The ACS-SA 20th Annual Undergraduate Research Symposium

Abstracts

Department of Chemistry and Biochemistry
University of California, San Diego

Thursday, May 14, 2026
4PM – 7PM

Keynote Speaker:

Dr. Christine Hrycyna

Christine (Chris) Hrycyna (her-SIN-a) is currently the Dean of the School of Physical Sciences and Chancellor's Associates Chair in Chemistry and Biochemistry at the University of California, San Diego. Prior to joining UC San Diego in September 2023, Professor Hrycyna was the Head of the Department of Chemistry in the College of Science and a 150th Anniversary Professor at Purdue University. She received her BA in Chemistry from Middlebury College and her PhD in Biochemistry with Prof. Steven Clarke in the Department of Chemistry and Biochemistry from UCLA. Prof. Hrycyna was subsequently a Jane Coffin Childs Memorial Fund for Medical Research Postdoctoral Fellow at the NIH in the National Cancer Institute with Dr. Michael Gottesman before starting as an Assistant Professor at Purdue in 2000. During her time at Purdue, she also served as Associate Department Head, Head of the Biochemistry Division in the Department of Chemistry, Head of the Interdisciplinary Life Science Graduate Program (PULSe), and as a representative on the University Senate and the University Educational Policy Committee.

Dr. Hrycyna's research focuses on the biochemical basis of aging disorders and cancer and the development of new treatments for brain, pancreatic and lung cancers. Her work has been supported by both federal and society research grants and she was named a University Faculty Scholar in 2017. Dr. Hrycyna has authored over 90 articles and book chapters and has graduated 24 Ph.D. students, 5 master's students and mentored more than 40 undergraduate researchers. She currently serves on the Editorial Board of the Journal of Biological Chemistry and has served as a standing member of the peer review committee on cancer drug development (CDD) of the American Cancer Society, and as a reviewer for the Ford Foundation and various NIH Special Study Sections.

Dr. Hrycyna has also received numerous teaching awards at Purdue including the Murphy Award for outstanding undergraduate teaching from the University, its highest teaching award. She also received the Arthur Kelly Undergraduate Teaching Award from the Department of Chemistry, was selected as Top Ten Teacher in the College of Science and is a member of the Purdue Teaching Academy and the Book of Great Teachers. In 2018, Prof. Hrycyna was named a Purdue University 150th Anniversary Professor to further recognize her high level of achievement in teaching and learning. Over the course of her career at Purdue, Prof. Hrycyna has also been involved with curricular development in the Department of Chemistry, through re-envisioning the chemistry sequence for life sciences majors and creating a new joint major in Chemical Biology in collaboration with the Department of Biological Sciences.

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Exploring the Effects of Mechanotransduction on RNA Base Editing in Hematopoietic Stem and Progenitor Cells

Tanner Abbruzzese, **Fumi Kaneko**
Principal Investigator: Dr. Catriona Jamieson

Hematopoietic disorders are highly complex, due to the variability in stem cell phenotypes across different disease contexts and the dynamic microenvironments they occupy (1). The hematopoietic system arises from hematopoietic stem and progenitor cells (HSPCs) that give rise to blood cell lineages (2). HSPCs sense extrinsic factors within their niches that influence their survival, proliferation, self-renewal capacity, and stress response, which can give rise to various diseases (3). The mechanosensitive cation channel, Piezo-type mechanosensitive ion channel component 1 (PIEZO1), enables cells to sense physical stimuli, such as the extracellular matrix composition, shear stress, and inflammation, thereby regulating key biological processes (4). In response to changes in the cellular microenvironment, PIEZO1 is activated, inducing calcium influx and influencing signaling pathways essential for cell survival and proliferation in both healthy and diseased states (5,6). Additionally, HSPC maintenance and responses to inflammation are regulated by enzymes that edit their diverse transcriptomes (7). For instance, adenosine deaminase acting on RNA1 (ADAR1) mediates adenosine-to-inosine (A-to-I) RNA base editing that modulates inflammation and HSPC self-renewal (7). Perturbations in ADAR1 activity are associated with various hematopoietic disorders (7). Previous research found an association between ADAR1 and PIEZO1 in regulating HSPC maintenance in samples derived from astronauts before, during, and after missions to the International Space Station (3). Therefore, we will explore how mechanical forces and RNA base editing by ADAR1 shape cellular pathways that contribute to hematopoietic health.

Mediating Mitochondrial Division: Decoding Drp1 Interactions With Mitochondrial Lipids

***Inci Alemdaroglu*, Tristan Gunther, Kailash Venkatraman**
Principal Investigator: Dr. Itay Budin

Mitochondria generate energy to be used throughout the cell. In human cells, mitochondrial fission is mediated by Dynamin-related Protein 1 (Drp1). After its recruitment to the outer mitochondrial membrane (OMM) with the help of a binding protein, Drp1 subunits oligomerize to form a ring around the mitochondria, and use energy from GTP hydrolysis to squeeze the mitochondria, inducing fission by mechanical forces. Dysregulation of mitochondrial fission is related to the development of various diseases, including cardiovascular diseases such as cardiomyopathy. Drp1 is a historically difficult protein to study as parts of it are naturally disordered and lack defined structures. These unorganized regions are called intrinsically disordered regions (IDRs). Drp1 binds to the OMM through its IDR, making this region crucial to study to learn more about mitochondrial fission. Cardiolipin, a mitochondrial membrane lipid, has been proposed to recruit Drp1 for mitochondrial fission, as Drp1 GTP hydrolysis is cardiolipin-dependent in vitro. However, in cells, cardiolipin is found almost exclusively in the inner mitochondrial membrane (IMM), so the mechanisms by which IMM-associated cardiolipin influences Drp1 activity are unclear. By carrying out an amino acid scan within the IDR, the positively charged C-terminal end of the Drp1 membrane binding domain was identified to have mitochondrial fission and Drp1 localization defects in cardiolipin containing and deficient cells, providing insight into possible Drp1-OMM binding mechanisms and future directions for investigating the IDR.

Regulation of chromatin conformation by the histone deubiquitinase BAP1 in the brain

Zakir Alibhai, **Challana Tea**
Principal Investigator: Dr. Cole Ferguson

BAP1 is a tumor-suppressor gene that removes the histone modification H2AK119ub, traditionally understood to mediate gene repression. Its loss causes severe neurodevelopmental defects including epilepsy, but how it shapes the three-dimensional folding of the genome has never been characterized in the brain. Given that H2AK119ub has been found to modulate active enhancer levels, we used Hi-C to map the physical contacts of DNA regions. We found genome-wide chromatin interactions to be perturbed in the BAP1 mutant, where 1 in 5 detected chromatin loops were dysregulated in the adult mouse cerebellum. At over 200 loci, long range loops associated with repression were preferentially lost in exchange for shorter range contacts. Additionally, the presence of H2AK119ub strongly predicted this distance-dependent loop-loss: if the histone modification was present at an anchor, contacts were ten times more likely to become disrupted. Analysis of differentially expressed genes revealed repression of developmental and synaptic genes where connections to enhancers were broken. This effect was progressive over neurodevelopment, expanding from under 200 differential loops in early development to almost three thousand in adulthood. These findings indicate that BAP1 is required for organization of the 3D genome in the developing brain, and its loss leads to architectural changes resulting in dysregulated synaptic gene expression. We propose that elevation of H2AK119ub, as a result of BAP1 loss, collapses long-range developmental loops, replacing them with proximal repressive contacts.

Exploring Amyloid-Beta Interactions in Disease Calcifications

Natasha Bercy, **Alexander Plonski**
Principal Investigator: Dr. Galia Debelouchina

Age-related macular degeneration (AMD) is the leading cause of vision loss in aging populations, significantly impacting quality of life. The condition is characterized by small extracellular deposits that accumulate in the retina named drusen. Drusen are composed of a lipid core, a mineral layer of calcium-based hydroxyapatite (HAP), and a protein-coated surface. Amyloid Beta (A-β) has been found in the protein layer of drusen and is also widely known to be associated with Alzheimer's disease where it forms cytotoxic fibrils. However, its role in AMD and its interactions with the minerals in drusen during the mineralization process is largely unknown. Therefore, this project aims to explore how A-β interacts with hydroxyapatite and whether these interactions lead to A-β aggregation. Here we show how A-β aggregation kinetics are modulated in the presence of hydroxyapatite using ThT kinetic assays and cosedimentation assays. In addition, transmission electron microscopy was used to characterize the deposits formed from kinetic assays. To enable these experiments, an optimized protocol was developed for the recombinant expression and purification of highly pure and monomeric A-β. These experiments provide insights into the interactions between A-β and calcification in diseases such as AMD.

Changes in the Lipid Profile and Amino Acid Composition of Various Organs Following Oophorectomy in Women

Nicole Cao, **Ramya Kuna**
Principal Investigator: Dr. Christian Metallo

An oophorectomy describes the surgical removal of one or more ovaries, commonly done in response to ovarian cancer, cysts, endometriosis, and other medical problems. The ovaries are responsible for secreting major sex hormones such as estrogen and progesterone. The removal of ovaries has negative effects on organs due to the sudden drop in major sex hormones. Studying the lipid and amino acid composition of various organs immediately following oophorectomy can reveal metabolic changes that could be associated with symptoms. In this research area, many studies focus on the effects of one or two organs. This study seeks to directly compare effects in multiple organs to map comprehensive metabolic changes. More research on immediate changes in various organs following oophorectomy, particularly amino acids, is an area this study will add to, as well as replicate changes in lipid metabolism outlined in previous studies. Each of the various organ samples were collected in two phases: polar (amino acids) and nonpolar (lipids). The polar samples were run under gas chromatography-mass spectrometry (GC-MS), and the nonpolar samples were run under liquid chromatography-mass spectrometry (LC-MS). Amino acids and lipids were analyzed by their relative concentration in Excel and GraphPad Prism. Current results from blood plasma, skeletal muscle, and liver show an overall significant decrease in most amino acids in the organs with a few exceptions. Overall lipid composition results match previous studies. There is ongoing work in characterizing remaining tissue samples.

