior is significantly increased in *dlg-1* mutant worms. The high proportion of conducting in *dlg-1* is recessive and rescuable with the wild-type gene. Conducting phenocopied with another DLG4 patient variant and the proportion of conductors increased when worms were raised on textured "3D" plates. This provides an example of autism variants increasing the proportion of a low-penetrant individual behavior and illustrates a potential interaction between this behavior and sensory experience. We plan to screen for pharmacological agents which alter conducting rates and may lead to potential therapeutics for patients with DLG4 synaptopathy.

P31

Title: Synthetic Morphogenesis in *Saccharomyces cerevisiae* using human Epidermal Growth Factor (hEGF) and Signal Transducer and Activator of Transcription (STAT)

Presenter: Hazel Ruibal

Author List: Hazel Ruibal, Autumn Schmitt, Brian Teague*

Abstract: Waitlists for organ donations consistently surpass donor availability, even in the most medically advanced countries. To address this issue, there has been a long-standing effort to develop artificial tissues and organs. These require cells that communicate and coordinate properly; however, current bioengineering methods cannot yet achieve this. To overcome this, our research looks to *Saccharomyces cerevisiae* (baker's yeast). In these well-studied cells, we are implementing cell-to-cell communication using the hormone human epidermal growth factor (hEGF). We are developing different yeast strains that can both sense and synthesize the hormone. In humans, certain genes express the hEGF receptor and the proteins that carry the signal into the nucleus (STAT3β and STAT5α). We are implanting these genes into our yeast and testing subsequent strains. To test this pathway, we are introducing hEGF into the yeast environment and monitoring the gene expression via fluorescence. We are also assessing the varying intensities across different hEGF concentrations. By engineering our yeast to participate in human cellular communication, this research lays the groundwork for synthetic tissues to behave more like organs. This could eventually enable the creation of synthetic organs with coordinated cellular function, reducing the dependency on human donors and leading to more people getting the help they deserve.

P32

Title: Comparative genomic insights into neo-sex chromosome formation and evolution in treehoppers

Presenter: SUKANYA SAMADDAR

Author List: Sukanya Samaddar, Diogo C. Cabral-de-Mello, Daniela H. Palmer Droguett

Abstract: Sex chromosomes have a crucial role in sex determination and reproduction in many organisms, but their evolutionary origins are surprisingly diverse. Sex chromosomes begin as an identical pair of autosomes and often differentiate from one another forming heteromorphic sex chromosome systems (e.g., XX/XY). These heteromorphic systems are hypothesized to act as evolutionary traps that prevent subsequent evolutionary transitions of the sex chromosome system. A group of hemipteran insects known as treehoppers carry newly formed sex chromosomes, or neo-sex chromosomes, that are the product of chromosomal fusions between the ancestral sex chromosome and autosomes. While cytological studies indicate the presence of neo-sex chromosomes, their genomic identities remain unknown. In this study, we investigated the evolutionary origin of neo-sex chromosomes in *Amblyophallus exaltatus* using a genomic coverage approach. We obtained male and female DNA sequence reads and mapped them to the *A. exaltatus* genome. Furthermore, the relative male to female coverage ratios were calculated across the genome to infer autosomal and sex-linked sequences. Here we tested whether neo-sex chromosomes occur by an autosomal fusion or any other chromosomal rearrangements. We also test whether the *A. exaltatus* neo-sex chromosomes and the neo-sex chromosomes of another treehopper convergently recruited the same autosome, or if these evolved by fusions involving different autosomes. This study addresses how neo-sex chromosome systems arise by X-autosome fusion and the role of chromosomal fusions in the dynamic evolution of sex chromosomes.

P33

Title: A Biosensor for Measuring Mitochondrial Outer Membrane Permeabilization in Humanized Yeast Strain

Presenter: Mark Shklovskiy

Author List: Mark Shklovskiy, Siddharth Nelakurthi

Abstract: Dysregulation of apoptosis plays a pivotal role in tumor malignancy across various cancers and degenerative diseases. The initiation of apoptosis, mitochondrial outer membrane permeabilization (MOMP), is regulated by members of the Bcl-2 protein family. Upon activation, proapoptotic proteins insert themselves into the mitochondrial outer membrane, creating proteinaceous pores that allow the release of factors that lead to cell death. Despite extensive research, the details of the protein-protein interactions governing MOMP remain incompletely understood, largely due to the complexity of the network of interacting Bcl-2 proteins. To unravel these complex interactions in isolation, we sought to engineer a simplified eukaryotic model in the yeast *Saccharomyces cerevisiae*. This yeast was chosen because it has only one Bcl-2 protein,YBH3, allowing for a more controlled study of Bcl-2 protein-protein interactions. In order to measure MOMP in this yeast strain, we developed a fluorescence-based biosensor that detects mitochondrial membrane rupture using a split fluorescent protein (sFP). In this system, mitochondrial matrix protein AIF, which translocates to the nucleus upon release from the mitochondrial intermembrane, is tagged with the β11 subunit of the sFP. During MOMP, tagged AIF exits the mitochondria and fuses with a nuclear-localized β1-10 subunit, producing a detectable fluorescence signal. Our results show that upon hydrogen peroxide-induced apoptosis, cell death occurred, as evidenced by a growth assay. We also observed a ~10-fold increase in fluorescence after inducing apoptosis. These results suggest that our designed yeast system detects MOMP, making it a promising tool for studying Bcl-2 protein interactions. Understanding these interactions could help to develop cancer treatments that restore or enhance apoptotic signalling in cells where it has been dysregulated.

