Chemistry & Biochemistry Poster Fair Presenters Abstracts

Thursday, April 30, 2026
1:00pm – 3:30pm
Bear Down Gym

THE UNIVERSITY OF ARIZONA
COLLEGE OF SCIENCE
COLLEGE OF MEDICINE TUCSON
Chemistry & Biochemistry

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2026 CBC Senior Thesis/Capstone Presentations

Emma Avouris – Biochemistry & Physiology and Medical Science #44

Research Faculty Advisor: Bryan Wong, Speech Language and Hearing Sciences

Fitting the Future of Hearing Healthcare: Comfort Measures of 3D-Printed Earmolds

Background: Hearing loss prevalence continues to grow, yet there are still many barriers to receiving care. Some of these include high cost and geographic barriers. Using innovative technology to address these barriers is critical in addressing the rising levels of hearing loss. 3D printing technologies have been historically utilized by third-party manufacturers, but with recent improvements, these technologies are now commercially available for clinicians. Implementation of 3D printing has been well studied in dental and prosthetic fields, but hasn't reached the same level of utilization in audiology. Purpose: Our study aims to assess the comfort of in-house 3D printed earmolds in elastic and resin compared to a professional third-party manufactured earmold, which served as the gold standard control. Study design: Within subjects, counter-balanced, comparative effectiveness study

Methods: A modified version of the Effectiveness of Aural Rehabilitation- Outer (EAR-Outer) questionnaire was used to compare naturalness, quality of voice, physical comfort, acoustic comfort, and global score across each condition. Results: Overall, our study found no significant difference between in-house and third-party manufactured earmolds across global comfort scores and individual items. Conclusion: These findings suggest that the overall comfort of in-house 3D printed models may be comparable to the third-party manufacturer. The results found in this study support the potential application of an in-house 3D printed model.

Cole Bellomo – Chemistry #10

Research Faculty Advisor: Jeffrey Pyun, Chemistry and Biochemistry

Introduction of Molybdenum Disulfide in Sulfur Copolymers as Composite Material with Increased Mechanical Strength

Molybdenum disulfide (MoS2) is a transition metal chalcogenide obtained as a mining byproduct. It's mainly used as a solid lubricant for industrial purposes, but research discussing its ability to form composites in polymeric systems has been lacking. Here, MoS2 was dispersed in a liquid sulfur environment and polymerized through inverse vulcanization to study mechanical performance. The composite polymers and polymers without MoS2 were melted and casted into three-point bend molds. The materials were then evaluated with ASTM flexural testing, showing incorporation of MoS2 resulted in increased flexural strength. These findings demonstrate the ability to tune the properties of sulfur-based polymers via addition of transition metal chalcogenides.

Kelsey Buffington – Biochemistry #29

Research Faculty Advisor: James Galligan, Pharmacology and Toxicology

Analysis and Quantification of Histone Tiglylation and Histone Post-Translational Modification in HEK 293 Cells from a Mouse Model of ECHS1 Deficiency

Post-translational modifications (PTMs), also known as epigenetic marks, on histone proteins are highly regulated through enzymes and co-substrates in order to allow for proper cellular functioning throughout all living organisms. Within this study, we have been able to quantitatively discover a new PTM mark on the lysine residues of histone proteins generated from tiglyl-CoA, an intermediate that plays an integral role within isoleucine metabolism. In the isoleucine metabolism process, tiglyl-CoA acts as a substrate for the enzyme ECHS1. Deficiency of this enzyme contributes to Leigh syndrome, a neurometabolic pediatric disease, thus indicating the value this research can have for those with this incurable disease and its possible connection to this epigenetic mark on histone proteins. Various assays and techniques were undertaken throughout this experiment to accomplish the quantification of this PTM. Three cell lines of human embryonic kidney, HEK 293, cells were

cultured including Wildtype, ACADSB knockout, and ECHS1 knockout to accurately measure the generation of tiglyl-CoA using liquid chromatography-mass spectrometry (LC-MS), indicative of this histone mark. In-solution digestion of histone proteins via propionylation was a precursor for the LC-MS method in order to increase histone length through the blockage of unmodified lysine residues that ultimately prevents trypsin from cutting at certain sites. This was integral for accurate LC-MS quantification. Through this, the experiment demonstrated the use of biochemical techniques to quantify and analyze histone tiglylation and the new PTM on lysine residues on histone proteins allowing for future directions to be taken within research in hopes of making strides towards a cure for Leigh syndrome.

Anna Campbell – Biochemistry #13

Research Faculty Advisor: Matthew Cordes & John Jewett, Chemistry and Biochemistry

Investigation into the autoinhibitory motif lost in recluse spider phospholipase D venom evolution using a customizable peptide mimic

Phospholipase D enzymes are a key recluse spider venom toxins that induce paralysis in insect prey and necrosis in envenomated humans. These toxins evolved from a family of endogenous phospholipase Ds that are ubiquitous in arachnids. This venom recruitment step is associated with the loss of a C-terminal tail motif that is present in the most closely related non-venom expressed phospholipase Ds. Replicating venom recruitment associated tail truncation in a Loxosceles rufescens non-venom expressed phospholipase D enhances sphingomyelinase activity. Conversely, introducing the tail motif into a reconstructed venom common ancestor lowers sphingomyelinase activity. Thus, the tail appears to be autoinhibitory, and its removal could therefore have been a key activation step in its evolution into a venom toxin. Based on an AlphaFold structural prediction, we suspect the autoinhibition may result from the tail blocking a recently discovered allosteric site. Using a customizable peptide analog of the tail terminus, we show that the tail's final 7 residues are able to bind the proteins core PLD domain and recreate the inhibitory activity. These results bring us closer to understanding the tails inhibitory mechanism, which could facilitate both inhibitor design and development of a model for the biological regulation of the non-venom proteins

