2026 Yale Postdoctoral Association Symposium

Location: OC Marsh Lecture Hall

Date: June 5, 2026

Registration & Poster Setup
8:00 am - 8:30 am.

Opening session ceremony
8:30 am - 8:45 am.
Chairs: Nebal Abu Hussein & Eunate Gallardo (Co-coordinators)

Session I: Oral Presentations
8:45 am - 9:25 am.
Format: 4 talks (8 min presentation + 2 min Q&A)
Chair: Cameron Gardner

Time	Presenter	Title
8:45–8:55	Jeadran Malagón Rojas	Persistent inequities in pneumoconiosis mortality in Colombia's mining regions: a 25-year ecological analysis
8:55–9:05	Eman Salih	Assessment of War-Induced Structural Damage to Pharmaceutical Infrastructure in Sudan Using Remote Sensing and GIS Analysis: Implications for Essential Medicines Availability
9:05–9:15	Chloé Bâtie	Pathway toward consensus: Lessons from the WHO Pandemic Agreement negotiations
9:15–9:25	Clare Tonks	William Bullock: A Natural History Showman

Keynote Lecture

9:30 am - 10:30 am

Mónica I. Feliú-Mójer, PhD. "Identity as a Superpower: On Belonging and Building a Career That's Yours"

Session hosts: Nebal Abu Hussein & Eunate Gallardo

Coffee and Networking

10:30 am - 11:00 am.

Session II: Oral Presentations

11:00 am - 12:15 pm.

Format: 7 talks (8 min presentation + 2 min Q&A)

Chairs: Meredith Gwin & Devika Gireesan

Time	Presenter	Title
11:00-11:10	Claire Laxton	Herpesviruses and their role in Long COVID
11:10-11:25	Swapnil Chandrakant Devarkar	Redefining the mechanism of action of tetracyclines
11:25-11:35	Lin Li	KLF15 Acts as a Brake on Adipocyte Progenitor Expansion
11:35-11:45	Suparna Basu	Targeting metabolic–DNA repair vulnerabilities in SDHB- and FH-deficient renal cell carcinoma with the novel alkylating agent KL-50
11:45-11:55	Chetna Dhembla	Insights into the nuclear import of 26S proteasomes using a nuclear pore complex (NPC) mimic system
11:55-12:05	Linhui Xie	Benchmarking generative scaffold design methods for peptide engineering in TCR–MHC complexes

12:05-12:15	Michael Chung	Discovering interpretable low-dimensional dynamics from high-dimensional scientific data

Lunch and Poster Session

12:15 pm - 2:15 pm.
Chairs: Tianyu Zhu & Chloé Bâtie

Session III: Oral Presentations

2:15 pm - 3:15 pm.
Format: 6 talks (8 min presentation + 2 min Q&A)
Chairs: Jesslyn Hariyanto & Bingqing Yao

Time	Presenter	Title
2:15 - 2:25	Fatma Zehra Calikusu	Multi-locus genetic dosage shapes cognitive disease progression in Parkinson's: 15-year meta-analysis of 24 cohorts
2:25-2:35	Buket Esen	Evaluating the Effect of Continued FIT Screening: A Target Trial Emulation Using the Clone–Censor–Weight Method
2:35-2:45	Amit Kumar Nagwani	Cryptobiosis and Aging: A Multilevel Framework for Studying Stress Resilience in Tardigrades
2:45-2:55	Alexandra Scalici	Phenotype risk scores identify distinct clinical features and genetic correlates of eating disorders
2:55-3:05	Siyuan Cheng	ATHENA: A deep learning–based AI for functional prediction of genomic mutations and synergistic vulnerabilities in prostate cancer
3:05-3:15	Anne-Marie Rickmann	Using Foundation Models as Pseudo-label Generators for Pre-clinical 4D Cardiac CT Segmentation

Cultural performance

3:15 pm - 3:45 pm.

Session host: Chetna Dhembla

Performances: Shenhui Liu – *Chinese Classic Rock Song*; Yuting Wang and Grayce – *K-pop Dance Performance*; Anmol Chahil – *Indian Folk Dance*; Abhishek Kumar – *Bollywood Dance Mashup*.

Coffee Break

3:45 pm - 4:00 pm

Panel Session

4:00 pm - 5:30 pm

Session host: Anubha Seth

Moderator: Montrell Seay

Breaking the "only academia" myth: Postdoc careers that actually exist

Panelists: Tyler Goddard, PhD. – *Patent agent*

Samantha Rossano, PhD. – *Medical Science Liaison*

Morag Grassie, PhD. – *B.F. Executive Director Yale Venture*

Theresa Allio, PhD. – *Global regularly lead at Sanofi*

Awards and Closing Remarks

5:30 pm - 6:00 pm

Chairs: Nebal Abu Hussein & Eunate Gallardo

Reception: Networking with Panel

6:00 pm - 7:00 pm

YPA Social Event @ Blue Orchid

7:30 pm – onwards

Session I: Oral Presentations

8:45 am - 9:25 am.

Abstract Title:	Persistent inequities in pneumoconiosis mortality in Colombia's mining regions: a 25-year ecological analysis
Abstract Authors:	**Jeadran Malagón Rojas ;** Diana Diaz Jiménez; Karol Cotes; Salin Avellaneda; Diemen Delgado-García
Abstract: **TALK session 1 talk 1 8:45–8:55**	Transient intercellular bridges connecting daughter cells arise from contractile cleavage furrows during cytokinesis. Midbody organelles assemble in these bridges to mediate final membrane abscission and complete separation of new cells. However, in animal germ cells and certain somatic cells, midbodies instead mediate incomplete cytokinesis, in which abscission is blocked, and intercellular bridges are stabilized to enable ongoing transfer of molecules, organelles, and signals between sibling cells during synchronous development and gametogenesis. Our research program explores the mechanisms by which a transient cytokinetic structure develops into a stable intercellular bridge, or ring canal. Our lab used live imaging to discover a "new" highly conserved midbody remodeling step where midbody core proteins rapidly transform from a large spherical organelle into a stable ring canal with an open lumen. We are testing the hypothesis that conserved midbody proteins are regulated by germ cell-specific post-translational modifications and protein interactions to result in stable ring canal formation. To do this, we are studying the Drosophila melanogaster (fruit fly) midbody proteins Pavarotti (MKLP1) and Sticky (Citron Kinase), and Sticky's binding partner Rho1 GTPase using CRISPR gene editing, live cell imaging, genetic RNAi, and clonal analysis in germ cells. We are testing Pav phosphorylation status by generating non-phosphorylatable (4T-A) or phosphomimetic (4T-D) mutant transgenic flies. We are using a structure-function analysis to identify key domains of Sticky, while also testing its interaction with Rho1 GTPase by CRISPR editing of predicted binding sites. Our study will reveal regulatory mechanisms controlling the ancient and conserved process of incomplete cytokinesis used by germ cells during gametogenesis.

Abstract Title:	Assessment of War-Induced Structural Damage to Pharmaceutical Infrastructure in Sudan Using Remote Sensing and GIS Analysis: Implications for Essential Medicines Availability
Abstract Authors:	**Eman Salih**, Kaveh Khoshnood, Zena Ahmed, Charles Thomas Minicucci, Nadia Hamed, Jeremy Schwartz
Abstract: **TALK Session 1 talk 2 8:55–9:05**	Background When war reached Khartoum in April 2023, it did not only destroy buildings. It destroyed the supply chain on which 50 million people depend for essential medicines. The Rapid Support Forces looted NMSF warehouses before quarterly resupply reached most states. Local manufacturing, which supplied 30% of national medicine needs focused on Essential Medicines List (EML) drugs, collapsed to less than 10%. EML availability fell to 45.1%, far below the WHO 80% target, with insulin at only 11%. This destruction represents a long-term erosion of health system capacity, including production lines, cold-chain systems, and regulatory-certified facilities that cannot be rapidly reconstituted after conflict ends. Aim To determine the extent and progression of war-induced structural damage to Sudan's pharmaceutical infrastructure and its implications for EML medicine availability. Methods We constructed a georeferenced database of 55 facilities, including NMSF warehouses and manufacturing sites, validated against the National Medicines and Poisons Board registry. Sentinel-2 imagery from April 8, 2023 and January 6, 2026, processed in ArcGIS at Yale University, classifies structural damage at the building level and within 1 km buffer zones. Damage is graded on a five-class scale at six-month intervals across six snapshots, cross-validated against ACLED and the WHO Surveillance System for Attacks on Health Care. Results Imagery from April 8, 2023, seven days before the outbreak of war, establishes an immediately pre-conflict baseline. Comparative analysis with January 6, 2026 imagery at Tabuk Pharmaceuticals Co. Ltd and DAL Group Company Ltd documents structural change across three years of conflict. Full damage classification of all 55 sites, with linkage to their EML medicine profiles, is underway.