P34

Title: Between genes and species: Insights into the mystery of *Sceloporus formosus* group lizards from the Middle American landscape (Squamata: Phrynosomatidae)

Presenter: Biraj Shrestha

Author List: Biraj Shrestha, Diego Iván Sánchez Aguilar, Victor Gabriel Castillo-Sanchez, Uri O. García-Vazquez, Eric N. Smith

Abstract: Clarifying species boundaries remains one of the essential tasks to undertake among evolutionary biologists. However, with the application of large-scale genomic data, it has been useful to understand the species boundaries across widespread taxa uncovering cryptic speciation, rapid diversification, and intraspecific variation. Spiny lizards from the genus Sceloporus are rich in species diversity, containing 119 species arranged into 18 groups, with *S. formosus* group being the largest, accommodating 18 species and multiple populations. The *S. formosus* group is widely distributed from southern Mexico to Panama, across a varying elevational gradient from sea level to 3600m. There is great phenotypic variation in this group of lizards, though closely related species are difficult to distinguish based on appearance alone and establishing accurate species boundaries has always been a challenging task for systematists. Researchers have suggested that species within the *S. formosus* group were engaged in hybridization. Preliminary analyses based on mitochondrial sequences and morphology have inferred the existence of some level of hybridization involving *S. adleri*, *S. stejnegeri*, *S. scitulus*, and *S. formosus* populations. However, the complete molecular underpinnings of hybridization among members of the *S. formosus* group have not been elucidated. Overall, no studies have yet explored the population genetics among members of *S. formosus* group, that could potentially offer insights into genetic diversity and structure across geographical landscapes. Such remarkable events and paucity of data present an interesting case for understanding their evolutionary processes. Based on the background information, some research questions pertaining to this study are 1) How does genetic differentiation vary within and among populations of the *S. formosus* group? 2) What is the extent of hybridization between different species within the group? 3) How should we define species boundaries and how many species should we recognize? For this project, we performed Restriction-Site Associated DNA (RAD) sequencing to generate the genome wide single nucleotide polymorphism (SNP) data. We utilized >250 tissue samples stored at the Amphibian and Reptile Diversity Research Center (ARDRC) of The University of Texas at Arlington (UTA) for molecular lab work. Dataset generated by RAD-seq were used for conducting analyses pertaining to population genetics and phylogenetics. SNP-based analyses revealed strong genetic structuring across populations with limited hybridization events. Phylogenetic analysis based on nuclear DNA data revealed distinctly evolving multiple clades of taxa within *S. formosus* group with confirmed instances of hybridization between *S. adleri* populations and *S. scitulus* western populations on overlapping regions. Furthermore, the *S. malachiticus* complex revealed the presence of three different taxa: western Honduras clade as *S. schmidti*, eastern Honduras and western Nicaragua clade as *S. hondurensis*, and lower Costa Rican clade as *S. malachiticus*. In near future we plan to analyze population genetic parameters for each population along with other computations such as introgression analysis, cline analysis, hybrid index analysis and phylogenetic analyses incorporating hybridization events. Our study will refine taxonomic understanding, offer insights into population genetics of these lizards, with conservation implications and advance the knowledge of *S. formosus* evolution.