Branham Carpenter – Biochemistry & Molecular and Cellular Biology #23

Research Faculty Advisor: George Sutphin, Molecular & Cellular Biology

The Effects of Short-Term Fasting and Tamoxifen on FOXO Transcription Factors

Nutrient availability plays a critical role in determining cancer cell survival and therapeutic response. Although cancer cells are known for their ability to tolerate metabolic stress, short-term nutrient deprivation can paradoxically increase their sensitivity to chemotherapy, a phenomenon known as differential stress resistance (DSR). This response is largely regulated by the FOXO family of transcription factors, which act as critical molecular components of nutrient-sensing pathways and cellular stress signals. In this study, we investigated how serum starvation influences cancer cell responses to tamoxifen and examined the role of FOXO transcription factors in mediating this effect. Using MCF-7 human breast cancer cells, we found that serum restriction significantly enhanced sensitivity to 4-hydroxytamoxifen (4-OHT), as measured by crystal violet viability assays. This increased sensitivity was associated with elevated intracellular reactive oxygen species (ROS), as detected by H_2DCFDA fluorescence, and increased double-stranded DNA breaks, as indicated by γH2AX staining. Combined serum starvation and tamoxifen treatment produced greater oxidative stress and DNA damage than either condition alone. Immunofluorescence analysis demonstrated that nutrient deprivation and tamoxifen promoted nuclear localization of FOXO transcription factors, indicating their activation under stress conditions. Importantly, genetic knockout of FOXO1 partially rescued cell survival during combined starvation and tamoxifen treatment, demonstrating that FOXO1 is required for full starvation-induced drug sensitization. Overall, our findings support a model in which nutrient deprivation enhances therapeutic sensitivity by increasing oxidative stress and DNA damage in a FOXO-dependent manner. These results identify FOXO1 as a key regulator linking metabolic stress to treatment response and suggest that targeting metabolic FOXO signaling pathways may represent a potential strategy for improving cancer therapy outcomes.

Elizabeth Cashwell – Chemistry minor Biochemistry #09

Research Faculty Advisor: Jon Njardarson, Chemistry & Biochemistry

Development of a Novel and Stereoselective Route to Nitrogen Heterocycles Via Versatile δ-lactam Scaffolds

Nitrogen heterocycles are ubiquitous motifs in FDA-approved pharmaceuticals due to their versatile biological and physicochemical properties. Research within the Njardarson group has demonstrated that chiral imines, in the presence of a γ-nitro-substituted dienolate, enable the formation of chiral piperidine precursors through a lithium-assisted cyclization. An optimized method has been developed that selectively produces a δ-lactam and conjugated variants with up to 8:1 stereoselectivity. This core provides a divergent platform where chiral piperidines, pyridines, and carbazoles have been synthesized.

Lainey Caswell – Biochemistry, Vet Sciences #38

Research Faculty Advisor: Evan MacLean, College of Veterinary Medicine

Effects of Visual Dimensionality on Canine Social Cognition and Decision-Making

Dogs exhibit heightened sensitivity to human social cues, such as pointing gestures, which involve the integration of visual and communicative information to guide behavior. While prior studies have largely relied on two-dimensional (2D) stimuli, real-world interactions occur within dynamic three-dimensional (3D) environments that may engage different sensory and neural processing pathways. This study investigated how stimulus dimensionality influences visual attention and cue processing in domestic dogs.

Using head-mounted eye-tracking technology as a proxy for visual information processing, gaze behavior was recorded from ten domestic dogs during a two-choice object selection task presented in both 2D and 3D conditions. Fixation duration and fixation count across predefined Areas of Interest were used to assess visual processing patterns, while behavioral performance was measured based on choice accuracy.

Dogs demonstrated significantly higher accuracy in following communicative cues in the 3D condition compared to the 2D condition, suggesting enhanced integration of visual-social information in live contexts. However, fixation patterns across Areas of Interest did not significantly differ between conditions, indicating that visual sampling of stimuli remained consistent. Across both conditions, attention to relevant communicative cues was associated with improved performance, suggesting a link between visual attention and downstream decision-making processes.

These findings suggest that while the initial stages of visual attention may be conserved across stimulus formats, 3D environments may facilitate more effective sensory integration and processing of social cues. This work highlights the importance of ecological validity in studying perceptual and cognitive processing and provides insight into how stimulus dimensionality influences information processing in a biological system.