Activity Optimization of a de novo Vanadium Dependent Haloperoxidase

Lancia Carson, **Ryan Herold**
Principal Investigator: Dr. Akif Tezcan

Due to recent advances in machine learning technology, allowing accurate protein structure prediction from amino acid sequence alone, possibilities for enzyme design projects have expanded greatly. Metalloenzymes, which make up over 40% of all enzymes, possess unique catalytic abilities that make them attractive targets for de novo design. Haloperoxidases, which are a class of metalloenzymes capable of adding halogens to organic substrates, perform reactions that are vital for the synthesis of many pharmaceuticals, industrial chemicals, and agrochemicals. A new de novo enzyme called VAN1 uses a VO43- center to catalyze the challenging oxidation of bromide (Br-) for reactions with nucleophilic substrates, making it the first example of a de novo haloperoxidase. However, since the activity of the enzyme is relatively low, optimization with rational mutagenesis has been the goal of recent work. Based on the x-ray crystal structure, two glutamates in the primary coordination sphere suspected of impeding on the VO43- binding site were substituted with either alanine or asparagine. V51 NMR data show that the four protein variants have stronger VO43- affinity compared to the original construct. Metal binding titrations with Zn2+ also showed the variants having decreased affinity for Zn2+ compared to VAN1, which further supports that the mutation had the intended effect on the protein, as the glutamates supported Zn2+ binding. As the original construct has been shown to be highly thermo- and chemically stable, improvement of this construct would be an advance in the field of metalloenzyme design and potentially industrially relevant.

Investigating the role of rhomboid Dfm1 in maintaining misfolded protein and ubiquitin homeostasis in the ER

Aranyani Carter-Perkins, **Devanshi Agarwal**
Principal Investigator: Dr. Sonya Neal

Proteins are essential to cell function and are synthesized into specific structures. The folding site for most proteins are in the endoplasmic reticulum (ER). During the protein folding process however, they may become misfolded and aggregated, which has been linked with numerous diseases. This misfolding problem requires degradation via the cell's protein quality control pathway. The rhomboid Dfm1 is an intermembrane pseudoprotease localized in the ER membrane that mediates such process either by retrotranslocation or a chaperone-like function to prevent protein aggregation. It works alongside pathways like the Ubiquitin Proteasome System (UPS) that uses ubiquitin enzymes to tag target misfolded proteins for degradation. In the absence of Dfm1, cells with misfolded proteins elicited cell stress, indicating Dfm1's importance in mediating cellular stress levels. What remained unanswered however, was the possible cause of cellular stress. When misfolded proteins aggregate, they are tagged via polyubiquitination where much of the free ubiquitin available in the cell is sequestered. Since ubiquitin has various signalling roles in the cell other than protein degradation, a free ubiquitin pool is required for cellular function, and its depletion leads to cellular stress. We have shown that restoration of this free ubiquitin pool in Dfm1-deficient cells rescued this cellular stress phenotype, implicating Dfm1 to possess a dual protective role in ER protein quality control modulating protein as well as ubiquitin homeostasis. Our work reveals important links between protein aggregation and ubiquitin exhaustion, offering a new framework for how misfolded proteins lead to cellular stress.

The expression of LPMO in Enterococcus faecalis (EF)

***Jeremiah Cheav,* Armin Kousha, Fatemeh Askarian**
Principal Investigator: Dr. Victor Nizet

Lytic Polysaccharide Monooxygenases (LPMOs) are enzymes that rely on the redox chemistry of copper to oxidize polysaccharides. The redox chemistry allows the breakdown of recalcitrant polysaccharides associated with an increased rate of infection. Our project aims to investigate the expression of LPMO in Enterococcus faecalis (EF) by evaluating the growth of EF WT V583 and ΔLPMOEF in artificial pooled human urine(aPHU) and artificial urine medium(AUM). We will also examine if LPMOEF deletion is impacted by the differing adaptations of aPHU and AUM.

Reducing Bias in Virtual Screening: A Chemically Diverse Benchmark for NSP13 Inhibitor Discovery

***Akshat Choudhari*, Alma Castañeda**
Principal Investigator: Dr. Rommie Amaro

The development of realistic and diverse benchmarking datasets is essential for consistent evaluation of virtual screening (VS) performance in drug discovery. Existing benchmarks are often compromised by artificial enrichment, limited chemical diversity, and analogue bias, making them unrepresentative of real screening libraries and leading to overly optimistic results. Here, we present a new benchmarking dataset derived from ~650,000 experimentally tested compounds against SARS-CoV-2 nsp13. The subset created is characterized by having a maximized 1,621:N active-to-decoy ratio to ensure a large and ML stable behavior and incorporates structurally similar decoys to increase task difficulty. The primary objective of this work is to construct a dataset that closely mirrors the structural distribution of the virtual screening (VS) space, using over 250 million compounds from 16 commercial libraries to ensure broad coverage and adherence to frequently observed chemotypes in VS campaigns. To support this, we introduce and evaluate two complementary dataset construction strategies: UMAP-based embedding and high-throughput structural clustering via BitBirch. UMAP enables broad chemical space coverage but may distort local structure, whereas clustering preserves fingerprint-level detail but can bias selection toward dense regions. We evaluate both approaches independently and in combination. Our results demonstrate that UMAP-based strategies achieve superior coverage of the VS space, as supported by physicochemical profile analyses and nearest-neighbor similarity distributions. Furthermore, UMAP-based datasets yielded more challenging benchmarks, characterized by similar diversity values to the VS space and leading to consistently lower machine learning performances.

Mitochondrial transplantation reduces inflammation and modulates metabolism in macrophages during metabolic dysfunction-associated steatohepatitis (MASH)

***Daniella Esho*, Avinash Mukkala, Rykann Tamayo, Alexander Medina, Katherine Quach, Nihar Pasupuleti**

Principal Investigators: Dr. Michael Karin

Mitochondrial transplantation (MitoTx) has been shown to improve cellular energy metabolism, and restore mitochondrial function. Here, we assessed the effects of MitoTx in bone marrow–derived macrophages (BMDMs) under metabolic dysfunction–associated steatohepatitis (MASH)-like conditions ex vivo. MASH is a chronic liver disease characterized by inflammation that can progress to cirrhosis and hepatocellular carcinoma. To model key features of MASH ex vivo, bone marrow was isolated from mouse hind limbs and differentiated into BMDMs over 7 days. Cells were then exposed to MASH medium (MM; palmitate, oleate, lipopolysaccharide, and fructose) for 24h, followed by co-incubation with mouse skeletal muscle–derived mitochondria (25 μg/mL) for an additional 24h. MM induced mitochondrial dysfunction in BMDMs, evidenced by decreased oxygen consumption rate, spare respiratory capacity, maximal respiration, and coupling efficiency, and mitochondrial membrane depolarization. BMDMs rapidly internalized exogenous mitochondria ex vivo. MitoTx attenuated inflammatory cytokine expression in MM-treated BMDMs, significantly reducing Il-6, Il-1b, and Tnfa. Concurrently, MitoTx upregulated NRF2-dependent metabolic genes, including Hmox1 and Gclc, and modulated metabolic regulators such as Plin2 and Arg2. Importantly, MitoTx markedly reduced intracellular lipid accumulation, as assessed by BODIPY staining and confocal microscopy, indicating reduced activation. Collectively, these findings demonstrate that MitoTx reprograms macrophage immunometabolism under MASH-like conditions by suppressing inflammatory cytokine expression, enhancing NRF2-driven metabolic gene programs, and decreasing lipid burden. These results support MitoTx as a therapeutic strategy for MASH and provide a foundation for future mechanistic studies to define its translational potential.

Cobinamide, A Vitamin B12 Analog, Attenuates Benzo[a]pyrene and Pyrene Toxicity Through Selective Redox Modulation

Weirui Gao, **John Tat**
Principal Investigator: Dr. Gerry Boss

Polycyclic aromatic hydrocarbons (PAHs) are widespread environmental contaminants produced during incomplete combustion and are associated with mutagenic and cytotoxic effects due to their persistence and bioaccumulation. Benzo[a]pyrene (B[a]P), a prototypical carcinogenic PAH, and pyrene generate reactive oxygen species (ROS) through CYP1A1-mediated metabolism and quinone redox cycling, leading to oxidative stress and cellular damage. This study evaluated cobinamide, a vitamin B12 analog with potent antioxidant properties, as a protective agent against PAH-induced toxicity.