Abstract Title:	Pathway toward consensus: Lessons from the WHO Pandemic Agreement negotiations
Abstract Authors:	**Chloé Bâtie**, Anna Bezruki, Colin Carlson, Alexandra Phelan
Abstract: **TALK Session 1 talk 3 9:05 – 9:15**	

Abstract Title:	William Bullock: A Natural History Showman
Abstract Authors:	**Clare Tonks**
Abstract: **TALK Session 1 talk 4 9:15–9:25**	

Session II: Oral Presentations

11:00 am - 12:15 pm.

Abstract Title:	Herpesviruses and their role in Long COVID
Abstract Authors:	**Claire S. Laxton PhD*[1]**, Alexandra Tabachnikova MPhil*[1], Lily Cooke MS*[2], Kexin Wang*[3], Simone Blaser MD[1,4], Julio Silva PhD[1], Jamie Wood PT, PhD[2], Henna Nam[1], Zhenni Lu MPH[1], Christine Miller DO, PhD[5], Gisele Rodrigues MS[6], Victoria Fisher MS[6], Christian Guirgis[1], William B. Hooper[6], Alexandra Lee[1], Mackenzie Doerstling MPH[2], Bornali Bhattacharjee PhD[1,6], Leying Guan PhD† [3], David Putrino PT, PhD† [2], Akiko Iwasaki PhD† [1,6,7]
Abstract: **TALK Session 2 talk 1 11:00 – 11:10**	

Abstract Title:	Redefining the mechanism of action of tetracyclines
Abstract Authors:	**Swapnil Chandrakant Devarkar**, Ivan B. Lomakin, Jimin Wang, Ayman Grada, Christopher G. Bunick
Abstract: **TALK Session 2 talk 2 11:10-11:20**	Bacterial antimicrobial resistance (AMR) has led to over a million deaths per year globally since 2019 and is one of the most significant threats to public health worldwide. Tetracyclines are one of the most widely used class of antibiotics and have been a mainstay in the clinic over the past seven decades for treating a wide range of bacterial diseases including Lyme disease, anthrax, acne vulgaris, and pneumonia. To overcome widespread AMR, tetracycline derivatives like Doxycycline, Sarecycline, and Minocycline carrying additional modifications and substitutions to the naphthacene core have been developed over the past few decades. Current understanding of the mechanism of action of tetracyclines stems from structural studies on ribosomes from model bacteria like Escherichia coli and Thermus thermophilus. No structural information is available on how clinically prescribed tetracyclines like Doxycycline and Minocycline interact with the bacterial ribosome and whether any differences exist for their binding to ribosomes from pathogenic bacteria compared to model bacteria like Escherichia coli. Using a series of high-resolution cryo-electron microscopy (cryo-EM) structures of three clinically prescribed tetracyclines bound to ribosomes from the acne causing bacterium Cutibacterium acnes and the commensal model bacterium Escherichia coli, we show that tetracyclines can simultaneously target the mRNA decoding center in the 30S subunit and the nascent peptide exit tunnel (NPET) in the 50S subunit of the bacterial ribosome. Structure-guided mutagenesis of the 30S subunit canonical binding site revealed that tetracyclines can inhibit bacterial ribosomes via this previously uncharacterized secondary binding site in the NPET. Among the tested tetracyclines, Doxycycline was unique in its ability to dimerize and bind the NPET at multiple locations, potentially explaining its superior clinical performance. Structural comparison of Doxycycline, Minocycline, and Sarecycline bound to the Escherichia coli and Cutibacterium acnes 70S ribosome revealed species-specific differences affecting binding pose, interaction network, and occupancy of tetracyclines. These results reveal that tetracyclines target both activity centers of the bacterial ribosome and help redefine decades-old dogma on the mechanism of action of tetracyclines. The mechanistic and structural insights from this work present novel avenues for rational design of narrow spectrum tetracyclines for combating the rising threat of bacterial AMR.

Abstract Title:	KLF15 Acts as a Brake on Adipocyte Progenitor Expansion

Abstract Authors:	**Lin Li,** Brian Feldman
Abstract: **TALK Session 2 talk 3 11:20-11:30**	Background: Healthy adipose tissue expansion requires coordinated proliferation of adipocyte progenitor cells (APC), permitting hyperplastic growth that maintains metabolic homeostasis. Failure of this process promotes adipocyte hypertrophy, inflammation, and insulin resistance, key drivers of obesity and type 2 diabetes. Despite the clinical importance of adipose tissue remodeling, the transcriptional mechanisms governing progenitor proliferative control remain poorly defined. We hypothesized that the nutrient-responsive transcription factor Klf15 orchestrates a transcriptional program that restrains APC proliferation to ensure healthy adipose tissue development. Methods: An APC-specific Klf15 conditional knockout mouse model (Prx1-Cre; Klf15fl/fl) was used. APC proliferation was quantified in vivo and ex vivo using EdU incorporation and Ki67 flow cytometry in $CD31^{-}/CD45^{-}/SCA1^{+}$ cells. RNA sequencing of FACS-isolated APC was integrated with Klf15 ChIP-seq datasets to identify downstream regulatory targets. Results: Klf15 deficiency induced a cell-autonomous increase in APC proliferation, identifying Klf15 as a key intrinsic regulator of progenitor expansion. Transcriptomic analysis revealed significant downregulation of Robo4, an anti-proliferative membrane receptor, in Klf15-deficient APC. Robo4 suppression was sufficient to recapitulate the hyperproliferative phenotype, establishing it as a functional downstream effector. Mechanistically, Klf15 did not directly regulate Robo4 but instead directly activated Sox7. Restoration of Sox7 rescued Robo4 expression and normalized APC proliferation, demonstrating that Sox7 is required for Klf15-mediated proliferative restraint. These findings define a hierarchical transcriptional pathway for APC proliferation control. Conclusions: A previously unrecognized Klf15–Sox7–Robo4 transcriptional axis functions as a molecular brake on APC proliferation. This work establishes a mechanistic framework connecting nutrient sensing to adipose tissue developmental programming and identifies progenitor regulation as a potential therapeutic entry point to prevent unhealthy adipose expansion and metabolic disease.

Abstract Title:	Targeting metabolic–DNA repair vulnerabilities in SDHB- and FH-deficient renal cell carcinoma with the novel alkylating agent KL-50
Abstract Authors:	**Suparna Basu**, Ranjini Sundaram, Brian M. Shuch, Susan Gueble, Juan C. Vasquez
Abstract: **TALK Session 2 talk 4 11:30-11:40**	Metabolic enzyme deficiencies define a subset of aggressive renal cancers and may create exploitable therapeutic vulnerabilities. In renal cell carcinoma (RCC), loss of tricarboxylic acid (TCA) cycle enzymes succinate dehydrogenase B (SDHB) and fumarate hydratase (FH) results in accumulation of the oncometabolites succinate and fumarate. These metabolites disrupt epigenetic regulation and impair DNA damage response pathways, potentially sensitizing tumors to DNA-damaging agents, particularly alkylating therapies. We investigated the therapeutic vulnerability of SDHB- and FH-deficient RCC models to DNA alkylating agents by comparing KL-50, a novel imidazotetrazine compound, with the clinically used agent temozolomide (TMZ). TMZ exerts cytotoxicity through formation of O6-methylguanine DNA lesions, which are repaired by O6-methylguanine-DNA methyltransferase (MGMT). In MGMT-deficient cells, these lesions mispair during replication and activate mismatch repair (MMR), leading to replication stress and cell death. In contrast, KL-50 induces MGMT-dependent but MMR-independent cytotoxicity by forming O6-(2-fluoroethyl)guanine, which undergoes chemical rearrangement to generate DNA interstrand crosslinks. Using CRISPR-engineered SDHB- and FH-deficient Renca and LVRCC67 murine RCC models, we observed enhanced sensitivity to KL-50 compared to TMZ in vitro and in vivo. Both parental models exhibited baseline MGMT silencing, consistent with reports linking SDHB/FH loss to a hypermethylated phenotype and increased MGMT deficiency. Restoration of MGMT expression conferred resistance to both agents, establishing MGMT status as the primary determinant of treatment response. Collectively, these findings identify SDHB- and FH-deficient RCC as a molecular subset characterized by MGMT silencing, revealing a therapeutic vulnerability to KL-50, which may overcome resistance mechanisms that limit TMZ efficacy.