Diezel Cochenour – Biochemistry #49

Research Faculty Advisor: Ray Runyon, Environmental Science

Profiling anti-inflammatory molecules in non-alcoholic beer using liquid chromatography mass spectrometry

Nonalcoholic beer has gained attention as a functional beverage because it retains hop-derived bioactive compounds without the harmful physiological effects of ethanol. This study aimed to identify and quantify nutritionally relevant prenylated flavonoids from Humulus lupulus, with emphasis on xanthohumol, isoxanthohumol, and 8-prenylnaringenin. Hops pellet samples were extracted using methanol to isolate target compounds from the plant material. Extracts and beer samples were separated by liquid chromatography and analyzed by tandem mass spectrometry (LC–MS/MS), where compound identity was confirmed by characterized precursor-to-product ion transitions. Calibration curves generated from external standards (1–1000 ppb) were used for quantitative analysis. All three target compounds were successfully detected in hops samples and beer

samples using distinct retention times and fragmentation patterns. Quantitative analysis allowed calculation of compound concentrations within the samples. These findings demonstrate that prenylated flavonoids can be reliably detected and quantified in hops plant material and beer samples, suggesting that hops plants and products could be screened for relative secondary metabolite concentrations.

Tanner Cohrs – Biochemistry #08

Research Faculty Advisor: M. Leandro Heien, Chemistry and Biochemistry

Development of Iridium Oxide as a Novel Reference Electrode Material for In Vivo Electrochemistry

The ability to monitor neurotransmitter levels in the brain chronically is critical for continued characterization of existing neurodegenerative diseases. The main way to do this however, using fast scan cyclic voltammetry (FSCV), presents issues in its efficacy over the long term, with biofouling of the reference electrode causing inaccuracies in the applied waveform, leading to difficulty identifying analytes, and diminished sensitivity. The standard Ag/AgCl-wire reference electrode used for in vivo FSCV is cytotoxic, which causes an exacerbated immune response and prohibits its use in humans. Thus, there exists a need for a biocompatible reference electrode that does not induce a strong immune response and can

be evaluated for eventual human FSCV use. Here, we fabricate and investigate an iridium oxide (IrOx) reference electrode, evaluating its attractive characteristics in vitro, with its stable potential profile, comparable sensitivity to dopamine to that of the Ag/AgCl-wire reference, and the chemical composition of its surface which provides these qualities. These metrics combined with the biocompatibility of IrOx makes it intriguing for use in chronic FSCV in vivo experimentation in animal models and potentially even humans. Further work will reveal whether the feasibility of IrOx as a biocompatible reference electrode exhibited herein will transfer to chronic experimentation.

Michael Davis – Biochemistry #45

Research Faculty Advisor: Lee Ryan, Psychology

Perceived Stress and Episodic Memory: Investigating The Moderating Effect of Physical Activity among Hispanic and non-Hispanic White middle aged to older adults

Objective: The present study examined the relationship between perceived stress and episodic memory performance, as well as the moderating role of physical activity (PA), with a focus on potential differences between Hispanic Latino and non-Hispanic White (NHW) adults.

Methods: Participants included 314 middle-aged to older adults (241 NHW, 73 Hispanic Latino) from the Precision Aging Network. Perceived stress was assessed using the Perceived Stress Scale (PSS), physical activity using the Quick Physical Activity Rating (QPAR), and episodic memory using an online paired associates learning (PAL) task. Multiple linear regression models tested main and interaction effects of perceived stress and PA on PAL performance. Propensity score matching was conducted to control demographic differences between groups.

Results: Perceived stress and physical activity were negatively correlated; however, neither variable was significantly associated with episodic memory performance. Physical activity did not moderate the relationship between perceived stress and PAL performance, and this effect did not differ by ethnicity. In the full sample, Hispanic Latino participants reported higher perceived stress and lower physical activity than NHW participants, though these differences were no longer significant after matching. Education emerged as the strongest predictor of memory performance.

Discussion: Our findings do not support an association between perceived stress and episodic memory or any moderating role of physical activity in this relationship. Results suggest that demographic factors, particularly education, may play a more prominent role in cognitive outcomes than psychosocial or lifestyle variables in this sample. Future research should employ longitudinal designs and more precise measures of stress and physical activity to better understand these relationships across diverse populations.

Adrian De la Peña – Biochemistry #05

Research Faculty Advisor: Minying Cai, Chemistry and Biochemistry

MC1R LEAD OPTIMIZATION THROUGH PROTEIN COEVOLUTION AND BIOPHYSICAL ASSAY PIPELINE

Understanding GPCR activation is crucial for many physiological pathways. The complexity of not only pharmacological intervention at GPCRs, but also downstream effect specificity, makes GPCRs a promising but convoluted target for disease management. To help reveal functional motifs and "hotspots" in the melanocortin-1 receptor (MC1R), coevolutionary analysis of receptor orthologs was used. Coevolution finds residues that are pressured to mutate together over the course of evolution; these links have previously been found to indicate intra- or interprotein contacts. Combined with MMGBSA molecular docking, three experimental ligands (Melanotan-II, SHU9119, MSG606) were functionally analyzed. Coevolution models found links between residues in the binding pocket and the transducer interface of hMC1R and Gs-Protein/β-Arrestin1/β-Arrestin2. Docked and crystallized MC1R ligands exhibited interactions with orthosteric site residues that informed possible mechanisms of biased pathway choice by these functionally covariating residues. Experimental validation of biased agonism came from an optical assay for isolating binding events in real-time: plasmon waveguide resonance spectroscopy. This SPR-like assay can view real-time mass density changes in receptor-embedded membrane systems that isolate transducer affinity differences across experimental MC1R ligands. This combined structure-activity relationship workflow has shown promise in unraveling the mechanism behind transducer bias at MC1R using receptor evolution. It is hoped that this can be universally applied as a novel approach to rationale-based drug design at any protein receptor.