In H9C2 rat cardiomyoblasts and A549 human lung epithelial cells exposed to B[a]P (10 μM) or pyrene (10–100 μM), cobinamide (5–10 μM) significantly improved cell viability, reduced JNK/p38 phosphorylation, and decreased oxidative damage to DNA and proteins. Cobinamide also lowered DNA strand breaks and cleaved caspase-3 levels, indicating reduced apoptosis. Gene expression analysis revealed suppression of inflammatory markers (TNF-α, IL-1β, IL-6) and oxidative stress genes (HMOX1, NOX4), alongside increased antioxidant defense (SOD2) and maintenance of CYP1A1 expression.

In Drosophila melanogaster, cobinamide (2 mM) enhanced survival and fully restored locomotor function following PAH exposure, outperforming cobalamin and N-acetylcysteine. Spectroscopic analysis showed no direct binding between cobinamide and PAHs, suggesting its protective effects are mediated through redox modulation rather than chemical interaction.

Overall, these findings demonstrate that cobinamide effectively mitigates PAH-induced toxicity while preserving metabolic function, supporting its potential as a therapeutic strategy against environmental toxicant-induced damage.

Recent Advances and Current Challenges in Cancer Nanomedicine

Kassidy Gillespie, **Torrey Rhyne**
Principal Investigator: Dr. James Kadonaga

Cancer remains a leading cause of mortality worldwide, highlighting the need for more precise and effective therapies. Nanomedicine offers a promising approach by utilizing nanoscale systems to enhance drug delivery, improve targeting, and reduce systemic toxicity. This review examines recent advances in nanoparticle-based cancer therapeutics, focusing on targeting strategies, multifunctional platforms, and barriers to clinical translation. A literature review of studies published between 2006 and 2026 was conducted using PubMed, the National Institutes of Health database, the American Chemical Society database, and Google Scholar. Keywords included cancer nanomedicine, enhanced permeability and retention effect, theranostics, and clinical trials. Relevant studies were identified through title and abstract screening, followed by full-text analysis. Advances in nanomedicine have improved drug delivery through passive and active targeting. Passive targeting exploits tumor vascular permeability, while active targeting enhances specificity via ligand–receptor interactions. Emerging approaches, including carrier-free and stimuli-responsive systems, improve delivery efficiency. Theranostic platforms further integrate diagnostics and therapy, enabling enhanced detection and monitoring. However, tumor heterogeneity, biological barriers, and nanoparticle design limitations contribute to inconsistent outcomes. Despite its potential, clinical translation remains limited by variability in tumor microenvironments, incomplete understanding of nano–bio interactions, and challenges in reproducibility and manufacturing. Discrepancies between preclinical models and human physiology further reduce efficacy. Nanomedicine represents a significant advancement in oncology, with potential to improve treatment precision and outcomes. Future work should optimize nanoparticle design, refine preclinical models, and integrate combination strategies to enhance clinical success.

Exploring the Role of α6GABAA Receptors in Visual Processing Potential Therapeutic Strategies

Angelina Ruoxi Huang, **Yu-Hsuan Lee**
Principal Investigator: Dr. Lih-Chu Chiou

Glaucoma is a leading cause of irreversible blindness, driven in part by excitotoxicity resulting from an imbalance between excitatory and inhibitory neurotransmission in the retina. Excessive glutamate signaling can lead to retinal ganglion cell (RGC) death and progressive neurodegeneration. While current treatments focus primarily on lowering intraocular pressure (IOP), they do not directly address the underlying neurodegenerative mechanisms. In this study, we investigated the neuroprotective potential of DK-I-56-1, a highly selective positive allosteric modulator (PAM) of α6GABAa receptors, in a mouse model of acute glaucoma induced by retinal ischemia-reperfusion (IR) injury. We confirmed that Gabra6, the gene encoding the α6 subunit, is expressed in the inner plexiform layer of the retina and that its expression is significantly downregulated following IR injury. Mice treated with DK-I-56-1 shortly after injury exhibited preserved retinal architecture and significantly increased RGC survival compared to vehicle-treated controls, as demonstrated by histological and immunohistochemical analysis. These findings suggest that enhancing GABAergic inhibition via α6GABAa receptor modulation can counteract excitotoxic damage and promote retinal neuroprotection. DK-I-56-1's receptor specificity offers a promising therapeutic advantage by minimizing off-target effects, highlighting its potential as a novel adjunct or alternative to traditional IOP-lowering therapies in the treatment of glaucoma and related neurodegenerative eye diseases.

Prime Editing Streamlined Target Optimization (PESTO) of V617F mutation in JAK2 gene

Sanjana Kumar, **Kara Dunne-Dombrink**
Principal Investigator: Dr. Alexis Komor

Prime editing (PE) is capable of doing genomic DNA edits without a need for double stranded breaks (DSBs) or a homologous template (HDR). A major bottleneck of Prime Editing technology is the optimization of prime editing guide RNAs (pegRNAs). Prime Editing Streamlined Target Optimization (PESTO) is a high-throughput system that refines gene editing efficiency by decreasing the optimization time of various pegRNAs. PESTO involves constructing a lentiviral library of pegRNAs to test on the same plasmid containing the respective target genes APP, PSEN1, JAK2, CFTR, and BAP1. With this cellular system, PESTO screens can be designed and executed by testing different prime editor backbones (via cellular transfection) alongside a cell line containing the pegRNAs in mammalian cell lines HEK293T and K562s. After editing, the cells are sorted via fluorescent proteins like GFP and BFP. Genomic DNA from the cell line is amplified for Illumina Next-Generation Sequencing. In optimizing the bioinformatics pipeline for the readout of the screen from NGS, the optimal pegRNA:PE combination that corrects these harmful mutations can be determined in an efficient manner.

Spatiotemporal Organization of the Fibrotic Scar After Spinal Cord Injury

Gauri Mathrakott, **Camilo Londoño**
Principal Investigator: Dr. Binhair Zheng

Trauma to the central nervous system triggers a robust inflammatory response that leads to the formation of a fibrotic scar at the lesion site. Activated fibroblasts infiltrate the SCI lesion site and progressively deposit inhibitory extracellular matrix components (ECM), including collagens, over the first 14 days post-injury (dpi). Performing immunohistochemistry on injured spinal cord sections to label for glial and fibrotic scar markers at 3, 7, and 14 dpi illustrated the dynamic maturation and formation of the fibrotic scar and progressive deposition of ECM from the subacute to chronic phase after severe SCI. The internal organization of the fibrotic scar was examined by immunostaining for ECM and fibrotic markers (Collagen 1 & 6 and PDGFRα & β). Quantitative mean-pixel intensity analysis indicated that the mature fibrotic scar is compartmentalized into a distinct core and border regions with differential fibrotic ECM composition. Using lineage tracing on spinal cord injured tissue sections, Col1a2+ cells exhibit temporally regulated localization into the fibrotic scar and express canonical fibroblast markers PDGFRα and PDGFRβ following SCI. These findings identify Col1a2+ fibroblasts as key contributors to fibrotic scar formation. Future perturbation of signaling pathways, such as TGF-β signaling, could provide mechanistic insights as to how fibrotic scar formation is regulated.

Identifying new small molecules as starting points for tropical disease drug discovery

***Alysh Mercer*, Karol R. Francisco, Denise Tran**
Principal Investigator: Dr. Conor R. Caffrey

Purpose: Human African Trypanosomiasis (HAT) is a deadly neglected tropical disease (NTD) affecting poor rural communities in sub-Saharan Africa. Current drugs have problems relating to toxicity, administration and resistance. To identify possible novel treatments for HAT, we screened the Trypanosoma brucei pathogen against 400 preclinical and clinical drug- like compounds that comprise the "Pandemic Response Box" (PRB), which is made available gratis by the Medicines for Malaria Venture (MMV) to kick-start NTD drug discovery.
Methods: Between November 2025 and February 2026, the PRB compounds were quantitatively screened for bioactivity against the T. brucei pathogen and the human HEK 293T cell line to identify compounds with a strong differential (selectivity index) for inhibiting parasite growth. Assays were performed in biological triplicate and technical duplicate. Data were analyzed using GraphPad Prism and the concentration that inhibits cell growth by 50% (EC50) calculated using a sigmoidal four parameter logistic curve.
Results: We identified 11 'hits' with EC50 values vs. T. brucei $\leq$ 1 μM. Of these, seven compounds generated selectivity indices > 10 relative to HEK 293T cells, with four compounds offering indices $\geq$ 50. Among the latter was a previously established antitrypanosomal agent, a human farnesyl diphosphate synthase inhibitor and an experimental antibacterial not known to possess parasiticidal activity.
Conclusions: This project identifies potent and relatively non-toxic small molecules, one or more of which might advance preclinically as a novel antitrypanosomal drug. Our findings demonstrate the value of the MMV's PRB to generate new uses for existing drugs and drug candidates.