P35

Title: Validating Foxo1 Target Genes in C2C12 Myoblasts

Presenter: Miranda Sifuentes

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Abstract: Foxo1 is a transcription factor that plays a critical role in regulating muscle metabolism, maintaining progenitor states, and guiding myogenic differentiation. However, the downstream targets through which Foxo1 influences nuclear architecture and cytoskeletal organization during myoblast differentiation remain incompletely understood. In this study, we investigated the effects of Foxo1 knockdown on selected target genes and nuclear morphology in C2C12 mouse myoblasts. C2C12 cells were infected with lentivirus encoding Foxo1 RNAi or control constructs and induced to differentiate. RT-qPCR was performed to validate the six Foxo1 target genes identified from RNA-sequencing data, including *Sh2d1b1*, *Zfp984*, *Mapt*, *Pax7*, *Ucp2*, and *Nup210*. Gene expression changes were compared between control and Foxo1 knockdown cells, and nuclear morphology/cytoskeletal organization were assessed using DAPI and phalloidin staining. Foxo1 KD resulted in significant transcriptional changes in multiple target genes. Most notably, *Nup210* and *Zfp984* were consistently upregulated, while *Sh2d1b1* and *Ucp2* were downregulated. The upregulation of *Nup210*, a structural component of the nuclear pore complex, suggests that Foxo1 may normally repress nuclear pore - associated genes. Microscopy also revealed the elongation of nuclei and increased F-actin organization in differentiated *Foxo1* knockdown cells, further reinforcing Foxo1's role in regulating nuclear remodeling during myogenesis. These findings suggest that Foxo1 may influence nuclear organization through targets like *Nup210* and may connect metabolic signaling, transcription, and nuclear architecture. Future studies will further characterize Foxo1-dependent changes in nuclear morphology and validate additional targets involved in nuclear pore regulation.

P36

Title: Nuclear Argonaute, HRDE-1, is required for oocyte-based fertility during heat stress in *C. elegans*

Presenter: Shreya Tantry

Author List: Shreya Tantry, Alicia K. Rogers

Abstract: Post-transcriptional gene silencing in *Caenorhabditis elegans* occurs when small RNAs, like small interfering RNAs (siRNAs), are loaded into Argonaute proteins like HRDE-1. The siRNAs and Argonaute proteins form the RNA-induced silencing complex (RISC), functioning in the nucleus or the cytoplasm. HRDE-1 functions in the germ cells by recruiting chromatin-modifying complexes to target sites for the deposition of repressive transmethylation marks on Histone H3 at Lysine 9 (H3K9me3), which ultimately prevents transcription, thereby silencing genes. HRDE-1 is therefore key in the development of the *C. elegans* germline. Brood sizes of the wild type (N2) and *hrde-1* hermaphrodites were observed under standard temperature conditions (20°C) and under heat stress (25°C). It was observed that heat-stressed hrde-1 animals went sterile and exhibited the mortal germline phenotype in 3–5 generations. The fertility of *hrde-1* hermaphrodites was not rescued when mated to wild-type males. This suggests that the HRDE-1 Argonaute is essential for the oocyte-based fertility of *C. elegans* during heat stress.

P37

Title: Investigating the effects of a Myhre syndrome SMAD4 mutation on TGF-β signaling using a *C. elegans* model

Presenter: Ricardo Umanzor

Author List: Ricardo Umanzor, Tina L. Gumienny

Abstract: Myhre syndrome is a rare genetic disorder with symptoms that include fibrosis and structural issues that result in short stature, characteristic facial features, and cardiovascular and respiratory defects. Myhre syndrome is caused by a missense mutation in the SMAD4 gene, an essential part of the transforming growth factor-beta (TGF-β) signaling pathway. Nucleotide substitutions occurring in Myhre Syndrome patients occur at one of two amino acids in SMAD4, either at R496 or I500, in the SMAD4 MH2 protein-protein interaction domain. However, it is debated whether these

variations cause a loss or gain of SMAD4 function, which is important to understand for possible therapeutic intervention. In *C. elegans*, the two amino acids altered in SMAD4 in Myhre syndrome are conserved in SMA-4. Variations in TGF-β signaling have clear phenotypes. Using CRISPR, we generated *C. elegans* mutant strains with the Myhre syndrome R496 or I500 variations (*sma-4(tex1[R511C]*) and *sma-4(tex4[I517V]*), respectively). First, we analyzed body length, a dose-dependent trait controlled by DBL-1/TGF-β. Increased DBL-1/TGF-β signaling results in a longer body, and decreased DBL-1/TGF-β signaling results in a small body. Interestingly, *sma-4(tex1)* animals displayed an average body size. To determine if DBL-1/TGF-β signaling is altered in these animals, we have created a panel of *sma-4* variant strains expressing fluorescent DBL-1/TGF-β pathway reporters. Results from this experiment will be presented. This and future work will provide foundational insights on how this mutation alters SMA-4/SMAD-4 function and how these SMAD-4 variations cause traits associated with Myhre Syndrome.