Abstract Title:	Insights into the nuclear import of 26S proteasomes using a nuclear pore complex (NPC) mimic system
Abstract Authors:	**Chetna Dhembla[1]*,** Qingzhou Feng[2]*, Chenxiang Lin[2], Mark Hochstrasser[1]
Abstract: **TALK Session 2 talk 5 11:40 – 11:50**	

Abstract Title:	Benchmarking generative scaffold design methods for peptide engineering in TCR–MHC complexes
Abstract Authors:	**Linhui Xie,** Gia-Bao Dam, Yashvi Patel, Lilian Denzler, Yanjun Shao, Ruimin Wang, Etienne Caron, Yoshiaki Yasumizu, David Hafler, Maria Rodriguez Martinez
Abstract: **TALK Session 2 talk 6 11:50-12:00**	T cell receptors (TCRs) recognizing of peptide-major histocompatibility complex (pMHC) serves as the gatekeeper of adaptive immunity, yet the de novo discovery of these antigenic peptide triggers remains a formidable computational challenge. This study provides the first systematic comparison of generative protein modeling methods applied to the spatially constrained MHC binding groove. We developed a new multi-stage computational pipeline and a comprehensive benchmark for generative peptide design within predefined TCR-pMHC contexts. The workflow comprises four sequential phases: (I) peptide scaffold generation and sequence sampling; (II) binding affinity filtering and structural threading; (III) dual-track physics-based energy optimization; (IV) structural validation via AlphaFold3. Benchmarking across 10 diverse human leukocyte antigen (HLA) class I templates, we reveal a fundamental trade-off between structural fidelity and binding propensity. RFdiffusion1 (RFd1) consistently outperforms RFdiffusion3 (RFd3) in structural accuracy, yielding significantly lower geometric deviation and higher structural confidence. Conversely, RFd3 designs exhibit a significantly higher predicted binder probability across all templates, suggesting it explores a broader, binding-competent conformational landscape beyond native geometries. We find that moderate constraint sets of 8 to 12 hotspots optimally balance geometric accuracy and conformational diversity. This standardized scoring protocol enables fair and reproducible comparison of generative methods by reducing method-specific biases and integrating sequence, structure, and physics-based metrics. Ultimately, this work not only establishes a foundation for benchmarking generative models in immunologically relevant interfaces but also provides actionable guidelines for the structure-based design of next-generation peptide therapeutics.

Abstract Title:	Discovering interpretable low-dimensional dynamics from high-dimensional scientific data
Abstract Authors:	**Michael Chung**, Purushottam Dixit
Abstract: **TALK Session 2 talk 7 12:00-12:10**	Across biology, neuroscience, and materials science, modern experiments routinely produce high-dimensional, time-resolved data whose sheer volume can obscure the mechanisms driving system behavior. Yet many such systems are believed to evolve on low-dimensional manifolds governed by a small number of underlying rules. Recovering those rules directly from data, in a form that is both physically interpretable and predictive, remains a central challenge. Existing approaches typically trade interpretability for reconstruction accuracy or recover symbolic equations whose latent variables cannot be tied to measurable quantities. We present Edwin, a unified framework that simultaneously compresses high-dimensional nonnegative time-series into low-dimensional latent coordinates and discovers sparse symbolic equations governing their evolution. Edwin couples the dynamic maximum entropy principle with a new information-theoretic model selection criterion, SLIC, that we derived from first principles to remain robust as dataset size grows, addressing a scaling pathology shared by classical criteria such as AIC and BIC. Crucially, Edwin jointly learns how each latent coordinate maps onto external metadata, giving every recovered variable direct physical meaning without post-hoc interpretation. We validate Edwin on stochastic processes, self-assembling particles, spiking neural populations, and low-rank recurrent networks, and apply it to fluorescence microscopy of aggregating RNA–liposome complexes used in cancer immunotherapy. In this experimental system, Edwin extracts a one-dimensional power-law model whose inferred parameters respond interpretably to controlled perturbations of formulation chemistry, recovering mechanistic insight without any prior kinetic assumptions. Together, these results establish Edwin as a general, principled route from raw high-dimensional measurements to interpretable scientific models.

Session III: Oral Presentations

2:15 pm - 3:15 pm.

Abstract Title:	Multi-locus genetic dosage shapes cognitive disease progression in Parkinson's: 15-year meta-analysis of 24 cohorts
Abstract Authors:	**Fatma Zehra Calikusu**, Xiaoying Kang, Zechuan Lin, Fatma Zehra Calikusu, Lauren A. Miller, Sofia Soriano, Yangyi Fan, Kara Mead, and Clemens R. Scherzer for the International Genetics of Parkinson Disease Progression (IGPP) Consortium
Abstract: **TALK Session 3 talk 1 2:15 – 2:25**	

Abstract Title:	Evaluating the Effect of Continued FIT Screening: A Target Trial Emulation Using the Clone–Censor–Weight Method
Abstract Authors:	**Buket Esen**, Lars Pedersen
Abstract: **TALK Session 3 talk 2 2:25-2:35**	Background: The effectiveness of the fecal immunochemical test (FIT) in reducing colorectal cancer (CRC) mortality is well-known. However, studies evaluating the effect of attending a second FIT screening following a negative initial result remain limited. Objective: To evaluate the effect of attending a second FIT screening on CRC-specific mortality and CRC diagnosis risk among individuals with an initial negative FIT. Methods: We conducted a cohort study of 622,265 individuals with an initial negative FIT. We used an emulated trial design with clone–censor–weight (CCW) methodology to compare two screening strategies: 1) attending a second FIT screening, versus 2) not attending a second FIT screening. Results: At 6-year follow-up, the risk of CRC-specific mortality was 0.07% (95% CI: 0.06%; 0.08%) for those attending a second FIT screening versus 0.17% (95% CI: 0.14%; 0.21%) for those not attending. This resulted in a relative risk (RR) of 0.39 (95% CI: 0.30; 0.52) and a risk difference (RD) of -0.10% (95% CI: -0.14%; -0.07%). For CRC diagnosis, the 6-year risk was 0.63% (95% CI: 0.61%; 0.66%) in the attendance group versus 0.66% (95% CI: 0.59%; 0.74%) in the non-attendance group, yielding an RR of 0.96 (95% CI: 0.85; 1.08) and an RD of -0.03% (95% CI: -0.11%; 0.05%). Conclusion: Participation in a second FIT screening following an initial negative result reduces the relative risk of CRC-specific mortality. However, even though the RR is large, the RD is small. Furthermore, despite the use of the CCW, residual bias cannot be ruled out.

Abstract Title:	Cryptobiosis and Aging: A Multilevel Framework for Studying Stress Resilience in Tardigrades
Abstract Authors:	**Amit Kumar Nagwani[1,2],** Łukasz Kaczmarek[1] & Hanna Kmita[1] [1]Adam Mickiewicz University, Poznań, Poland, [2]Yale University, New Haven, USA (Current address)
Abstract: **TALK Session 3 talk 3 2:35 – 2:45**	Aging is generally considered an inevitable and progressive biological process. Yet tardigrades ("water bears"); microscopic animals renowned for surviving extreme environmental conditions; challenge conventional understanding of life under stress through cryptobiosis, a reversible state in which metabolism becomes nearly undetectable. This remarkable phenomenon has inspired hypotheses such as the "Sleeping Beauty" hypothesis, proposing that biological aging may be stopped during cryptobiotic states. However, testing such concepts requires reliable multilevel markers of aging, which remain largely unexplored in tardigrades. To address this gap, this study established an integrative experimental framework using the dioecious tardigrade *Paramacrobiotus experimentalis*, a species characterized by exceptional anhydrobiotic capability and long lifespan. Females and males representing different age classes were examined under repeated desiccation and hypomagnetic conditions, linking organismal performance with cellular physiology under extreme environmental stress. The study combined analyses of survival, vitality, reproductive performance, mitochondrial inner membrane potential (Δψ), and intracellular reactive oxygen species (ROS) to identify candidate markers associated with tardigrade aging. The obtained results revealed distinct organismal and cellular patterns associated with age and sex, demonstrating that resilience to extreme conditions changes dynamically across lifespan in both females and males. Together, these findings establish one of the first multilevel aging frameworks in a dioecious tardigrade model and position tardigrades as promising systems for exploring the biology of aging, with potential applications in stress biology, astrobiology, and research into extraterrestrial life.