The C-terminal PDZ-binding motif of Cx40 is regulated by phosphorylation and required for the formation of functional intercellular channels

***Aiden Momtaz**, Luya Wei*, Curtis Furukawa, S Bandi, Nishant Sharma, Ben Bridgelal, E Zheng, Jingyi Shirley Xie, Sofia Endzhievskaya, Alexis Lona, Grant Wadman**
Principal Investigator: Dr. Irina Kufareva

Connexins are a family of proteins that form gap junction channels (GJCs) mediating intercellular communication and allowing for the passage of ions and small molecules. Connexin40 (Cx40) is downregulated in pulmonary arterial hypertension (PAH), and restoring its functions rescues the disease phenotype in mice, making Cx40 a potential therapeutic target for PAH. However, the mechanisms that mediate Cx40 trafficking, GJC formation, and intercellular permeability are not well understood. Via bioinformatics analyses, we discovered that, similar to other members of the connexin family, Cx40 has a C-terminal PDZ binding motif (PDZbm), but, uniquely, its PDZbm also has phosphorylatable residues. We hypothesized that phosphoregulated binding of Cx40 PDZbm to yet-unknown PDZ domain-containing proteins in the intracellular trafficking cascade may mediate its cellular localization and function. Through immunofluorescence microscopy, we demonstrated that deleting the PDZbm or mutating the phosphorylatable serines altered Cx40 GJC plaque formation on cell membranes. The likely kinases that generate different Cx40 PDZbm phosphoforms were predicted using motif-based scoring and confirmed to coexpress with Cx40 in publicly available endothelial cell scRNA-seq datasets. Furthermore, an in silico screen identified several PDZ domain-containing proteins (MAST4, SNX27) as candidates for Cx40 PDZbm interaction, which also coexpress with Cx40 in endothelial cells. Differentially phosphorylated forms of the PDZbm were predicted to preferentially bind to distinct PDZ domains. Taken together, these findings indicate that C-terminal phosphorylation could be a key regulator of Cx40 trafficking in endothelial cells and a promising target for pharmacologically manipulating Cx40 GJC permeability in PAH.

Optimized Protein Aggregation Capture Improves Peptide Recovery and Reproducibility in Proteomics

Lisa Morishita-Cartwright, **Dominic McGrosso**
Principal Investigator: Dr. Sam Myers

Protein Aggregation Capture (PAC) is an increasingly adopted method for proteomics sample preparation. Unlike traditional methods like FASP or in-gel digestion methods, PAC can offer faster, simpler, and lower cost sample preparation for MS-based proteomics. However, its performance is highly sensitive to experimental conditions, often leading to significant sample loss and inconsistent recovery.

Here, we systematically investigated the PAC workflow to improve protein capture efficiency and peptide recovery for MS-based proteomics. Through iterative optimization, we evaluated key parameters including buffer pH, detergent concentration, protein concentration, bead amount, and incubation conditions.

We found that protein aggregation and their binding to the beads require strictly basic conditions (pH > 8.5) and reduced detergent levels to promote efficient capture and recovery. Importantly, introducing extended incubation time and controlled mixing condition significantly reduced protein loss in flow-through fractions. Furthermore, optimization of incubation time and agitation revealed that continuous mixing for 45 minutes produced the most consistent results.

Overall, this work establishes a robust and reproducible PAC workflow and highlights critical parameters governing protein aggregation–based capture. These findings improve data quality and provide a practical framework for implementing PAC in proteomics pipelines.

Molecular Dynamics of the SARS-CoV-2 Helicase

***Benjamin Ngo*, Javier Sanlley Hernandez, Alma Castañeda**
Principal Investigator: Dr. Rommie Amaro

The SARS-CoV-2 helicase plays a critical role in viral replication by unwinding RNA during genome transcription and replication. To investigate how RNA binding influences the protein's conformational dynamics, we performed molecular dynamics simulations of the helicase in both its APO (unbound) state and RNA-bound state. Comparative analysis revealed that RNA binding stabilizes key structural regions involved in nucleic acid recognition and unwinding, while increasing flexibility in distal domains that may facilitate translocation along the RNA strand. Additionally, specific residues within the helicase active site and RNA-binding channel were identified as key contributors to RNA interaction through persistent hydrogen bonding and electrostatic contacts. These findings highlight the interactions between structure and function in the helicase and provide insight into how RNA binding modulates its activity. Overall, our studies can be helpful for understanding how protein dynamics can inform novel strategies for inhibiting helicase activity, and how these can be intercepted by small molecule inhibitors.

Exploring the mechanism behind RPS3 mediated NFkB transcription

Jeongwon Oh, **Anthony Cai**
Principal Investigator: Dr. Elizabeth Komives

NF-κB is a key transcription factor that regulates immune responses, inflammation, and cell survival. In resting cells, NF-κB is sequestered by its inhibitor IκBα in the cytoplasm, and its dysregulation is linked to diseases such as cancer and chronic inflammation. Ribosomal Protein S3 (RPS3) has been identified as a non-Rel regulator that enhances NF-κB-mediated transcription, but its mechanism of action remains unclear. This study aims to determine how RPS3 regulates NF-κB signaling through direct interaction with IκBα. I will use Biolayer interferometry (BLI) will measure the binding kinetics and affinity of this interaction, providing quantitative insight into its strength and stability. Fluorescence anisotropy assays will evaluate whether RPS3 alters NF-κB DNA-binding affinity, allowing assessment of the functional consequences of this interaction on transcriptional regulation. I anticipate observing measurable changes in NF-κB DNA-binding behavior in the presence of RPS3, supporting a functional regulatory role. These findings will provide insight into a previously unresolved regulatory mechanism within the NF-κB pathway. Understanding how RPS3 modulates NF-κB signaling may clarify its role in disease-associated dysregulation and identify potential targets for therapeutic intervention. Future studies could further investigate structural determinants of this interaction and its impact in cellular systems.

Investigating the sequence dependence of an alkaline-activated metal-sensing riboswitch

Avery Ontiveros, **Danea Palmer**
Principal Investigator: Dr. Tatiana Mishanina

RNA-based gene regulation is essential for most cellular processes, and riboswitches in bacterial cells are an important example of such regulation. Riboswitches are mRNA elements that undergo conformational changes to promote or inhibit the expression of a downstream gene. There are two manganese-sensing riboswitches in E. coli that control manganese transporter proteins, alx and mntP. The alx riboswitch also responds to changes in intracellular pH and is the only known riboswitch to respond to these two cellular stimuli. The alx riboswitch has a significantly increased response to Mn2+ at alkaline pH. My project aims to investigate whether the mechanism of this unique pH response is sequence dependent. To achieve this, I perform molecular cloning to swap the Mn2+ binding site sequences of alx and mntP. To assess the pH response of these mutated riboswitches, I perform in vivo β-Galactosidase assays to quantify the translation of the mutant alx and mntP riboswitches under high pH conditions. This work is key to understanding if the mechanism of alx riboswitch pH response is sequence or structure dependent.

De novo protein design of tale-based protein binders to target non-canonical DNA forms

***Richie Pham*, Luke Sebastian**
Principal Investigator: Dr. Neville Bethel

Z-DNA is a left-handed DNA conformation with demonstrated biological roles, including association with neurodegenerative diseases such as Alzheimer's and regulation of downstream genomic transcription. Despite its biological relevance, the functional role of Z-DNA remains poorly understood, largely due to the scarcity of high resolution Z-DNA-protein structures. This gap reflects the limited repertoire of existing Z-DNA binding proteins. De novo protein design has recently yielded novel binders to a range of protein and DNA targets, but has yet to be applied to target alternative DNA conformations. We chose Transcription Activator-Like Effectors (TALEs) as our design scaffold, given their modular architecture in which each repeat unit independently recognizes a single base pair through both specific sequence recognition and geometric shape complementarity. By reparameterizing these repeat modules to match Z-DNA backbone geometry and using the resulting designs as input seeds for deep-learning protein design models, we generated a set of TALE-based Z-DNA binders (zTALEs) with sequence-specific recognition capability and geometric complementarity to the Z-DNA backbone. Computational evaluation indicates structural viability and favorable predicted binding geometry, and these designs are now poised for experimental validation. If successful, zTALEs would provide a robust, programmable platform for detecting and investigating Z-DNA in biologically relevant contexts.

Solvatochromic probes capture inter-domain interactions in carrier protein-dependent biosynthesis

***Kyle Rich*, Matthew Miyada**
Principal Investigator: Dr. Michael Burkart

Carrier protein (CP)-dependent biosynthesis pathways produce a broad range of important metabolites, including many clinically relevant natural products. These pathways rely on a carefully orchestrated series of enzymatic reactions mediated by a central carrier protein and multiple partner proteins. However, the protein-substrate and protein-protein interactions that enable these processes are often subtle, transient, and difficult to monitor in situ. Solvatochromic fluorophores offer a promising strategy to probe these interactions. These fluorescent molecules exhibit shifts in emission wavelength and intensity depending on their local chemical environment, allowing them to report changes in protein interactions. Such probes can be chemically synthesized, selectively attached to carrier proteins, and used to visualize CP-dependent pathways involving partner proteins such as fatty acid synthases (FAS), polyketide synthases (PKS), and nonribosomal peptide synthetases (NRPS). In this work, we demonstrate the application of solvatochromic probes to monitor inter-domain interactions within type I NRPS system. Using mutagenesis at the peptidyl carrier protein-epimerization (PCP-E) domain interface, interactions were detected with the solvatochromic 5-(4″-dimethylaminophenyl)-2-(4′-phenyl)oxazole (dapoxyl) pantetheinamide probe. These findings highlight the utility of solvatochromic probes for detecting dynamic inter-domain protein interactions and suggest broader applications for studying carrier protein-mediated biosynthetic pathways.

Investigating the Role of Nuclear Localization in NAB2-STAT6 - Mediated Transcriptional Activity in Solitary Fibrous Tumors

Catalina Sepulveda Florez, **Qiushi Zhao**
Principal Investigator: Dr. Daniel J Donoghue

Solitary fibrous tumors (SFTs) are rare mesenchymal neoplasms defined by the NAB2–STAT6 fusion protein, which arises from an intrachromosomal inversion on chromosome 12. Although this fusion is a hallmark of SFT, its oncogenic mechanism remains incompletely understood. In its native form, NAB2 functions as a transcriptional corepressor of EGR1, while STAT6 acts as a transcriptional activator in cytokine signaling pathways, particularly downstream of IL-4/IL-13. The resulting fusion protein is proposed to act as an aberrant transcriptional regulator that requires nuclear localization to drive gene expression.