Chaz DeCoteau – Biochemistry #36

Research Faculty Advisor: Koenraad van Doorslaer, Immunobiology

Using Co-Immunoprecipitation to Investigate the Potential for SNX1.3 to Disrupt the Interaction Between p120 Catenin and HPV L1

Human papillomavirus (HPV) infections are responsible for 99% of cervical cancers and 80% of mouth and throat cancers in the U.S. With a vaccine that only covers half of the high-risk strains, and that is too expensive for low-income regions of the world to fully access, low vaccination rates persist, increasing the prevalence of HPV-related cancers from strains such as HPV16. In this project, we aim to see if the SNX1.3 peptide, known to inhibit HPV infection, disrupts the interaction between the HPV L1 protein and p120 catenin, which initiates the process for HPV to guide itself to the nucleus to establish infection. With co-immunoprecipitation, we use antibodies to isolate p120 in complex with L1 in HaCaT cells infected in the presence of SNX1.3. Immunoprecipitates of p120 isolated minimal amounts of the protein, with a majority of the protein remaining unbound. This has made it difficult to identify p120. Some adjustments, such as a change in incubation durations and temperatures, have minimally improved results. Use of larger concentrations of antibodies and protein have also been unsuccessful. Going forward, changes focusing on the quality of the antibodies used and the delicacy of reagents used may produce more productive results. If a successful version of this experiment can be designed, it will allow us to better visualize the HPV L1-p120 complex, and thus how this interaction is affected by the presence of SNX1.3.

Kylie Ernst – Biochemistry #24

Research Faculty Advisor: Andrew Paek, Molecular and Cellular Biology

Many Proteins Activated by H2O2 Stress Require Iron for Activation

Under normal conditions, reactive oxygen species (ROS) are important signaling molecules used by cells. However, ROS imbalances can oxidize proteins, lipids, and DNA, causing the cell to go into oxidative stress. This damage can ultimately result in cell death and is a driver of disease. Hydrogen peroxide (H2O2) is a common ROS that is nontoxic at low concentrations, coordinating processes such as proliferation, survival, and metabolism; meanwhile high concentrations cause toxic oxidation and activate a different group of proteins that signal damage and induce apoptosis. The precise mechanisms that differentiate these responses are not yet known, but

we hypothesized that it is related to the Fenton reaction, where hydrogen peroxide (H_2O_2) in the presence of iron is converted into highly reactive hydroxyl radicals (•OH). Here we show that iron is required to activate or inactivate several proteins (AKT, FOXO1, CHK2, p53, eIF2α, and S6) in response to H_2O_2-induced oxidative stress in breast cancer cells. We found that in the absence of iron, proteins that are typically only active at low doses of H_2O_2 remain active for higher concentrations. In contrast, proteins activated by high doses of H_2O_2 were inactive in the absence of iron, suggesting their activation is due to oxidation by •OH instead of H_2O_2 itself. Our results demonstrate that many of the dose-dependent effects of H_2O_2 are also Fenton-dependent. By elucidating the molecular mechanisms of eustress/distress signaling, we anticipate this research will provide a greater understanding of conditions associated with increased oxidative stress.

Luke Fasse – Chemistry #01

Research Faculty Advisor: Thomas Gianetti, Chemistry and Biochemistry

Out of the Blue: A Low-Energy Photooxidant for Furan-to-Pyrrole Skeletal Editing and Aromatic C-H Lamination

Photoredox catalysis is a powerful tool for modern organic synthesis. Yet many challenging oxidative transformations remain heavily reliant on high-energy blue light, which can limit substrate scope and restrict their applicability. In this work, we introduce a novel class of fluorinated azadioxotriangulenium (2F-ADOTA$^+$) organic photocatalysts that successfully shift the activation window to milder, low-energy orange light. Despite this red shift, the 2F-ADOTA$^+$ carbocation functions as a remarkably potent photooxidant, unlocking two highly demanding synthetic methodologies.

Carolina Figueroa – Biochemistry #28

Research Faculty Advisor: Kathleen Rodgers, Pharmacology

Neuroinflammation and Systematic Characterization in a Multiple Sclerosis Transgenic Mouse Model

The objective of the experiment is to explore the trend of mice behavior in neurodegenerative disease and to find if there is a dependence on sex in mice models. To test their behavior for observation and analysis, we performed Novel Object Recognition (NOR) tests and scored their nesting abilities. Key findings included a significant change in the behavior of diseased females compared to the diseased males at 15 months.