Abstract Title:	Phenotype risk scores identify distinct clinical features and genetic correlates of eating disorders
Abstract Authors:	**Alexandra Scalici**, Jiayi Xu, Keira J. A. Johnston, James T. Baker, Tyne Miller Fleming, Nancy J. Cox, Laura M. Huckins
Abstract: **TALK Session 3 talk 4 2:45 – 2:55**	

Abstract Title:	ATHENA: A deep learning–based AI for functional prediction of genomic mutations and synergistic vulnerabilities in prostate cancer
Abstract Authors:	**Siyuan Cheng**, Xiao Jin,Jiaying Qian, Zirui Fu, Yuyin Jiang, Isaac Yi Kim, Ping Mu
Abstract: **TALK Session 3 talk 5 2:55 – 3:05**	

Abstract Title:	Using Foundation Models as Pseudo-label Generators for Pre-clinical 4D Cardiac CT Segmentation
Abstract Authors:	**Anne-Marie Rickmann**, Stephanie L Thorn, Shawn S Ahn, Supum Lee, Selen Uman, Taras Lysyy, Rachel Burns, Nicole Guerrera, Francis G Spinale, Jason A Burdick, Albert J Sinusas, James S Duncan
Abstract: **TALK Session 3 talk 6 3:05-3:15**	Cardiac image segmentation is an important step in many cardiac image analysis and modeling tasks such as motion tracking or simulations of cardiac mechanics. While deep learning has greatly advanced segmentation in clinical settings, there is limited work on preclinical imaging, notably in porcine models, which are often used due to their anatomical and physiological similarity to humans. However, differences between species create a domain shift that complicates direct model transfer from human to pig data. Recently, foundation models trained on large human datasets have shown promise for robust medical image segmentation; yet their applicability to porcine data remains largely unexplored. In this work, we investigate whether foundation models can generate sufficiently accurate pseudolabels for pig cardiac CT and propose a simple self-training approach to iteratively refine these labels. Our method requires no manually annotated pig data, relying instead on iterative updates to improve segmentation quality. We demonstrate that this self-training process not only enhances segmentation accuracy but also smooths out temporal inconsistencies across consecutive frames. Although our results are encouraging, there remains room for improvement, for example by incorporating more sophisticated self-training strategies and by exploring additional foundation models and other cardiac imaging technologies.

Poster Session

12:15 pm - 2:15 pm.

Abstract Title:	Design-Based Weighted Regression Estimators for Average and Conditional Spillover Effects
Abstract Authors:	**Fei Fang**, Laura Forastiere
Abstract: **POSTER 1**	When individuals engage in social or physical interactions, a unit's outcome may depend on the treatments received by others. In such interference environments, we provide a unified framework characterizing a broad class of spillover estimands as weighted averages of unit-to-unit spillover effects, with estimand-specific weights. We then develop design-based weighted least squares (WLS) estimators for both average and conditional spillover effects. We introduce three nonparametric estimators under the dyadic, sender, and receiver perspectives, which distribute the estimand weights differently across the outcome vector, design matrix, and weight matrix. For the average-type estimands, we show that all three estimators are equivalent to the Hajek estimator. For conditional spillover effects, we establish conditions under which the estimands are consistent for the target conditional spillover effects. We further derive concentration inequalities, a central limit theorem, and conservative variance estimators in an asymptotic regime where both the number of clusters and cluster sizes grow.

Abstract Title:	Characterizing cell type-specific effects on transcription factor binding and function in C. elegans
Abstract Authors:	**Emma Murari**, Michelle Kudron, Valerie Reinke
Abstract: **POSTER 2**	Transcription factors (TFs) are a highly conserved class of regulatory proteins that bind specific genomic sequences and induce or repress the transcription of nearby genes. Many TFs have essential roles in regulating developmental signaling and maintaining cellular homeostasis–notably, about a third of human developmental disorders are linked to mutations in TFs. The mechanisms that govern TF function are extraordinarily complex and multi-factorial; many recent studies in human cell lines and in yeast have demonstrated that individual TF binding events rarely lead to changes in gene expression. Additionally, since most current research on TF binding and function is not tissue-specific, our understanding of how diverse regulatory environments across different cell types affect TF binding and function is incomplete. Thus, it is currently impossible to predict whether an individual TF binding event in a specific cellular context will yield a transcriptional response for any given target gene. To address this gap, we will use a novel genetic strategy in the model nematode C. elegans to link TF-dependent transcriptional changes to TF binding across multiple cellular contexts in vivo. We will analyze the resulting datasets alongside previously published data on TF binding, chromatin accessibility, and gene expression to reveal the regulatory mechanisms that determine the functional outcomes of any given TF binding event. We will also use this data to train predictive models to identify key genomic features predictive of TF function, which will improve our understanding of how diverse cell type-specific regulatory contexts influence the relationship between TF binding and changes in gene expression.

Abstract Title:	Toward Structured Mentorship: A Needs Assessment of Yale School of Medicine Postdoctoral Scholars
Abstract Authors:	**Fatma Zehra Calikusu**, Filippidis Paraskevas, Victoria Hallinan, Laura Meissner, Lauren A. Miller, Sofia Soriano
Abstract: **POSTER 3**	Background Mentorship is a sustained developmental relationship that supports personal, professional, and academic growth. In academic medicine, it extends beyond research supervision to include career development and psychosocial support. Postdoctoral scholars are early-career researchers navigating complex environments and high expectations while transitioning to independence. Despite its importance, institutional evaluation of mentorship needs among postdoctoral scholars remains limited. Objective To assess mentorship needs and experiences among Yale School of Medicine postdoctoral scholars and identify opportunities to strengthen institutional practices. Methods We conducted an anonymous cross-sectional survey distributed via the Yale Postdoctoral Office newsletter and postdoctoral networks. The survey included 5-point Likert-scale and open-ended questions on mentorship experiences, expectations, and institutional support. Quantitative data were analyzed using descriptive statistics, and qualitative responses underwent thematic analysis. Agreement was defined as Likert scores ≥4. Results Forty-four postdoctoral scholars responded, including 29 from Yale School of Medicine. Respondents reported a strong need for mentorship beyond research (median 5; 89.7% agreement), institutional guidance on academic norms (100%), and clearer mentorship expectations (89.7%). Mentors were valued for networking (89.7%), and most preferred one-to-one mentoring (75.9%). However, access to additional mentors was limited (17.2%), and fewer respondents reported clear expectation alignment (34.5%) or comfort addressing

	concerns (27.6%). Most indicated a need for stronger institutional mentorship structures (79.3%). Key themes included faculty mentorship training, multiple mentors, formal expectation setting, and structured programs. Discussion Postdoctoral scholars value mentorship but report gaps in structure, access, and clarity. Findings highlight the need for formalized mentorship frameworks, faculty training, and institutional support to enhance postdoctoral development.

Abstract Title:	Leveraging primate mitochondrial DNA variation to inform human disease variant interpretation
Abstract Authors:	**Li Tang**
Abstract: **POSTER 4**	Introduction: The human mitochondrial genome encodes genes essential for cellular energy production. mtDNA variation can cause mitochondrial disease and has been linked to common human diseases, but distinguishing harmful from harmless variants remains difficult because human data and functional evidence are limited. Closely related primates can provide complementary evolutionary information, as variants observed across primates are more likely to reflect biologically tolerated changes. However, a comprehensive primate mtDNA variation catalog for human variant interpretation has not yet been established. Methods: We analyzed whole-genome sequencing data from 789 non-human primate samples across 233 species using a bioinformatic pipeline designed for mtDNA and cross-species sequence divergence. Primate mtDNA variants were matched to corresponding human positions, followed by sample- and variant-level quality control. We compared primate mtDNA variation with gnomAD and assessed disease relevance using ClinVar. Results: We identified 57,344 mtDNA variants, covering nearly 40% of all mtDNA bases, with a mean of 209 variants per sample. The genomic distribution of primate mtDNA variation was broadly similar to that in gnomAD. Notably, 30.8% of primate variants were absent from gnomAD, highlighting non-human primates as a complementary source of naturally occurring mtDNA variation beyond current human population data. Overlap with ClinVar showed that shared variants were predominantly benign or likely benign (98.8%). Conclusion and General Impact: This study establishes a comprehensive primate mtDNA variation catalog as a complementary evolutionary resource for human variant interpretation.