We hypothesize that nuclear localization enhances NAB2–STAT6 transcriptional activity, leading to upregulation of EGR1 and downstream increases in growth-promoting signaling. Elevated expression of target genes such as IGF2 may contribute to autocrine activation of IGF1R signaling, sustaining proliferative pathways and promoting tumorigenesis. To investigate this, we generated NAB2–STAT6 mutants targeting predicted nuclear localization domains and evaluated their effects on subcellular localization. In parallel, we assessed downstream transcriptional output to determine the functional consequences of altered nuclear localization. This work aims to clarify the role of nuclear localization in regulating NAB2–STAT6 function and to provide mechanistic insight into how this fusion protein contributes to oncogenic transcriptional programs in SFT.

AI-powered discovery of peptide binders for the orphan GPCR BB3, a new target in lung adenocarcinoma

Lilian Spock, **Tyler Thurlimann**
Principal Investigator: Dr. Irina Kufareva

Bombesin receptor 3 (BB3) is a class A orphan GPCR homologous to the neuromedin B receptor and gastrin-releasing peptide receptor. BB3 expression in the human body is scarce, but it is uniquely upregulated in lung adenocarcinoma (LUAC). The specific and limited expression in LUAC makes BB3 a viable target for toxic payload delivery to cancer cells. Unfortunately, the few reported agonists and antagonists of BB3 have low solubility, selectivity, and potency.

This project seeks the discovery of novel peptide binders for BB3 with the help of artificial intelligence (AI). Starting peptide geometries were identified by an unbiased in silico screen of ~1000 known secreted bioactive peptides (6-105aa) against BB3, in addition to a natural amino-acid analog of dY-Bn(6-14), a known BB3 peptoid agonist. A total of 31 models of five BB3-peptide complexes were partially diffused by RFdiffusion to produce 295 diversified interaction geometries, redesigned as 7317 new AA sequences with ProteinMPNN, modeled ab initio with BB3 using AlphaFold3, and evaluated by an in-house pipeline to prioritize the most likely binders. All high-scoring peptides surpassed the design starting points, and 22 were chosen for laboratory verification to provide starting points for the discovery of potent and selective BB3 binders and the endogenous ligands of BB3. Of the 22 peptides, 2 showed partial agonism and 2 antagonism of BB3 in the Ca^{2+} mobilization assay using the GCaMP6f biosensor.

Mitochondrial transplantation reduces inflammation and rewires metabolic gene programs during murine metabolic dysfunction-associated steatohepatitis (MASH)

Rykann Tamayo, **Avinash Naraiah Mukkala**
Principal Investigator: Dr. Michael Karin

Metabolic dysfunction-associated steatohepatitis (MASH) is a chronic liver disease with increasing incidence, characterized by persistent inflammation that drives progression to hepatocellular carcinoma (HCC). Mitochondrial dysfunction is a key feature of MASH. Mitochondrial transplantation (MitoTx) is a novel biotherapeutic with potential to prevent MASH-driven HCC, as prior studies demonstrate immunomodulatory effects following MitoTx. Previous work has shown that MitoTx protects murine livers from acute injury through the VSIG4 immunoreceptor. We hypothesized that MitoTx reduces hepatic and systemic inflammation and modulates metabolic gene programs in MASH. Six-week-old, ER-stress prone, MUP-uPA mice were fed high-fructose diet (HFrD) or corn-starch diet (CSD) control for 16 weeks and received biweekly intraperitoneal injections of isolated muscle-derived mitochondria or vehicle between weeks 6–12 (132.5 μg/mouse, ~5.5 μg/g). At 16 weeks, liver and plasma were collected for sandwich ELISAs, qPCR, and colorimetric assays. Hepatic macrophages (CD45+/F4/80+/CD11b+) rapidly internalized transplanted mitochondria. MitoTx was hepatoprotective in HFrD-fed mice, reducing cholesterol, ALT, and bilirubin. MitoTx decreased hepatic Il6 and Il1b mRNAs and reduced liver IL-6, TNF, and IFNβ proteins. Systemically, plasma IL-6, TNF, IL-1β, IFNβ, and IL-17A/F were diminished. MitoTx upregulated NRF2 target genes (Hmox1, Gclm, Gclc), downregulated lipid metabolism genes (Plin2, Lipa, Fabp5, Fabp4), and rewired amino acid metabolism genes (Aco2, Arg1, Gls). Collectively, MitoTx reduced inflammation and reprogrammed metabolic gene expression. Future studies will define mechanisms to position MitoTx as a MASH and cancer-preventative agent.

Using computational tools to find optimized drug candidates to treat TB

Praharshitha Thumati, **Haixin Wei**
Principal Investigator: Dr. Andy McCammon

Mycobacterium tuberculosis (Mtb), the bacteria that causes tuberculosis (TB), is estimated to affect a quarter of the world's population and kills more than a million people per year. The current treatments for TB are a very costly process and require an intense cocktail of drugs to be taken for months. Given the prevalence of this disease, there is a need to develop a more optimal cure. Computational tools can cover this gap and optimize already working drugs or drug candidates, leading to a more efficient and faster treatment. Cytochrome bd, which is an oxidase found in the electron transport chain (ETC) of Mtb, is the target that is the focus of this research. Previous research on this project has discovered three drug candidates that inhibit the function of cytochrome bd--in effect killing the bacteria. Running Molecular Dynamic (MD) simulations on variations of the new drug candidates yield narrowed selections that will be tested experimentally by collaborators. From these MD simulations, we can determine the potential of the various ligands as TB drugs, by harnessing the power of computational biochemistry. Methods such as membrane-building, clustering, docking, and top-hit fragmenting are used to potentially find a better cure for TB.

Ubiquitination of HIV-1 p6 Enhances ALIX Recruitment via Avidity-Driven Interactions

Allison Woo, **Spencer L. Nelson**
Principal Investigator: Dr. Lalit Deshmukh

Human immunodeficiency virus type 1 (HIV-1) hijacks the host Endosomal Sorting Complex Required for Transport (ESCRT) machinery to facilitate viral budding and membrane scission. This recruitment is primarily mediated by the interaction between the p6 domain of the viral Gag polyprotein and the host adaptor protein ALIX. While Gag ubiquitination is known to occur during budding, its precise biophysical impact on ALIX recruitment has remained unclear. In this study, we utilized fluorescence anisotropy to quantify the binding dynamics between ALIX and a recombinant Ubiquitin-p6 fusion protein. Our results demonstrate that the presence of ubiquitin increases the binding affinity for ALIX by approximately 3-fold compared to p6 alone. This enhancement is driven by an avidity effect, suggesting that ubiquitin provides a secondary docking site that stabilizes the ALIX–Gag complex. These findings highlight how post-translational modifications can cooperatively regulate host-pathogen interfaces, providing a biochemical basis for how ubiquitination may accelerate HIV-1 release.

Comparative Metabolomic and Molecular Profiles in Methamphetamine-Associated and Idiopathic Pulmonary Arterial Hypertension

Emma Yuguchi
Principal Investigator: Dr. Mona Alotaibi

Methamphetamine-associated pulmonary arterial hypertension (Meth-APAH) is an increasingly recognized cause of pulmonary arterial hypertension (PAH) and is associated with worse outcomes than idiopathic PAH (IPAH). While both forms are characterized by vascular remodeling and plexiform lesions, the metabolomic differences between meth-APAH and IPAH remain underexplored. To examine the metabolomics signatures of meth-APAH and IPAH, patients with meth-APAH and matched IPAH controls were enrolled from the PAH biobank. Plasma samples were analyzed using targeted high throughput metabolomics. Multivariate and regression analyses adjusted for demographics, hemodynamic severity, and PAH therapies were used to identify distinguishing metabolites. Ninety-eight patients were enrolled (49 meth-PAH) and both groups shared similar baseline characteristics, although Meth-APAH patients had higher smoking prevalence and lower PAH therapy use. Out of 1400 metabolites, 119 were significantly ($p<0.05$) different, with 49 significantly increased in meth-APAH. Pathway enrichment analysis identified several pathways enriched in meth-APAH including ascorbate and aldarate metabolism, eicosanoids and arachidonic acid metabolism, pentose pathway, histidine metabolism, and linoleic acid metabolism. In contrast, IPAH patients exhibited significant elevation in metabolites associated with lipid oxidation and fatty acid metabolism, specifically mono and omega-6-poly unsaturated fatty acids and their oxidative intermediates. Meth-APAH is characterized by signatures indicative of elevated oxidative stress, inflammation, and mitochondrial dysfunction, while IPAH shows a profile suggestive of impaired fatty acid oxidation and dysregulated lipid metabolism. These findings reveal metabolic heterogeneity across PAH etiologies and may guide future therapeutic interventions.