P38

Title: Galactose as a metabolic stressor in *Saccharomyces cerevisiae*

Presenter: Kailey Vick

Author List: Kailey M. Vick, Amanda Vanderford, David P. Aiello

Abstract: Carbohydrate metabolism is an essential cellular process that provides substrates for both energy metabolism and cellular biosynthetic pathways. Phosphoglucomutase (PGM) is a key enzyme that sits at the intersection of glucose and galactose metabolism. In *Saccharomyces cerevisiae*, the major isoform of PGM is encoded by PGM2 which accounts for 90% of total cellular PGM activity. In *pgm2*Δ mutants, galactose metabolism is disrupted and cells hyperaccumulate the intermediate sugar phosphate glucose 1-phosphate and have a resulting cellular imbalance of glucose 1-phosphate and glucose 6-phosphate. Concomitantly, *pgm2*Δ mutants exhibit a wide variety of phenotypes including sensitivity to the calcineurin inhibitor cyclosporin A, increased calcium uptake and accumulation, glycogen hyperaccumulation, and they exhibit an increased unfolded protein response. They also exhibit increased sensitivity to osmotic, temperature, oxidative, and ionic stressors as well as sensitivity to DNA damaging and cell wall damaging agents. To understand these phenotypes, prior work in the lab performed RNA sequencing. Data analysis indicates that *pgm2*Δ mutants show increased expression for a large number of genes involved in cellular stress response pathways. Additional work with knockout alleles of transcriptional regulators for these pathways often result in lethal phenotypes when combined with the *pgm2*Δ mutation. The data collectively suggest the intriguing hypothesis that *pgm2*Δ phenotypes result from hyperactivation of normal cellular stress responses that occur as yeast cells adapt their metabolism during the carbon source switch from glucose to galactose. To investigate this hypothesis, we examined whether stress responsive genes show increased expression in wildtype cells metabolizing galactose versus glucose.

P39

Title: AEDES AEGYPTI - LIVERPOOL LABORATORY COLONY PANGENOME PROJECT

Presenter: Natalie Wideman

Author List: Natalie Wideman, Zach N. Adelman

Abstract: Aedes aegypti mosquitoes, native to many parts of Texas, are a vector species that transmit dangerous pathogens for diseases such as Zika, Dengue, Yellow Fever, and Chikungunya. *Ae. aegypti* - Liverpool (LVP) is a standard lab-bred population used in vector research. This project aims to build a Pangenome to evaluate and represent the local laboratory population of *Ae. aegypti* used in vector research at the Adelman Lab. The goal is to develop a comprehensive reference for the local laboratory population to accurately predict target sites for successful genome editing. PacBio sequencing technology was used to develop the current reference genome for *Ae. aegypti* - LVP, AaegL5.0 (GCF_002204515.2), first

published in 2017. Although this assembly is of high quality, it has limitations in identifying regions of the genome conserved within specific laboratory populations. Using pooled *Ae. aegypti* - LVP embryos from the laboratory population, sequencing data was generated using PacBio High-fidelity (HiFi) long-read sequencing. The current focus is to estimate haplotype diversity within the population before assembling the final pangenome. Initial work has focused on developing the analytical framework and pipelines. Preliminary analyses indicate that this data is sufficient to capture different haplotypes relevant for genome editing targets, such as the M-locus gene, nix. Further research will expand on these findings.

P40

Title: Characterizing the role of Yap1 in *S. cerevisiae* mutants lacking PGM2 under cellular stress

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Abstract: In *Saccharomyces cerevisiae*, the enzyme phosphoglucomutase (PGM) allows for interconversion between glucose-1-phosphate (G1P) and glucose-6-phosphate (G6P). When grown on galactose, cells lacking Pgm2p, the major isoform of PGM, display an accumulation of G1P relative to G6P, in comparison to wild-type (wt) cells. Additionally, the *pgm2*Δ mutant displays calcium homeostasis defects with sensitivity to cyclosporin A, a calcineurin inhibitor. Phenotypes of *pgm2*Δ mutants on galactose-containing media have also demonstrated defects with unfolded protein response (UPR), heat sensitivity, and cell wall stability. The current working model of the lab hypothesizes that the altered carbohydrate ratio within the *pgm2*Δ mutant causes abnormal accumulation of calcium within the cell, hyperactivating stress signaling pathways. Genes upregulated in the *pgm2*Δ mutant were identified and analyzed using RNA-Seq, DESEQ-2, and DREME to determine potential transcription factors contributing to these hyperactive stress responses. Yap1, a key transcriptional activator of antioxidant genes was identified from this screen. While the removal of transcriptional elongation regulator Spt4p from *pgm2*Δ mutants has consistently suppressed lethality related to calcium homeostasis and UPR induction defects, a lethal impact on the *pgm2*Δ*yap1*Δ mutant was observed under oxidative stress. The removal of antioxidant regulatory gene HYR1 in the context of *spt4*Δ and *yap1*Δ displayed a normal growth phenotype. To further examine the role of Yap1p in the *pgm2*Δ mutant, this project is using green fluorescent protein (GFP) tagging to determine if there is increased nuclear localization of Yap1p in *pgm2*Δ cells metabolizing galactose.