Abstract Title:	Status of the CHIME experiment and its cosmological prospects
Abstract Authors:	**Kana Sakaguri**
Abstract: **POSTER 5**	

Abstract Title:	Modified RNA Extraction Methods to Eliminate Agarose Carryover Interferences in Human Precision-Cut Lung Slices
Abstract Authors:	Reina Rangel[1], Sabina Anderson[1], Giuseppe Deluliis[1], Edward P. Manning[3,1], Farida Ahangari[1], Abhay Pandit[2], Naftali Kaminski[1] and **Joan Marti-Munoz[1,2]**. 1. Section of Pulmonary Critical Care & Sleep Medicine, Yale School of Medicine, New Haven, CT, USA 2. CÚRAM Research Ireland Centre for Medical Devices, University of Galway, Ireland 3. VA Connecticut Healthcare System, West Haven, CT, USA

Abstract: **POSTER 6**	**Introduction:** Precision-cut lung slices (PCLS) have emerged as a novel tool to study biology in living human lungs. PCLS involves agarose gel inflation to facilitate vibratome sectioning and maintenance of pulmonary microarchitecture. However, agarose carryovers during RNA extraction reduce RNA integrity and yield.[1] Here we assess two methods whether dissolving agarose before RNA extraction or using a different RNA kit to clean out agarose carryovers. **Methods:** This study received approval from the Yale University Institutional Review Board. The National Disease Research Interchange (NDRI) provided a human lung from a deceased 74-years-old white male donor. PCLS were generated as previously described. Briefly, human lungs were inflated with a 2% agarose solution. Inflated lungs were cored using 12 mm punch and sectioned in a Precisionary vibratome. 400 µm-thick sections were incubated in DMEM:F12K with 0.1% BSA and 1% Antibiotic-Antimycotic with or without fibrotic cocktail (FC). PCLS were harvested after 6 days. We used two RNA extraction methods (Qiagen miRNeasy or Plant Kit) and PCLS with agarose (Agarose+) or PCLS which agarose was dissolved using ZymoResearch RAD buffer (Agarose-) (**Fig.1a**). We monitored PCLS viability by Calcein (live) and Ethidium homodimer-1 (dead) staining, imaged in a LeicaStellaris Confocal microscope and quantified fluorescence in ImageJ. We measured RNA quantity, quality and integrity in NanoDrop2000, Qubit2.0 and 2100Bioanalyzer. We measured gene expression by qPCR in a QuantStudio6Pro. Graphs and statistics were performed in GraphPad 9.5.0. **Results:** Live-dead staining confirmed cells were viable during culture conditions showing no changes to day one and a 6-fold increased life signal compared to dead control (**Fig.1b**). The RNA extraction for conventional agarose-containing PCLS showed inconsistent measurements among quantification techniques, high presence of impurities (260/280=1.56±0.05; 260/230=0.12±0.02) and absence of intact rRNA (**Fig. 1c,d,e,f**). Agarose dissolution led to consistent RNA quantifications, reduced impurities (260/280=1.88±0.06; 260/230=1.33±0.45) and intact rRNA (RIN=9.07±0.47). Plant kit still showed inconsistent RNA quantifications but showed reduction of impurities (260/280=1.75±0.06; 260/230=1.56±0.16) and partially intact rRNA (RIN=5.95±0.93). Agarose dissolution and Plant kit significantly increased the mRNA transcription of GUSB and COL1A1 by qPCR evidencing vast improvement in RNA integrity and showing a profound effect of the FC (**Fig.1g**). **Conclusions:** We showed that the presence of agarose in PCLS leads to high impurities that compromises RNA integrity during extraction. Dissolution of agarose efficiently leads to robust readouts while Plant Kit shows improvement but still inconsistencies in quantification and integrity.

Abstract Title:	Microplastics in the Human Distal Lung: Detection, Quantification and Characterization
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Abstract: **POSTER 7**	Introduction: We are surrounded by household plastics that slowly break down into smaller stable fragments.[1] The effects of airborne small (1-100 μm) microplastics (sMPs) inhalation in human health are concerning due to their environmental accumulation. We investigated the presence and impact of sMPs in human lung parenchyma. Methods: We received human frozen distal lung tissues from 56 different subjects; one cohort from the Lung Tissue Research Consortium, NHLBI (US) comprising five healthy donors and five Idiopathic Pulmonary Fibrosis (IPF) (10 subjects), one cohort from the KU Leuven Lung Biobank (Belgium 1) comprising ten donors, ten IPF and ten Chronic Obstructive Pulmonary Disease (COPD) (30 subjects) and a second cohort from KU Leuven Lung Biobank (Belgium 2) from eight young and eight old healthy donors (16 subjects). Tissues were digested in KOH solution and filtered or embedded in optimal tissue compound and sectioned in a cryostat (Fig 1a). Filters and tissue sections were imaged in an epifluorescence microscope at 390/420 nm long pass filter. We manually counted sMPs (particles and fibers) in filters and normalized by wet lung sample weight. A selection of sMPs in filters were analyzed by Quantum Cascade Laser Infrared Spectroscopy (QCL-IR) and statistically matched to standards. We imaged regions of interest showing presence of sMPs in tissue sections. Graphs and statistics were performed in GraphPad 9.5.0. Results: Filtration of digested lungs showed abundant and variable amounts of sMPs with most samples containing between 5,000 and 20,000 sMPs per gram of wet lung tissue (Fig 1b,c,g). QCL-IR confirmed heterogenous sMPs composition in lungs with Nylon, crumb rubber from used tires and Polyvinyl chloride being most abundant in the samples analyzed. Histology suggested sMPs alignment in the mucosal layer of some airways (Fig 1d,e) although staining confirmation is ongoing. The second Belgium cohort showed significantly higher sMPs amount than the rest (Fig 1g). We did not find a significant difference in sMPs among diseases (Fig 1h) but we observed a decreasing trend with age, that did not reach significance (Fig 1i). We are performing bulk RNA sequencing between samples containing high and low abundance of sMPs (Fig 1j) to assess molecular changes in tissues, followed by spatial transcriptomics to localized different biological responses.

Abstract Title:	Detecting heterogeneous multi-membrane protein assemblies using nativeCDMS: Identifying interactions in rare neurological disorders
Abstract Authors:	**Samantha Shepherd**, Dipayan Bose, Chiara Mancinelli, Shyam Krishnakumar, Kallol Gupta
Abstract: **POSTER 8**	In cellular membranes, ultrastructural organization of integral and membrane-associated proteins (MP) and lipids form highly dynamic multiprotein-lipid assemblies that drive membrane-associated signaling in a broad range of assembly states at organellar membranes. Detecting and identifying these dynamic, heteromeric, and multiscale assemblies necessitates bioanalytical techniques that provide both dynamic detection range and molecular resolution. Further, the membranes often play a key role in their biogenesis. Addressing this, we present a Charge Detection Mass Spectrometry (CDMS)-based MS platform that can capture and identify dynamic and heterogenous assembly states of multi-MP complexes, which we apply to study molecular interactions of protein variants in rare neurological disorders. Native CDMS enables precise mass identification of heterogenous multimeric complexes. We demonstrate that this technique enables detection of dynamic, membrane-associated multiprotein signaling complexes from heterogeneous mixtures, revealing low-abundance protein-protein complexes that evade ensemble measurements. We next demonstrate our ability to combine this analysis with native top-down and complex up experiments to identify complex subunits and sequence. This combination of techniques enables unambiguous identification of the molecular architecture and composition of the detected multi-proteo-lipid complexes. We next apply this platform to trans-bilayer multimeric neuronal SNARE-protein assemblies that drive fusion of synaptic vesicles (SV) a process that is fundamental to all signal processing in the brain. Application of our nCDMS platform on SNARE and associated proteins, reveals key multiprotein signaling complexes. Subsequently, we investigated the complex formation of SNARE variants causing rare neurological disorders, showing the ability of nCDMS to reveal key changes in the molecular landscape of signaling complex formation leading to impaired neuronal signaling.