Exploring viral mRNA stability-enhancing 3' UTR components for Therapeutic Applications

Alina Zavala, **Sam Landry**
Principal Investigator: Dr. Gene Yeo

Messenger RNA (mRNA) has different functional segments that can be modified to influence translational efficiency and gene expression. Different mRNA molecules have a variety of non-coding or untranslated regions (UTRs) that are not directly synthesized into proteins – nonetheless UTRs play a critical role in mRNA translational initiation, regulation, and overall stability throughout complex cellular processes. The 3'UTR is extremely important for mRNA stability, degradation, localization, and regulatory protein interactions, thereby directly mediating mRNA longevity within protein production. Especially in the context of mRNA gene therapies, this region has been shown to be of great importance to enhance the durability of payload expression. Groups have shown that viral elements used in the 3'UTR can enhance expression of mRNAs, increasing drug potency. RNA base modifications, like N1-methylpseudouridine (m1Ψ), are essential to decrease the immunogenicity of the drug product but can impact the structure and function of the mRNA. Therefore, I aim to understand the functionality of 3' UTR viral regulatory elements in modified mRNAs to improve mRNA therapeutic design.

Cobinamide, A Vitamin B12 Analog, Attenuates Benzo[a]pyrene and Pyrene Toxicity Through Selective Redox Modulation

Tong Zhong, **John Tat**
Principal Investigator: Dr. Gerry Boss

Polycyclic aromatic hydrocarbons (PAHs) are widespread environmental contaminants formed during incomplete combustion of organic material. Their persistence, bioaccumulation, and metabolic activation contribute to mutagenic and cytotoxic effects. Among them, benzo[a]pyrene (B[a]P) is a well-studied benchmark for PAH carcinogenicity, while pyrene is commonly monitored via its urinary metabolite, 1-hydroxypyrene. B[a]P undergoes CYP1A1-mediated oxidation, generating reactive oxygen species (ROS) through epoxide and quinone redox cycling, whereas pyrene primarily produces ROS via pyrene-quinone cycling. We investigated cobinamide, a vitamin B12 analog with strong antioxidant properties, for its protective effects against B[a]P- and pyrene-induced toxicity. In H9C2 rat cardiomyoblasts and A549 human lung epithelial cells exposed to B[a]P (10 μM) or pyrene (10–100 μM), cobinamide (5–10 μM) significantly restored cell viability, normalized JNK/p38 phosphorylation, reduced DNA and protein oxidation, decreased DNA strand breaks, and lowered cleaved caspase-3 activation. At the transcriptional level, cobinamide suppressed inflammatory markers (TNF-α, IL-1β, IL-6) and oxidative stress genes (HMOX1, NOX4), while enhancing antioxidant defense (SOD2) and maintaining xenobiotic metabolism via CYP1A1 induction. In Drosophila melanogaster exposed to 5 mM B[a]P or pyrene, cobinamide (2 mM) improved survival and fully restored locomotion, outperforming cobalamin and N-acetylcysteine. Spectroscopic analysis showed no direct binding between cobinamide and PAHs. Overall, cobinamide effectively mitigates ROS-mediated PAH toxicity through redox modulation while preserving metabolic function, supporting its potential as a therapeutic agent against PAH-induced damage.

Organic Division

Cerqueira, Lana Badan

Gurtler, Jennifer

Kazerani, Parsa

Wu, Stephanie

Yu, Jeffery J.

Functionalized Microtubule-Active Triazolopyrimidines as Possible Therapeutic Agents for Schistosomiasis

Lana Badan Cerqueira, **Darius Yohannan**
Principal Investigator: Dr. Thibault Alle

Microtubules (MTs), which are dynamic, self-rearranging protofilaments of tubulin, form the cytoskeleton of eukaryotic cells, playing a crucial role in the maintenance of cellular integrity, growth, and transport. Due to tubulin's seven distinct binding sites that either degrade, destabilize, and stabilize the MT network, the development of site-specific MT-targeting agents is a promising therapeutic strategy for infections dependent on errant MT dynamics for disease progression. Indeed, MT-stabilization is an effective therapeutic strategy for various indications such as Alzheimer's, cancer, and parasitic infections, notably Schistosomiasis. The latter is a debilitating disease of poverty caused by the parasite Schistosoma mansoni, with only one available treatment, Praziquantel (PZQ). Resistance mechanisms and poor pharmacokinetics of PZQ necessitate the discovery of novel therapeutics. A new class of MT-active agents, known as the triazolopyrimidines (TPDs) have exhibited potent anti-schistosomal activity in vivo. Recently, it was demonstrated that structural changes to TPDs, specifically at C2 and C5 positions, impact the stability of tubulin by targeting of degrading gatorbulin and stabilizing vinblastine sites, respectively. The exploration of MT-destabilizing compounds presents a unique opening for the development of new anti-parasitics. In this work, we synthesized a series of aryl functionalized TDPs to explore the structure-activity relationships (SAR) of the C2 position for its anti-parasitic effects. We show that direct arylation with electron-rich substituents is a viable method to synthesize a variety of compounds for future trials. The exploration of new synthetic pathways through a chemistry-based approach will be a powerful strategy to expand the library of available compounds.

Synthesis and Characterization of Aryl-Functionalized Dipyrrolonaphthyridinedione Annihilators

Jennifer Gurtler, **Lukas Naimovičius**
Principal Investigator: Dr. Andrew Pun

Triplet-triplet annihilation upconversion (TTA-UC) is a photophysical process by which two low-energy photons are converted into a single higher-energy photon. TTA-UC is superior to other upconversion pathways due to its operation under incoherent low-power density light. Owing to its efficiency, TTA-UC has many promising applications, ranging from photovoltaics to bioimaging. TTA-UC systems rely on a sensitizer that absorbs low-energy photons and transfers this energy to an annihilator, which undergoes TTA and emits higher-energy light. Common TTA-UC annihilators, such as acenes and pyrenes, suffer from numerous drawbacks, including difficult synthesis, excimer formation, and poor stability. Dipyrrolonaphthyridinedione (DPND) annihilators are more robust and easier to functionalize, making them a more reliable choice for TTA-UC. For this project, a series of aryl-functionalized DPNDs was synthesized to explore the effects of electron-withdrawing groups (EWGs) and their subsequent stabilization of radical character on DPND TTA-UC performance. Based on the enhanced performance of DPNDs containing nitro and cyano functionalized aryl moieties, we show that stabilization of radical character is essential for efficient TTA-UC. Furthermore, the positioning of EWGs at the para-position of aryl moieties resulted in a high UC quantum yield (UCQY) of 4.5%, an increase from the UCQY of 1.0% observed for those in the meta-position. Therefore, we demonstrate that control of the electronic structure-property relations to optimize DPND annihilators is a promising strategy to enhance their TTA-UC performance, required for practical applications.

Evaluation of Metal Binding Pharmacophore Target Specificity via Photo-Cross-Linking

Parsa Kazerani, **DoYoung Kim**
Principal Investigator: Dr. Seth Cohen

Metalloenzymes are enzymes that contain a metal ion and facilitate many vital biological activities in organisms. They play central roles in processes such as protein degradation, sugar metabolism, and numerous other biochemical pathways. Because of their importance, metalloenzymes are closely linked to disease etiology and are attractive targets for drug development. However, fewer than 5% of all U.S. FDA-approved drugs target metalloenzymes, making them a relatively neglected class in drug design. To address this gap, the laboratory of Prof. Seth Cohen has developed a library of novel metal-binding pharmacophores (MBPs). These molecular fragments contain functional groups that bind to metal ions in the active site of metalloenzymes, thereby inhibiting their activity.

MBPs can be used in Fragment-Based Drug Discovery (FBDD), an approach in which small synthetic fragments are screened against target enzymes. Fragments that demonstrate inhibitory activity are then elaborated into more complex and potent inhibitor candidates.

With an MBP library in hand, it is essential to evaluate binding specificity for target enzymes relative to other cellular proteins. This step is critical for understanding in vivo behavior and identifying potential off-target toxicity. To expand this analysis, the Cohen lab is developing a method that conjugates biotin to MBP analogs for use in streptavidin–biotin pull-down assays. These biotinylated assays enable purification of metalloprotein complexes under mild, non-denaturing conditions, facilitating improved characterization and providing a powerful tool for assessing drug specificity.

<u>One-pot synthesis of 1,1-aminoboranes</u>

***Stephanie Wu*, Leonel Jimenez Barrios**
Principal Investigator: Dr. Erik A Romero

1,1-aminoborones compounds are highly valuable motifs because they unite a nitrogen-containing functionality with a synthetically versatile boron handle in a single scaffold. Their importance is further emphasized by their role in medicinally important boron-containing enzyme inhibitors, including Bortezomib and Ixazomib, and therefore, new methods to access this structural motif have become attractive. Herein, we report a one-pot multicomponent bora-Petasis-type reaction that couples ubiquitous starting aldehydes, secondary amines, bis(pinacolato)diboron (B2Pin2), and N-heterocyclic carbene (NHC), as well as cyclic(alkyl)(amino)carbene (CAAC)-ligated copper catalysts under mild, base-free conditions. This transformation provides direct access to >50 isolable 1,1-aminoboranes with broad functional group compatibility and synthetic utility for downstream derivatization. Additionally, this strategy facilitates the efficient synthesis of the aminoboronic acid drug, Bortezomib, an FDA-approved treatment for multiple myeloma and lymphoma. Mechanistic insights support that this transformation does not proceed via a Cu-boryl intermediate, indicating an alternative pathway that adheres more towards a Cu-boronate/iminium ion-pair reminiscent of Petasis-type chemistry to forge the carbon-boron bond.