Mariella Gunther – Biochemistry #11

Research Faculty Advisor: Tarjani Thaker, Chemistry and Biochemistry

Understanding the role of COQ3 and COQ8B in Coenzyme Q biosynthesis

Coenzyme Q is an essential component of the electron transport chain for the generation of ATP. It is synthesized in the mitochondria by a metabolon of different proteins, Complex Q, some of whose functions are not fully understood. CoQ10 deficiency can lead to neurological and renal diseases, making the study of its biosynthesis important. COQ3 is an o-methyltransferase involved in the formation of the CoQ head group, while COQ8B is an atypical kinase proposed to regulate CoQ10 biosynthesis. The goal of this study was to purify mammalian COQ3, as well as an ancestral COQ3 construct to enable future investigation of its interaction with COQ8B. Although mammalian COQ3 expression was detected at the expected molecular weight, significant protein loss during purification limited recovery. In contrast, purification of the ancestral COQ3 construct was successful. It produced higher yields in two independent purifications, with largely pure elution fractions observed. Some protein loss was still observed in the flow-through and wash fractions, along with uncleaved products. These results highlight the challenges associated with mammalian COQ3 purification while identifying ancestral COQ3 as a stronger construct for future optimization and interaction studies.

Rylan Hammond – Biochemistry, Molecular and Cellular Biology #06

Research Faculty Advisor: Michael F. Brown, Chemistry and Biochemistry

Effect of Free-Flowing Water on Rhodopsin Activation

Rhodopsin is the archetypal visual receptor and a canonical member of the largest and most pharmacologically targeted membrane protein family, the G-protein-coupled receptors (GPCRs). Rhodopsin activation, which involves a complex sequence of structural changes, provides an excellent model for how GPCRs adopt ligand-induced active conformation, which then goes on to activate G proteins. Upon photon absorption, the 11-cis-retinal ligand isomerizes to all-trans-retinal, culminating in the formation of the fully activated signaling state, which is called metarhodopsin II (Meta II). Meta II is distinct from the inactive Meta I state by the presence of the deprotonated retinylidene Schiff base and is stabilized by the protonation of the conserved E(D)RY motif. The active Meta II state binds the heterotrimeric G protein, transducin, and initiates nucleotide exchange. This coupling requires the G protein's C-terminal α-helix to form the major contact interface with rhodopsin's transmembrane helices. Rhodopsin activation necessitates significant conformational changes. In addition, this process is coupled to a large influx of bulk water into the protein core. The methods that exist for analyzing the influx of this bulk water are insufficient in revealing the receptor's full dynamic allostery, especially regarding the crucial influence of this water. The free energy effects of this influx of bulk water must be further explored. Osmotic stress studies suggest that approximately 80–100 water molecules hydrate rhodopsin during Meta II formation, resulting in a dynamic, solvent-swollen, sponge-like state. This influx of bulk water stabilizes the active GPCR conformation and forces the intracellular side of the receptor to open, mediating signaling and recruitment of G protein binding. From a thermodynamic perspective, rhodopsin activation is governed by a delicate balance between enthalpic and entropic contributions that together define the stability of the Meta I and Meta II states. The influx of such bulk water into the receptor core increases the overall disorder of the system, providing a favorable entropic driving force that stabilizes the active conformation. The enthalpy of the system is also modulated by this destabilization caused by bulk water movement. Thus, the effects of osmotic stress on the entropic and enthalpic differences between the inactive and active states of rhodopsin are important to explore in understanding the nature of GPCRs.

Leah Harroun – Biochemistry #03

Research Faculty Advisor: Oliver L.A. Monti, Chemistry and Biochemistry

SOMO-Mediated Transport in Verdazyl Radicals: Limitations Imposed by Fermi Level Pinning

Inefficiencies and size limitations of modern electronics have motivated research into molecular electronics, working to identify organic single molecules that can perform the functions of semiconductors and electronic switches. This work used a mechanically controlled break junction (MCBJ) to measure the conductance of the Verdazyl radical series, a series of molecules with the same backbone but differing in substituent. Due to the unpaired electrons, radicals have a singly occupied molecular orbital (SOMO) that lies higher in energy than the highest occupied orbital (HOMO) of any closed-shell molecule counterparts. It was hypothesized that these features could be harnessed to tune a radical until the SOMO overlaps the Fermi energy of the gold electrodes, allowing free conductance of electrons through the molecule. This would be expected to produce metallic conductance, or conductance on the order of 100 G0, but experimental results showed conductances limited to 10-2 G0 as the highest measured conductance in the series. Despite the special case of the -Phx substituent, which experiences orbital inversion, transport was still observed through the SOMO, although it was far from the magnitude of the conductance values expected. While radicals have a higher energy orbital that is only half filled, this orbital does not behave as a half-filled HOMO, and remains subject to Fermi level pinning.

Riley Haveman – Pharmaceutical Science minors: Biochemistry, Thematic #27

Research Faculty Advisor: Tally Largent-Milnes, Medical Pharmacology

Endocannabinoid Signaling in Medication Overuse Headache

Migraines affect over one billion individuals globally each year, yet effective treatments for symptoms like severe headache and sensory hypersensitivities remain limited, which can cause many individuals to overuse pain management medications. This can lead to the development of a secondary headache disorder known as medication overuse headache (MOH). With such high prevalence and morbidity, identifying potential targets for therapeutic benefit is critical. Previous research posits that reduced endocannabinoid tone may contribute to migraine development and other neuropathic pain conditions. Moreover, our previous work in the lab has revealed that the levels of 2-arachidonoylglycerol (2-AG), a major endocannabinoid, are reduced in the periaqueductal grey (PAG), a key area of the brain associated with migraine, following headache induction. This phenomenon is due to the increased breakdown of the molecule by its corresponding hydrolysis enzymes, α/β-hydrolase domain-containing 6 (ABHD6) and monoacylglycerol lipase (MAGL). This project seeks to elucidate the receptor mechanism of endocannabinoid action during medication overuse headache (MOH) and mitigation of MOH by blocking 2-AG degradation. We do so by inducing MOH in rats following the implantation of mini osmotic pumps filled with sumatriptan and assessing for periorbital allodynia at multiple points throughout the study. Preliminary results have revealed that the dosing of CB1 receptor antagonist on day 5 has no statistically significant effect on collected periorbital von frey data compared to controls. More trials with the CB1 receptor antagonist are needed to ensure statistical power and further exploration of the effect of a CB2 receptor antagonist may expand our current understanding of the mechanism of action.