Abstract Title:	Structural Basis of TACO1-Mediated Efficient Mitochondrial Translation
Abstract Authors:	**Shuhui Wang*,** Michele Brischigliaro*, Yuekang Zhang, Chunxiang Wu, Wei Zheng, Antoni Barrientos[#], Yong Xiong[#]

Abstract: **POSTER 9**	Translation elongation is a universally conserved step in protein synthesis, relying on elongation factors that engage the ribosomal L7/L12 stalk to mediate aminoacyl-tRNA delivery, accommodation, and ribosomal translocation. Using in organello cryo-electron microscopy, we reveal how the mitochondrial translation accelerator TACO1 promotes efficient elongation on human mitoribosomes. TACO1 binds the mitoribosomal region typically bound by elongation factor Tu (mtEF-Tu), bridging the large and small subunits via contacts with 16S rRNA, bL12m, A-site tRNA, and uS12m. While active throughout elongation, TACO1 is especially critical when translating polyproline motifs. Its absence prolongs mtEF-Tu residence in A/T states, causes persistent mitoribosomal stalling and premature subunit dissociation. Structural analyses indicate that TACO1 competes with mtEF-Tu for mitoribosome binding, stabilizes A-site tRNA, and enhances peptidyl transfer through a mechanism distinct from EF-P and eIF5A. These findings suggest that bacterial TACO1 orthologs may serve analogous roles, highlighting an evolutionarily conserved strategy for maintaining elongation efficiency during challenging translation events.

Abstract Title:	Erratic Maternal Care Induces Avoidant-Like Attachment Deficits in a Mouse Model of Early Life Adversity
Abstract Authors:	**Zoë MacDowell Kaswan**, Christian Bowers, Sahabuddin Ahmed, Arie Kaffman
Abstract: **POSTER 10**	Attachment theory offers an important clinical framework for understanding and treating negative effects of early life adversity. Attachment styles emerge during critical periods of development in response to caregivers' ability to consistently meet their offspring's needs. Attachment styles are classified as secure or insecure (anxious, avoidant, or disorganized), with rates of insecure attachment rising in high-risk populations and correlating with a plethora of negative health outcomes throughout life. Despite its importance, little is known about the neural basis of attachment. Work in rats has demonstrated that limited bedding and nesting (LB) impairs maternal care and produces abnormal maternal attachment linked to increased pup corticosterone. However, the effects of LB on attachment-like behavior have not been investigated in mice where additional genetic and molecular tools are available. Furthermore, no group has utilized home-cage monitoring to link abnormal maternal care with deficits in attachment-like behavior. Using home-cage monitoring, we confirmed a robust increase in maternal fragmentation among LB dams. Abnormal maternal care was correlated with elevated corticosterone levels on post-natal day seven (P7) and a stunted growth trajectory that persisted later in life. LB did not alter maternal buffering at P8 or maternal preference at P18, indicating that certain attachment-like behaviors remain unaffected despite exposure to high levels of erratic maternal care. However, LB pups vocalized less in response to maternal separation at P8, did not readily approach their dam at P13, and exhibited higher anxiety-like behavior at P18, suggesting that LB induces avoidant-like attachment deficits in mice.

Abstract Title:	Preliminary results on the plastic properties of potassium chloride under high-pT conditions.
Abstract Authors:	**Yuichiro Mori**
Abstract: **POSTER 11**	

Abstract Title:	Latent profile analysis of actigraphy-derived sleep regularity, timing, and duration in adults with type 2 diabetes: clinical and psychosocial correlates
Abstract Authors:	**Eunbee Kim**, Soohyun Nam, PhD
Abstract: **POSTER 12**	

Abstract Title:	Framework for Property-Informed Design of Safer Chemicals: Case Study with UV Radiation Protection Functionality
Abstract Authors:	**Rachel Broughton**, Predrag V. Petrovic, Tobias D. Muellers, Paul T. Anastas, Julie B. Zimmerman

Abstract: **POSTER 13**	Effective innovation in designing safer chemicals must come from the realization that predicting a chemical's hazard is insufficient; rather, a comprehensive design approach should consider not only the safety of a chemical, but also the intended functionality. Physicochemical properties influence chemical behaviors and understanding the underlying mechanisms leads to safe and functional chemical design. Much of the information needed to elucidate the relationships between properties, function, and hazard exists in databases or is obtainable through quantum chemical calculations. However, integration of such information that leads to clear design rules translatable across multiple chemical classes is difficult due to the diversity of chemical space, lack of unified functionality definitions, data uncertainty, and other considerations. In this work, the steps necessary for designing safer chemicals considering both efficacy and toxicity are examined via a case study of substances that serve the function of preventing the harmful effects of UV radiation. Challenges and decisions that arise throughout the process are addressed and existing databases, information, and design criteria that are useful for informing chemical toxicity and functionality are highlighted. Approximately 250 substances that are used as UV radiation protectants were identified from databases and literature sources. Boundaries of this specified functional chemical space were defined based on commonalities in physicochemical properties and then extended to safe chemical space by applying property-based design rules for reduced toxicity, specifically related to bioavailability. Integration of information about both function and safety within a defined chemical space led to the identification of low to no hazard chemical candidates capable of performing the desired function.

Abstract Title:	Healing in caring and knowledge-making through everyday food practices among LGBTQ+ BIPOC
Abstract Authors:	**Elizabeth Wong**

Abstract: **POSTER 14**	The work of therapy, from a liberation psychology view, is for recovering historical memory, de-ideologizing everyday experiences, cultivating individual and community strengths and virtues, and moving towards psychological freedom (Martín Baró, 1994). In our clinical work, more efforts can be placed in understanding how wellbeing is understood, cultural connections and strengths, and building activities of caring. Given that food is an essential component of survival, we can take more care to help clients think through food beyond the lens of physical nutrition. Three different models are used in this study to understand how everyday food practices carry healing potential through the interwoven process of caring and knowledge-making. Daily food practices reflect a mixture of tradition and modernity— borne out of our roots and our contexts. Anzaldúa's (1987) Borderland Theory helps us understand how this mixture is made by people who straddle two borders or worlds and contest those confines. Chavez-Dueñas et al.'s (2019) HEART model names strengthening our cultural and familial traditions and roots as part of the broader processes of building cultural consciousness and connection and engaging in collective healing. Finally, Galliher et al.'s (2017) Integrated Identity Development maps our identities and deep interconnectedness with one another, observed in daily food behaviours like cooking, eating, and sharing. Methods (research methods or innovation approach) This study uses food photos and interviews of 14 Black, Indigenous, People of Color (BIPOC) individuals, who are Lesbian, Gay, Bisexual, Transgender, Queer (LGBTQ+) from United States and Canada, to explore how they heal, learn, and make meaning of their food practices. Participants photographed food moments across two weeks, brought these photos during a semi-structured interview at four weeks, and responded to a follow-up after eight months. Participants' daily food moments reflected their personal, social, and cultural contexts. Individually, participants shared definitions of healing, and intentionality and gratitude as important wellness attitudes. They experienced caring and connection through social aspects of sharing and receiving food, particularly individualizing food to express care and connecting to a sense of togetherness. Participants connected to food on a cultural level by engaging with values such as resourcefulness and marking special occasions. Participants experienced food's historical memory by connecting with ancestry, land, and the sacred, acknowledging oppression, and creating memories. Lastly, food is a landscape of contradictory notions of authenticity and hybridity, and an arena to develop new family traditions, given caregivers' roles as knowledge keepers. Seven participants endorsed increased mindfulness around food practices, and seven participants reported increased awareness and connection to their background, loved ones, and communities at the eight-month follow-up. We offered this study as an intervention in making time and space to intentionally engage in culturally-significant and embodied practices. Everyday practices can be imbued with new meaning, intention, and alignment to love and care. This can amplify the cultivation of wellness through processes of eating, preparing, or sharing. Centering people's storytelling, archiving, and documenting efforts (including journaling) can be helpful in cultivating historical memory among clients. Our well-being is holistic and grounded in care, and our care for one another is tied to our interconnected struggles. Connecting to local and grassroots programming, such as supporting indigenous food sovereignty initiatives, community fridge and kitchen programming for continued knowledge keeping, public health policies around holistic food wellbeing, funding for cultural gatherings and celebrations, and gleaning teams for food insecurity can be some avenues to support the strengthening of our cultural connections and work toward dismantling oppressive systems.