Overall, this work demonstrates an efficient synthetic approach to synthesizing 1,1-aminoboranes with commercially available starting material, low catalyst loading, and mild reaction conditions.

Two Methods for Radical C–C Bond Formation: Electrochemical Coupling and Hydrazide Chemistry

Jeffery J. Yu, **Yin Li, Jiayan He, Yu Kawamata**
Principal Investigator: Dr. Phil S. Baran

This project focuses on the selective functionalization of saturated nitrogen-containing heterocycles using radical cross-coupling methods. In this poster, two methods used in the project are introduced: electrochemical decarboxylative coupling based on redox-active esters (RAEs) and sulfonyl hydrazide-based radical cross-coupling.

The electrochemical method uses carboxylic acids as radical precursors and provides a greener, efficient, and controllable way to form $C(sp^3)$–$C(sp^2)$ bonds. The hydrazide method can generate radicals from a wider range of functional groups, including alcohols, amines, and carbonyl compounds, and offers a practical redox-neutral approach to C–C bond formation. More recent work has also shown that this chemistry can retain stereochemistry during coupling, making the synthesis of cis-substituted and enantioenriched products more efficient.

The goal of this poster is to introduce these two methods through their reaction design, mechanisms, and representative examples from the literature. It will also include a few examples from the author's internship work to show how these methods can be used for the selective modification of heterocyclic scaffolds.

Inorganic Division

Cho, Seokhyeong

He, Chloe

Martin, Danny

Truong, Ethan P.

Electrocrystallization of Lanthanide-Polyoxometalate Coordination Networks

Seokhyeong Cho, **Kody Acosta**
Principal Investigator: Dr. Alina Schimpf

Polyoxometalates (POMs) are a promising class of inorganic clusters for constructing functional metal-oxide frameworks, owing to their multi-electron redox activity, tunable electronic structure, and high chemical and thermal stability. The Preyssler-type cluster [NaP5W30O110]14- (denoted {P5W30}) is particularly attractive as a building block, as it exhibits stability over a wide pH range, ability to exchange the central cation and dope heterogeneous atoms (V, Mo) to create emergent properties in the framework. Here, we report electroreduction-driven self-assembly of the anionic Preyssler cluster {P5W30} with lanthanide cations (La^{3+}–Yb^{3+}, Y^{3+}) as bridging nodes. Electrochemical reduction of the LUMO triggers cluster assembly, yielding crystalline lanthanide-POM structures ranging from discrete units to one-dimensional chains and two-dimensional networks, depending on the lanthanide identity and synthetic conditions (solvent: HOAc, LiOAc, KOAc; counterion of Ln; cluster type). We hypothesize that the degree of networking is further governed by lanthanide ion concentration, which controls the balance between framework extension (networking) and cluster surface decoration. These results establish electroreduction as a tunable strategy for directing the self-assembly of inorganic cluster-based frameworks and provide rational design guidelines for lanthanide–POM frameworks.

Basal plane functionalization of Tungsten Diselenide by phosphine ligands through interlayer separation

Chloe He, **Sourajit Dey Baksi, Shan Yu**
Principal Investigator: Dr. Alina Schimpf

Covalent functionalization is a key strategy for tuning the electronic properties of group VI transition metal dichalcogenides (TMDs) by modulating interlayer spacing. However, it remains challenging due to their layered "sandwich" structure, in which metal atoms are shielded by chalcogenide layers, leaving the surface chemically unreactive. Conventional approaches typically rely on defects or edge sites. In this work, we present a strategy for basal plane functionalization of tungsten diselenide (WSe2) nanocrystal via phosphine ligand incorporation. A colloidal synthesis approach is employed to promote self-assembly while minimizing defect formation and structural disruption. Tri-octyl phosphine (TOP) is used as a model ligand, followed by a series of phosphine ligands (e.g. Tricyclohexylphosphine, Tri(1-adamantyl)phosphine, Tri-n-butylphosphine) with varying steric and electronics properties to systematically investigate their effects. Powder X-ray diffraction analysis reveals significant interlayer expansion in ligand incorporated samples compared to pristine WSe2, suggesting successful layer expansion. Further characterization using X-ray photoelectron spectroscopy (XPS) and infrared (IR) spectroscopy will be done to confirm successful surface functionalization with phosphine ligands.

Tuning the Redox Entropy of Highly Charged Polyoxometalates for Thermogalvanic Cells

Danny Martin, **Thompson Marinho**
Principal Investigator: Dr. Alina Schimpf

The majority of global waste heat is low grade heat (under 100C), as a result of natural/industrial processes. A promising strategy to collect and reuse this heat is by using thermogalvanic cells, which generate electrical potential from an established temperature gradient. The efficiency of a thermocell depends on the changes in redox entropy (ΔSr), which is proportional to the Seebeck coefficient (Se). The Born model of solvation shows that ΔSr is directly proportional to the difference in squared charges between the oxidized and reduced species (Zox2 – Zred2).

This work studied the electrochemical properties of Keggin polyoxometalates (POM) ([XW12O40]^n-) as tunable, redox-active electrolytes for thermocells. POMs with various central heteroatoms were synthesized and analyzed with FT-IR, NMR spectroscopy, and cyclic voltammetry to confirm identity and purity. Two important techniques applied were variable-temperature cyclic voltammetry (VT-CV) and square wave voltammetry (SWV), which quantitatively measured the redox behavior of the POMs in solution for calculating Se.

Se values reached up to -1.89 mV/K, which corresponds to a large redox entropy. The first two POM redox events generally showed linear behavior, in accordance to the Born model. However, higher reduction events showed significant deviations from Born linearity. Pourbaix analysis displayed that the redox events underwent PCET (proton-coupled electron transfer).

The findings of this experiment show the importance of protons in understanding thermocells, as well as the importance of the structure, and provide new insight on designing thermogalvanic cells.

Closed-loop Optimization of Metallo-Organic Framework Synthesis and Solvent Selection Parameters

Ethan P. Truong, **Madison Esposito**
Principal Investigator: Dr. Seth Cohen

Metal–organic frameworks (MOFs) are crystalline porous materials built from metal nodes connected by organic linkers, with applications in gas storage, catalysis, drug delivery, and chemical sensing. Low-Valent MOFs (LVMOFs) are constructed from electron-rich metal centers in the 0 or +1 oxidation state. LVMOFs enable substrate activation and organometallic redox catalysis within a porous scaffold, but their synthesis is sensitive. Because the synthesis space is combinatorially large and each synthesis requires a week to run, trial-and-error exploration is slow and prone to bias. We present a machine-learning surrogate, trained on 755 prior LVMOF syntheses, and Bayesian Optimization (BO) framework that informs experimental design

Each reaction is represented by a high-dimensional matrix assembled by descriptors that capture physical, chemical, and electronic properties of metal precursor, organic linker, solvent, and modulator. The success of each experiment, indicated by Powder X-ray Diffraction (PXRD), is retrospectively labeled from 0-9, 0 indicating formation of no solid and 9 a highly crystalline material. A surrogate was fit on this data and shown to accurately rank crystalline candidates above amorphous ones. We embedded this surrogate into a BO loop that proposes novel experimental conditions, by ranking predicted crystallinity of hypothetical parameters, balancing exploitation against exploration. New experimental data is fed back to the surrogate, continuing the cycle. Retrospective testing demonstrated a 3x improvement in the identification of successful MOF synthesis. Using this framework, we have successfully synthesized 3 novel gold-based crystalline products, with full structural characterization currently forthcoming.

Physical/Analytical Division

Ayers, Margaret

Chen, Zhenkun

Dandin, Aarushi

Heyang, Haoye

Lu, Yoyo

Majeed, Dawoud David

Shane, Amanda

Ball Milling Enhances Ionic Conductivity in a Lithium Closo-hydroborate Solid Electrolyte

Margaret Ayers, **Marta Vicencio, Alex Liu, Julien L'Her, JinKwan Jung**
Principal Investigator: Dr. Shirley Meng

As global energy usage continues to rise, efficient energy storage is essential to ensure that demand is met. All-solid-state batteries (ASSBs) have emerged as a promising candidate, offering improved safety and potentially higher energy density compared to commercial lithium ion batteries. ASSBs contain a solid electrolyte (SE) that serves as a barrier between the electrodes, preventing short circuit and mediating ion transport between anode and cathode. The electrochemical and mechanical behavior of SEs can critically influence battery performance: an effective SE must exhibit high ionic conductivity, stability with both electrodes, and scalable synthesis. Lithium closo-hydroborates have attracted interest due to their electrochemical stability and enhanced ionic conductivity in high-temperature disordered phases.

This work investigates the effects of high-energy ball-milling on the ionic transport mechanism in a SE mixture of dilithium decahydrodecaborate and dilithium dodecahydrododecaborate (LBH). Ball milling is a mechanochemical technique that involves bombardment of solid materials with blending media to induce chemical reactions such as phase transitions and amorphization. The LBH materials were synthesized in an equimolar ratio, and samples collected at different milling times were characterized to evaluate the effects of milling on ionic conductivity and crystal structure.

These findings indicate that longer milling times induce substitution of the $B_{10}H_{10}^{2-}$ anions into the $B_{12}H_{12}^{2-}$ lattice, leading to increased disorder and high ionic conductivity (1.6 mS/cm). This work shows our milling parameters yielding higher conductivity than previously reported in the literature, making LBH a strong candidate for next generation ASSBs.