Kyla Holmquist – Biochemistry #04

Research Faculty Advisor: Minying Cai, Chemistry and Biochemistry

Amyloid-beta 40 Aggregation Using Plasmon Waveguide Resonance (PWR)

Amyloid monomers form when the amyloid precursor protein (APP) proteolytic amyloidogenic pathway is activated. APP is cleaved by β-Secretase and γ-Secretase, releasing extracellular Aβ. Amyloid plaques form when Aβ40 and Aβ42 proteins accumulate and aggregate in the fatty lipid membrane bordering neurons. Amyloid plaques cause inflammation and neurodegeneration. Amyloid-beta 40 protein aggregation is dependent on lipid environments. Exploring the relationship between membrane composition and Aβ40 aggregates would have applications in Alzheimer's Disease treatments and other neurodegenerative diseases. The Cai Lab is measuring Aβ40 aggregation in different regions of a rat brain using plasma waveguide resonance. PWR measures P- and S-polarized light to discern binding on a membrane. The P and S shift in incident angle for the midbrain region is the most dramatic, with a 68.93mdeg shift parallel to the membrane and a 97.12mdeg shift perpendicular to the membrane. Aβ40 aggregates at lower concentrations (.0166 μM) than the indicated Aβ40 critical aggregation concentrations of 0.5 ± 0.3 μM, further supporting the midbrain lipid composition being an ideal environment for Aβ40 aggregation.

Chase Johnson – Biochemistry #48

Research Faculty Advisor: Michael A Riehle, Entomology

Biological and molecular phenotypes associated with RNAi-mediated COPI Beta and Gamma deficiency in Anopheles stephensi female mosquitoes

Blood meal digestion is essential for reproduction in female Anopheles stephensi mosquitoes. The COPI vesicle coatomer complex plays a crucial role in intracellular vesicle trafficking in all organisms. In this study, RNA interference was employed to knockdown both COPI beta and COPI gamma subunits to evaluate their overall role in blood meal digestion and survival in An. stephensi. BSA digestion assays showed that the control mosquitoes microinjected with dsRNA-Fluc displayed normal digestion by 24 hours post blood meal (PBM), whereas COPI

knockdown mosquitoes retained high levels of intact BSA, strongly indicating delayed digestion. The early time points (2 hours PBM) showed no differences, suggesting that COPI is specifically required during active blood protein digestion by digestive proteases. Survivorship assays revealed rapid mortality in COPI knockdown mosquitoes following a blood meal, while the control mosquitoes showed high survival rates. Taken together, these results demonstrate that COPI-mediated trafficking of proteases is essential for blood meal protein degradation and mosquito survival, suggesting digestive pathways as potential physiological targets for mosquito vector control strategies.

Isaac Kailat – Biochemistry, Physics #42

Research Faculty Advisor: Yeran Bai, Optical Sciences

Label Free Metabolic Profiling via MIP Microscopy

Artificial sweeteners, or non-nutritive sweeteners (NNS), are increasingly prevalent in the modern diet, yet growing evidence suggests they alter the metabolic footprint of gut bacteria, with cascading effects along the gut-brain axis. Understanding how these compounds disregulate cellular metabolism at the single-cell level is critical, but conventional biophysical instrumentation faces a fundamental tradeoff: techniques can either characterize molecular metabolites or spatially resolve single-cell morphology, and those that accomplish both—such as NanoSIMS—suffer from low throughput and high cost. This study investigates whether mid-infrared photothermal (MIP) microscopy can serve as a high-throughput, label-free platform for probing single-cell metabolism, addressing the central question: how do artificial sweeteners disregulate cellular metabolism in E. coli? MIP microscopy enables super-resolution infrared spectroscopic imaging of living systems by detecting photothermal responses to mid-infrared excitation, providing chemical specificity at the single-cell level. E. coli, a representative member of the gut microbiome, were cultured under exposure to common artificial sweeteners, and MIP imaging was employed to characterize metabolic states based on intrinsic molecular vibrational signatures. We expect MIP microscopy to reveal distinct spectral fingerprints corresponding to altered metabolic profiles in sweetener-exposed bacteria compared to controls, demonstrating the technique's capacity for rapid, label-free metabolic phenotyping. These findings would establish MIP microscopy as a powerful tool for studying the bottom-up metabolic effects of dietary compounds on gut bacteria, with broader implications for understanding the impact of artificial sweeteners on human health through the gut-brain axis.