Abstract Title:	Differential regulation of cytokinetic proteins during midbody remodeling to form ring canals in Drosophila germ cells.
Abstract Authors:	**Akanksha Pandey**, Kari Price, Kathleen Ayers, Ellery Winkler and Lynn Cooley

Abstract: **POSTER 15**	Transient intercellular bridges connecting daughter cells arise from contractile cleavage furrows during cytokinesis. Midbody organelles assemble in these bridges to mediate final membrane abscission and complete separation of new cells. However, in animal germ cells and certain somatic cells, midbodies instead mediate incomplete cytokinesis, in which abscission is blocked, and intercellular bridges are stabilized to enable ongoing transfer of molecules, organelles, and signals between sibling cells during synchronous development and gametogenesis. Our research program explores the mechanisms by which a transient cytokinetic structure develops into a stable intercellular bridge, or ring canal. Our lab used live imaging to discover a “new” highly conserved midbody remodeling step where midbody core proteins rapidly transform from a large spherical organelle into a stable ring canal with an open lumen. We are testing the hypothesis that conserved midbody proteins are regulated by germ cell-specific post-translational modifications and protein interactions to result in stable ring canal formation. To do this, we are studying the Drosophila melanogaster (fruit fly) midbody proteins Pavarotti (MKLP1) and Sticky (Citron Kinase), and Sticky’s binding partner Rho1 GTPase using CRISPR gene editing, live cell imaging, genetic RNAi, and clonal analysis in germ cells. We are testing Pav phosphorylation status by generating non-phosphorylatable (4T-A) or phosphomimetic (4T-D) mutant transgenic flies. We are using a structure-function analysis to identify key domains of Sticky, while also testing its interaction with Rho1 GTPase by CRISPR editing of predicted binding sites. Our study will reveal regulatory mechanisms controlling the ancient and conserved process of incomplete cytokinesis used by germ cells during gametogenesis.

Abstract Title:	The Hidden Diversity of Antibody Heavy Chains: Potential Impact on Autoantibody Pathogenic Efficiency in Myasthenia Gravis
Abstract Authors:	**EASTON E. FORD, PhD[1]**; Veenasravani Pendyala[1]; Minh C. Pham, PhD[2,4]; Uddalok Jana[1]; Fatemeh Khani-Habibabadi, PhD[2,4]; Soumya S. Yandamuri, PhD[2,4]; Oscar L. Rodriguez, PhD[3]; Zach VanWinkle[1]; Nadia Richardson[1]; William D. Lees[5]; Elizabeth A. Hudson[1]; Corey T. Watson, PhD[1]; Kevin C. O'Connor, PhD[2,4]; Melissa L. Smith, PhD[1]; [1]Dept. of Biochemistry and Molecular Genetics, Univ. of Louisville Sch. of Med.; [2]Dept. of Immunobiology, Yale Sch. of Med.; [3]Dept. of Microbiology, Icahn Sch. of Med. at Mount Sinai; [4]Dept. of Neurology, Yale Sch. of Med.; [5]Institute of Structural and Molecular Biology, Birkbeck College, Univ. of London;
Abstract: **POSTER 16**	Current Adaptive Immune Receptor Repertoire sequencing (AIRR-seq) methods primarily resolve antibody (IG) variable regions while providing limited resolution of constant region (IGHC) diversity. Here, we applied complementary immunogenomic approaches to characterize IG heavy chain genetics and repertoires in individuals with acetylcholine receptor antibody-positive myasthenia gravis (AChR-MG). Genomic DNA and RNA were co-extracted from peripheral blood mononuclear cells from AChR-MG patients and healthy donors. Samples were processed for IG heavy chain (IGH) genotyping from genomic DNA and near full-length adaptive immune receptor repertoire sequencing (FLAIRR-seq). Preliminary profiling of IGHC regions identified extensive allelic diversity. In 48 healthy donors analyzed using FLAIRR-seq alone, we identified 70 IGHC alleles, including 26 previously curated in the IMGT database and 44 novel alleles (63%). Among AChR-MG individuals, 47 of 48 samples had both FLAIRR-seq and IGcapture data available, enabling internal validation of allele calls using an independent targeted genomic sequencing approach. Across these individuals, 89 total IGHC alleles were identified, including 19 curated IMGT alleles and 70 novel alleles (79%). FLAIRR-seq repertoire analysis revealed increased B cell clonal expansion in AChR-MG individuals at both isotype and subisotype levels based on Simpson’s diversity metrics ($p<0.05$), consistent with immune dysregulation in the antibody repertoire. We also observed biased IGHV gene usage, including elevated IGHG3 usage ($p<0.05$) in AChR-MG donors. Within IGHG3 repertoires, specific V genes (IGHV1-69, IGHV3-9, IGHV3-53) were frequently utilized across multiple individuals. IgG3 antibodies are known for complement activation efficiency compared to other IgG subclasses, a mechanism particularly relevant in MG where complement-mediated damage is a major contributor to disease pathology. Hinge length variants in IGHG3 were identified at both DNA and RNA levels, suggesting structural differences that may influence complement activation. We also identified amino acid substitutions in IGHG1 and IGHG3 alleles that may influence multiple antibody effector functions.

Abstract Title:	Hidden helpers in salty soils: how fungi protect plants from salt damage
Abstract Authors:	**Monika Kubalova**, Alex Canto-Pastor

Abstract: **POSTER 17**	Soil salinization is a serious threat to agriculture worldwide, limiting crop growth and yield. When exposed to high salinity, plants face two distinct physiological challenges: ionic toxicity caused by Na^+ accumulation in cells, and osmotic stress caused by reduced water availability. While plants have evolved sophisticated mechanisms to cope with both, beneficial soil fungi of the genus Trichoderma can dramatically improve plant survival under salt stress conditions. Yet, despite the significant potential of Trichoderma as a biotechnological tool for improving crop salt tolerance, the molecular mechanisms by which it coordinates stress responses remain largely unknown. To address this knowledge gap, we profiled the genome-wide transcriptional response of the model plant Arabidopsis thaliana to salt stress in the presence and absence of Trichoderma colonization. We show that Trichoderma enhances the plant's ability to expel excess sodium while simultaneously dampening the phytohormonal stress alarm involved in osmotic stress adaptation. These two complementary strategies together allow Trichoderma-colonized plants to maintain growth under conditions that would severely damage untreated plants. These findings provide insight into how beneficial soil microbes can be leveraged to enhance plant performance under salt stress conditions.

Abstract Title:	Genomically recoded organisms expedite single-codon labelling for single-molecule tracking
Abstract Authors:	**Filip Ilievski**
Abstract: **POSTER 18**	Genomically recoded Escherichia coli (GRE) represents a valuable tool as model organisms for single-molecule tracking via click-labelling. These strains are constantly being engineered to incorporate non-canonical amino acids enabling expanded chemical functionality. However, extensive genome-scale recoding frequently reduces their fitness and imposes metabolic re-wiring limiting the utility of GREs as practical model organisms for live-cell imaging in vivo. This study reports that previously optimized GRE6 was subjected to adaptive laboratory evolution (ALE) in the imaging rich-defined media (RDM) to recover fitness. The evolved strain displayed improved growth relative to the ancestral GRE6 and a wild-type-like growth confirmed by single-cell analysis relative to the commonly used MG1655. Whole-genome sequencing revealed deletion of the initiation inhibitor ratA. ncAA incorporation was significantly improved relative to the ancestral MG1655 strain. These results point out the importance of applying ALE in parallel to rational genome engineering in expanding the benefits of genomically re-coded organisms, applicable for both fundamental studies as well as improved protein production and biocatalysis.