Data-Driven Many-Body Framework for Azide Ion in Water

***Zhenkun Chen*, Jiwon Huh**
Principal Investigator: Dr. Francesco Paesani

The distribution of ions at aqueous interfaces, especially at the air/water boundary, remains an unsettled problem with key implications for environmental heterogeneous chemistry. Data-driven many-body energy(MB-nrg) potential energy functions provide predictive molecular models with quantum mechanical accuracy, enabling the study of molecular structure and dynamics across phases. These approaches bridge the gap between high-level electronic structure methods and large-scale simulations.

In this work, we develop an MB-nrg model for the azide ion(N_3^-) in aqueous environments to describe its solvation structure and interactions. A one-body(1B) potential is trained on high-level ab initio energies at the CCSD(T) level and is shown to accurately reproduce the corresponding normal mode frequencies of N_3^-. A central focus is the construction of a representative two-body (2B) training set. Enhanced sampling using PBMetaD in LAMMPS/PLUMED is employed to generate diverse configurations, including high-energy interaction regions. These structures are then represented using many-body tensor representation (MBTR) descriptors and refined through farthest point sampling (FPS) to ensure diversity and reduce redundancy, after which quantum chemical calculations are performed to obtain interaction and binding energies for fitting the 2B potential.

Model validation is performed through structural analysis, including radial distribution functions (RDFs), demonstrating that key features of azide–water interactions are captured. Remaining higher-body effects are examined through many-body decomposition of large clusters, and ongoing work focuses on the development of an explicit three-body (3B) potential to further improve the accuracy.

Molecular-Level Insights into Antifreeze Protein–Ice Interactions from Molecular Simulations

***Aarushi Dandin*, Suman Saha**
Principal Investigator: Dr. Francesco Paesani

Recent machine-learning based design approaches have been able to generate stabilized antifreeze protein (AFP) variants with new scaffolds and tunable properties. The central bottleneck, however, remains screening: each candidate must still be expressed and characterized experimentally before its function is known. We aim to address this problem by developing a computational framework that can rapidly evaluate whether a designed AFP preserves a functional ice-binding surface before wet-lab testing. The guiding hypothesis is that variants that preserve the geometry and chemistry of a productive ice-binding interface, including surface flatness, residue patterning, and ordered threonine-rich motifs, will preferentially stabilize ice-like water near that surface. To test this we combined all-atom molecular dynamics simulations with enhanced sampling. Initially, we benchmarked the approach on a well-characterized control, the Tenebrio molitor antifreeze protein (tmAFP), whose beta-solenoid scaffold presents a validated ice-binding face. After representing the protein with the ff14SB force field and solvating it in water represented by the TIP4P/Ice model, AMBER with PLUMED was then used to bias interfacial water toward ice-like order using the Q6 bond-orientational order parameter. Umbrella sampling and well-tempered metadynamics were then applied to quantify whether ice-like clusters form preferentially at the known binding surface and to estimate the free-energy cost of interfacial ordering. The outcome of this research will be a rapid pre-screen for AFP design to accelerate the development of AFPs for technologies that depend on precise control of ice formation.

Analysis of Excited-State Proton Transfer Dynamics of 2-(2′- pyridyl)benzimidazole in Acidic Solution Using Transient Absorption Spectroscopy

***Haoye Heyang*, Nicole Muir**
Principal Investigator: Haiwang Yong

Excited-state proton transfer (ESPT) protects biomolecules and functional materials from photodamage and occurs in protic environments for the photobase 2-(2′-pyridyl)benzimidazole (PBI). Previous studies investigated PBI in methanol using ultrafast transient absorption spectroscopy and in acidic solution using ultrafast fluorescence spectroscopy. While the methanol system has been studied in detail, transient absorption data of PBI in acidic solution have been collected but not yet analyzed. This project will analyze TAS data of PBI in acidic solution to determine ESPT dynamics.

Investigating Li^+ Transport Mechanisms in Anionic MOF Quasi-Solid-State Electrolytes via Molecular Dynamics

Yoyo Lu, **Suman Saha**
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Developing safer, high-conductivity electrolytes is critical for next-generation batteries, and anionic metal–organic frameworks (MOFs) are promising quasi-solid-state electrolytes. However, the molecular mechanisms controlling cation mobility under confinement remain poorly understood, limiting rational materials design. In this work, molecular dynamics (MD) simulations were employed to investigate Li^+ transport in the anionic MOF SU-102.

We hypothesize that under nanoconfinement, Li^+ transport lies between two limiting regimes: vehicular transport, where ions move with their solvation shells, and cooperative, network-assisted ("Grotthuss-like") transport. These regimes are expected to be influenced by framework charge distribution, binding-site topology, and the confined hydrogen-bond network.

Simulations were performed using LAMMPS under isothermal–isobaric (NPT) conditions to characterize the structural and dynamical behavior of Li^+ in confined propylene carbonate (PC). Radial distribution function and coordination analyses show that the MOF environment significantly alters Li^+ coordination. Strong competition between framework binding and solvent solvation prevents full solvation-shell formation, while pronounced interactions between Li^+ and framework oxygen atoms highlight the key role of framework charge in ion binding.

For dynamical characterization, microcanonical (NVE) simulations were used to compute mean squared displacement and extract diffusion coefficients. Preliminary results indicate that confinement initially suppresses diffusion, while model refinements that better capture framework–solvent interactions lead to improved descriptions of ion mobility. Ongoing work focuses on improving model accuracy and applying enhanced sampling methods (e.g., metadynamics via PLUMED) to map the free energy landscape of ion migration.

Soil mobility of virus-based nanoparticles for delivery of agrochemicals in Climate-Stressed Soil

Dawoud David Majeed, **Sean McDowell, Patrick Smith**
Principal Investigator: Dr. Ivonne González-Gamboa

Globally, 750 million people face hunger in 2023, with climate stressors threatening to worsen crop yields and food quality. In addition, human activities have led to increasing rates of soil salinization, pollution, and wildfires. To address food security challenges, we need efficient delivery systems that can help plants become more resilient to these abiotic stressors. Plant viral nanoparticles offer a promising approach for delivering agricultural compounds to crops. However, most studies use commercial potting soil rather than real-world stressed soils. We investigated whether TMGMV-based delivery systems maintain effectiveness in climate-stressed soils to ensure this technology can protect crops under actual field conditions. We used soil columns to emulate field conditions, tested the mobility of wild-type TMGMV across three soil types: a potting soil control and two climate-stressed samples, high saline and heavy metal-contaminated, sourced from the UC Natural Reserve System. We analyzed mobility and persistence via SDS-PAGE and protein quantification to determine delivery efficiency. Potting soil, had good mobility; good persistence with a profile that slowly tapers off. High saline soil showed good mobility but lower persistence than potting soil. Heavy metal-contaminated soil demonstrated good mobility with a brief peak and low persistence. Stressed soils showed good mobility for TMGMV-based particles, though with lower persistence than potting soil controls. This demonstrates that viral nanoparticle delivery systems can function effectively in real-world stressed soils, providing a viable pathway for enhancing crop resilience and food security under changing climate conditions.

Stratospheric Aerosol Injection Uptake Kinetics

Amanda Shane, **Wen Zhang**
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Stratospheric aerosol injections (SAI) offer a potential response to increasing global temperatures. Particles with high reflectivity would be injected into the stratosphere and scatter solar radiation away from the Earth, thereby lowering Earth's temperature. It is first necessary to understand what gases SAI would react with, how long they would remain in the stratosphere, and how they would degrade under extreme temperatures. This research is focused on determining the oxidation kinetics of potential SAI particles with gases characteristic of the stratosphere.

To measure this, SAI particles are aerosolized using a fluidized bed aerosol generator. Derivatives of silicon dioxide particles with varying amounts of hydroxyl ligands are used as potential SAI particles. Altering the number of hydroxyl ligands changes the particles' ability to retain water and has implications on the formation of cloud condensation nuclei. The SAI particles are then reacted with gases commonly found in the stratosphere such as ozone, nitrogen dioxide, and nitrogen monoxide. Real time measurements of reaction products are taken using a chemical ionization time of flight mass spectrometer, with sulfur hexafluoride as a reagent ion.

Measurements on the uptake coefficient of ozone onto SAI particles and benchmark materials including oleic acid and ammonium sulfate have been completed. In continuing studies, these reactions will be carried out at low temperatures to understand the temperature dependence of the reaction kinetics.

As the climate crisis worsens, it is essential to clarify the effects that SAI would have on the ozone layer, cloud formation, and formation of potential byproducts.

Chemical Education

Smeenk, Corey

Matter of Scale: Gamifying Size and Scale

***Corey Smeenk*, Sean McDowell**
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Students often struggle to conceptualize and contextualize Size and Scale (S&S), particularly at more extreme scales. A strong understanding of S&S is a critical, cross-cutting skill necessary for success in materials science, biology, chemistry, and advanced STEM careers. Conceptions of size and scale are often grouped into a framework with two board categories, qualitative and quantitative conceptions, and then further breakdown into concepts like categorical, proportional, relational, and absolute conceptions. Several critical conceptions of S&S have been identified, including skills and cognitive processes such as proportional thinking, viewing matter as on a continuum, and comprehension of scientific notation. To address misconceptions and promote engagement with S&S concepts, we developed the card game, "Matter of Scale." The primary research goal was to determine whether this informal educational activity could help undergraduate STEM students reduce misunderstandings about S&S and improve their ability and confidence to navigate S&S conceptions.

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