Dani Khatib – Biochemistry, French #37

Research Faculty Advisor: Deepta Bhattacharya, Immunobiology

Tumor-Infiltrating Monoclonal Antibody Production

The current state of cancer treatment is largely dependent on the use of chemotherapy and radiotherapy—two treatments that cause inadvertent damage to many healthy cells within patients. Immunotherapy, on the other hand, is a method of treating cancer using the patient's own immune system. One of the mechanisms through which the human immune system promotes tumor elimination is the production of antibodies that bind to cancerous cells. Therefore, characterizing both the antibodies and their respective cancer-dependent epitopes is vital for the development of monoclonal antibodies (mAbs). As a part of immunotherapy, mAbs can be administered to cancer patients, where they bind to cancerous cells and allow for the patient's immune system to specifically target cancerous cells while minimizing harm to healthy cells. The aim of this research project is to produce tumor-infiltrating monoclonal antibodies for immunotherapeutic treatment of lung cancer by isolating B cells from human lung tumors, sequencing them for their antibody sequences, and determining what those antibodies are binding to on the surface of lung cancer cells. Such research is essential for the advancement of the field of immunotherapy in order to implement a more direct treatment for lung cancer that is significantly less harmful to healthy human cells. Additionally, identifying the distinguishing cell surface markers between healthy and cancerous cells can uncover the fundamental processes to break the immune system's self-tolerance.

Kaylee Kimbrell – Biochemistry, Physiology and Medical Sciences #32

Research Faculty Advisor: Sean Limesand, Animal & Comparative Biomedical Sciences

Enhanced Insulin Signaling Impairs TGF-b Pathway Causing Increased Satellite Cell Differentiation in Growth Restricted Lambs

Transforming Growth Factor-b (TGF-b) is a critical cytokine that regulates the differentiation of satellite cells (SC). Previous findings indicate that while TGF-b inhibits differentiation in normal lamb (CON) SC, cells from lambs with fetal growth restriction (FGR) exhibit significantly reduced responsiveness. Because previous molecular analysis identified DNA hypomethylation in the promoters of insulin signaling pathway (ISP) genes in FGR SC, we hypothesized that the reduced TGF-b sensitivity results from upstream overactivity in the ISP, which functionally overrides inhibitory TGF-b signaling through molecular crosstalk.

To test this hypothesis, we quantified differentiation rates in primary FGR and CON SC following treatments that modulate ISP and TGF-b signaling. While TGF-b suppressed differentiation in both groups, it was less effective in FGR SC compared to CON SC (0.98-fold vs. 0.44-fold; $p = 0.044$). Notably, stimulation with insulin like growth factor-1, an activator of the ISP, produced a 2.69-fold increase in myogenin positive (MyoG+) nuclei in FGR SC compared to only a 1.07-fold increase in CON ($p = 0.021$). Furthermore, treatment with TGF-b inhibitor A83-01 resulted in a nearly twofold greater differentiation response in FGR SC compared to CON (10.37-fold vs. 5.33-fold; $p = 0.046$).

These findings suggest that FGR SC are epigenetically primed for ISP overactivity due to FGR-induced epigenetic modifications, allowing growth signals to override the regulatory signaling provided by TGF-b. This dysregulation may lead to the premature depletion of the skeletal muscle stem cell pool, providing a mechanistic link between FGR and the increased risk for muscle-related metabolic dysfunction later in life.

Jessica Klinger – Biochemistry #16

Research Faculty Advisor: Marielle Hegetschweiler, Chemistry and Biochemistry

The Stabilizing Effect of Cochaperone Hsp10 on ATP-Induced Monomerization of Hsp60

The human chaperonin complex composed of heat shock protein 60 (Hsp60) and heat shock protein 10 (Hsp10) play an essential role in cellular proteostasis by promoting the correct folding of substrate proteins. Protein misfolding increases under cellular stress conditions, including cancer and disease, highlighting the importance of this chaperonin complex in maintaining cell homeostasis. Hsp60 is an ATP-dependent molecular chaperone whose ATP binding induces large conformational rearrangements that expand the central folding cavity, enabling substrate encapsulation and subsequent binding of the co-chaperonin Hsp10. Previous work from others and our laboratory demonstrated that Hsp60 exists in multiple oligomeric states, including monomeric, heptameric, and tetradecameric assemblies. Notably, we observed that apo Hsp60 undergoes ATP-induced disassembly from its tetradecameric state, a behavior not previously reported in the literature. The present study investigates whether ATP similarly induces disassembly in the Hsp10-bound state of Hsp60. Using mass photometry and charge detection mass spectrometry, we examined the oligomeric stability of the Hsp60/Hsp10 complex following ATP addition. Our results demonstrate that Hsp10 binding stabilizes the Hsp60 heptamer- and tetramer- domain oligomers, preventing ATP-induced disassembly into monomers. These findings suggest that co-chaperonin association modulates the structural stability of naïve Hsp60 and provides insight into regulatory mechanisms governing chaperonin function.