Abstract Title:	Selection-based modeling reveals CNA drivers beyond frequency and identifies actionable targets in ovarian cancer
Abstract Authors:	**Meng Liu**, Jeffrey D. Mandell, Kira A. Glasmacher, Vincent L. Cannataro, and Jeffrey P. Townsend
Abstract: **POSTER 19**	Ovarian cancer (OV) remains one of the most lethal malignancies. Target discovery is challenging because OV features few recurrent oncogenic single-nucleotide mutations and pervasive chromosomal instability. Current strategies prioritize amplified genes based on frequency and amplitude of copy number amplification. However, these metrics are confounded by regional genomic architecture and passenger events and do not identify amplifications that truly increase cell proliferation and survival during tumor evolution. To address this limitation, we developed a ploidy-aware evolutionary model that quantifies the cancer effect size (CES) of amplifications. By integrating observed amplification patterns with regional background copy-number alteration (CNA) rates, this model estimates the selective advantage conferred by each event and distinguishes genuine drivers from passengers elevated by genomic instability. Applying this approach to segmented copy-number profiles from 554 ovarian tumors, we quantified amplification effects genome-wide. Known oncogenes such as MYC and CCNE1 exhibit high CES, reflecting strong positive selection. In contrast, some recurrent amplifications exhibit low CES, supporting a passenger interpretation. Notably, several focal amplifications at only moderate frequency exhibit strong selective advantage, identifying key CNA drivers unrecognized by conventional metrics. This stratification has direct therapeutic implications. High-CES amplifications identify loci that are both dosage-increased and selectively required for tumor fitness. These loci provide elevated triplex-forming oligonucleotide (TFO) binding sites and represent core cancer dependencies, linking target accessibility with selective vulnerability. Thus, they are promising targets for TFO therapies that induce DNA damage at triplex-forming motifs. By quantifying selection on amplifications, this approach provides an evolutionarily grounded strategy to identify true drivers and nominate actionable targets in chromosomally unstable cancers.

Abstract Title:	Targeting Oncogenic Chromatin Effectors: Enhanced BET inhibition via a BCL6-Recruiting RIPTAC
Abstract Authors:	**Skye Montoya,** Mackenzie W. Krone, J. Ignacio Vergara, Daniel E. Grinshpun, Krystyna Maruszko, Michael Q. Schottenhamel, Thomas Corner, Victoria Cantú-Rodriguez, Arseniy Butrin1, Maxime Mivelaz, Craig M. Crews
Abstract: **POSTER 20**	B-Cell Lymphoma 6 (BCL6) and Bromodomain and Extra-Terminal (BET) proteins are transcriptional regulators that drive neoplastic signaling. Despite preclinical success, no therapies targeting BCL6 or BETs have been approved to date due to insufficient in vivo efficacy and/or dose-limiting toxicity. To enhance the efficacy and precision of these monotherapies, we transformed potent BCL6 and pan-BET inhibitors into a RIPTAC (Regulated Induced Proximity Targeting Chimera), causing selective BET inhibition in BCL6-containing cells through chemically-induced proximity. Here we demonstrate that on-target inhibition through BET loss- of-function rather than transcriptional gain-of-function drives the RIPTAC mechanism, contrary to a prior study of bivalent molecules targeting these same proteins. These results suggest that dual engagement of BCL6 and BET proteins offers a promising induced proximity platform for treating lymphoma.

Abstract Title:	Glial Epigenomics of Opioid Use Disorder in the Human Postmortem Orbitofrontal Cortex
Abstract Authors:	**Diana L. Núñez-Ríos[1]**, José J. Martínez-Magaña[1], Melody Rivera-Hernández[1], Janitza L. Montalvo-Ortiz[1] 1. Division of Human Genetics, Department of Psychiatry, Yale University School of Medicine, New Haven
Abstract: **POSTER 21**	Epigenetic mechanisms—DNA methylation (5mC) and hydroxymethylation (5hmC)— are sensitive to environmental exposures and may modulate gene expression in OUD-affected brain cells. Reward-processing circuits like the orbitofrontal cortex (OFC) are involved in opioid use disorder (OUD). Our group previously identified OUD-associated 5mC and 5hmC alterations in neuronal cells isolated from the OFC. Here, we extended our investigation to OFC glial cells. Glial cells were isolated by fluorescence-activated cell sorting. Cases (n = 9; OUD diagnosis; mean age of death 34.8 years; mean postmortem interval (PMI) 28.3 hours; 6 European, 3 African ancestry) and controls (n=13; non-OUD; mean age of death 39.3 years; mean PMI 29.6 hours; 10 European, 3 African ancestry) from VABB were profiled using 6-base sequencing. Differential 5mC and 5hmC were identified with DSS and dmrseq R packages. Logistic regression models were adjusted for age at death, PMI, and three ancestry PCs (PC1–PC3). Single-site analysis identified two genome-wide significant CpG sites: one differentially methylated site (5mC; chr6:137406564) in an intergenic region, and one differentially hydroxymethylated site (5hmC; chr2:59078418) within LINC01122, a long intergenic non-coding RNA with high brain expression but as-yet uncharacterized downstream targets. This is the first study characterizing OUD-associated 5mC and 5hmC alterations in glial cells from the OFC. The limited epigenetic signal in glia - compared to the broader alterations we observed in OFC neurons – suggests that OUD-associated epigenetic dysregulation is more pronounced in neuronal than glial compartments of this brain region. Ongoing analyses will interrogate non-CpG methylation (CpH), to further elucidate cell-type-specific epigenetic mechanisms underlying OUD.

Abstract Title:	Switching off EGFR signaling: selective dephosphorylation through PhosTACs
Abstract Authors:	**Lorenzo Cavina**, Ryan Tsai, Saul Jaime-Figueroa, Craig Crews
Abstract: **POSTER 22**	Non-small cell lung cancer is often driven by overactive epidermal growth factor receptor, a protein on the cell surface that controls growth signals. Current drugs usually work by blocking the kinase activity of this receptor. Although these treatments can be effective at first, many tumors become resistant, and treatment can lose benefit. This project tests a different way to shut down the same cancer signal: removing the phosphate groups that keep epidermal growth factor receptor active. The proposed molecules are called phosphatase-targeting chimeras. They are designed to act as molecular bridges between epidermal growth factor receptor and PTPN2, a natural enzyme that removes phosphate groups from proteins. By bringing PTPN2 close to the activated receptor, these compounds aim to switch off cancer-promoting signaling in a catalytic manner, rather than simply blocking the receptor. This strategy could allow repeated signal removal by a single molecule and may offer a route to lower drug exposure and delayed resistance. The project will combine computer-guided molecular design, synthetic chemistry, protein-binding assays, and cell-based studies. Candidate compounds will be built from an epidermal growth factor receptor-binding component, a PTPN2-recruiting component, and linkers selected to bring the two proteins together. The compounds will first be tested for protein binding and induced complex formation, then evaluated for their ability to reduce receptor phosphorylation and downstream growth signaling in cell models. Lead

	molecules will be advanced into non-small cell lung cancer models, including cells with drug-resistant receptor variants. This work aims to make targeted dephosphorylation an accessible and adaptable therapeutic strategy for cancers driven by abnormal signaling.

Abstract Title:	Longitudinal High-Dimensional Proteomics Reveals Evolving CNS Immune Dysfunction during Early HIV
Abstract Authors:	**Nametso Kelentse**, Chang Lu, Jennifer Chiarella, Megan Byrd, Ruiqui Li, Kriti Agarwal, Richard Price, Yuval Kluger, Serena Spudich
Abstract: **POSTER 23**	We examined cerebrospinal fluid (CSF) samples from people with HIV (PWH) recruited during primary HIV infection (PHI, <1 year since HIV acquisition) followed longitudinally prior to ART initiation to interrogate the early effects of HIV in the CNS. PWH in the PISCES study donated CSF longitudinally prior to starting ART; age and sex matched people without HIV (PWoH) donated CSF once. Samples were assayed on the Olink Explore HT platform that quantitatively analyzes 5400+ protein biomarkers. Olink NPX values were used for analysis and longitudinal differential expression analysis was performed using the RolDE package. CSF was analyzed from 14 ART-naïve men with PHI enrolled at median 133 days after HIV acquisition then longitudinally followed for 42 total visits and from 20 male PWoH (median age 41 years in both groups). Thirty proteins were differentially expressed proteins as early as 133 days of HIV acquisition in men with PHI compared to PWoH. We identified 97 proteins by longitudinal differential expression analysis that distinguished PHI from controls and also evolved in untreated PHI over time during early infection (adjusted $p < 0.05$), with enrichment in pathways related to immune regulation and signaling. An immediate impact of HIV on the profile of protein expression in CSF was observed in men with PHI compared to matched PWoH, along with progressive changes over time in proteins associated with inflammation as well as healthy immune responses. Since ART is known to halt worsening of inflammation and injury in the CNS but not entirely ameliorate these processes, our findings support early identification and treatment after HIV transmission.

Abstract Title:	Structural Basis for Recruitment of Hypophosphorylated RNA Polymerase II for Late Gene Transcription
Abstract Authors:	**Pankaj Kumar Madheshiya**, Shannon Henry, Xinyu Chen, Taylor Gierke, Shaogeng Tang, Jimin Wang, Allison Didychuk
Abstract: **POSTER 24**	

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