Caleb Konecek – Biochemistry, Molecular and Cellular Biology #21

Research Faculty Advisor: Pascale Charest, Molecular & Cellular Biology

Role of Rap1 in Regulating mTORC2 Signaling in HEK293T Cells

Chemotaxis is a directed cell migration in response to external chemical stimuli that promotes biological processes like development and immune response. Chemotaxis dysregulation has been linked to disease spread like cancer metastasis, but its disruption isn't fully understood. Research has shown that the mechanistic Target of Rapamycin Complex 2 (mTORC2) is crucial for chemotaxis regulation and cytoskeletal protein rearrangement, though its precise role in these processes remains unclear. Recently, we have observed the binding of both Rap1, a Ras-associated small GTPase, and PIP3, a membrane phospholipid, to SIN1, a component of mTORC2. Previous experiments have also linked Rap1 overexpression to increased mTORC2 activity and have implicated PIP3 in mTORC2 activation. Although Rap1 and PIP3 have both been linked to the regulation of mTORC2, we wanted to further investigate the relationship between the two to determine if they play the same role in regulating the pathway in distinct cellular contexts or if they play different roles that coordinately regulate mTORC2. We hypothesize that Rap1 and PIP3 serve similar roles in the positive regulation of mTORC2 activity by binding to the pH domain of its SIN1 component and aiding in its membrane localization. This thesis attempts to clarify the relationship between these proteins to provide further insight into the mTORC2 pathway while also examining the effect of constitutively active Rap1 on downstream targets of mTORC2. To test this, we over-expressed Rap1 and used a PIP3-production inhibitor, examining their effects on mTORC2 activity in HEK293T cells stimulated by insulin, a strong activator of mTORC2. Our research indicates that Rap1 and PIP3 regulate mTORC2 activity in an additive fashion, and that constitutively active Rap1 promotes phosphorylation of NDRG1 while having little effect on the phosphorylation of FOXO and PKC in this cell type. By offering further insight into this signaling pathway, this research seeks to further our understanding of human disease and identify potential targets for future drug therapies.

Alicia Krusee – Biochemistry #12

Research Faculty Advisor: Tarjani Thaker, Chemistry and Biochemistry

Unveiling the Functional Role of ADCK4/COQ8B in Kidney Disease

Mutations in the human mitochondrial atypical kinase ADCK4, commonly referred to as COQ8B, are a primary cause of Steroid-Resistant Nephrotic Syndrome (SRNS), defined by kidney filtration barrier failure. SRNS is linked to the impairment of podocytes, a specialized kidney cell that relies on Coenzyme Q10 (CoQ10) for mitochondrial energy production and insulin signaling to regulate podocyte functions like filtration. While it has been established that ADCK4 function is essential for CoQ10 biosynthesis, it remains unclear how ADCK4 mutations impact CoQ10 levels and insulin signaling. As a result, further insight is needed to understand the kinase's role in bridging metabolic and signaling pathways. To investigate the structure-function relationship of ADCK4, we used two patient-derived mutations known to cause SRNS: the I346S interdomain mutation, which disrupts the interface near the active-site cleft, and the W520X truncation, which eliminates the C-terminal tail. Using ADCK4-knockout human podocyte cell lines, we quantified CoQ10 and performed Western blot analysis to evaluate the impact of these mutations on metabolic and signaling function. Quantification assays revealed that I346S and W520X independently caused significant CoQ10 depletion, whereas the double mutant (I346S + W520X) exhibited a rescue effect that restored CoQ10 levels. The double mutant result suggests an interesting correlation between the active-site cleft and the C-terminal tail, in which ADCK4's function, specifically for CoQ10 biosynthesis, is not hindered despite the mutations. Insulin stimulation assays demonstrated that loss or mutation of ADCK4 lead to dysregulated signaling kinetics, with Akt2 and MAPK exhibiting significantly faster phosphorylation activation followed by a rapid decline compared to the wild type control. Ultimately, these results are indicative of ADCK4's function regarding CoQ10 biosynthesis, energy productivity, and a critical link to the insulin signaling pathway necessary for a podocyte's role within the kidney. By uncovering this relationship, our preliminary work emphasizes that maintaining mitochondrial signaling crosstalk is crucial for preserving the glomerular filtration barrier, offering new insight into steroid-resistant kidney disease.

Jacquelyn Lo Bianco – Biochemistry, Molecular and Cellular Biology #26

Research Faculty Advisor: Tally Largent-Milnes, Pharmacology

Endocannabinoid Targets in the Brain Cortex for Medication Overuse Headache (MOH) Reversal

Medication overuse headache (MOH) is the largest secondary headache disorder that affects people around the world. While the clinical importance of the disease has been established, the molecular pathways that MOH affects are not well understood. The current theories lead to the trigeminovascular system of the brain as the source of headache-related pain and the endocannabinoid system (ECS) as the system's regulating factor. This poster discusses an investigation into the expression, quantification, and analysis of endocannabinoid system enzymatic constituents after sustained sumatriptan infusion in the presence and absence of the ABHD6 inhibitor, KT-182, as well as impacts on the Th17-associated cytokine profile within MOH pain-relevant brain regions.

Tiffany Luu – Biochemistry #07

Research Faculty Advisor: Michael Brown, Chemistry and Biochemistry

SOFT MATTER INFLUENCES RHODOPSIN G-PROTEIN BINDING IN LIPID MEMBRANES

Soft matter, namely water and lipids, comprises an essential part of the native environment of membrane proteins. However, the mechanism of how lipids and water interact with proteins remains poorly understood. To uncover these questions, we use rhodopsin which is an archetypical G-protein-coupled receptor (GPCR) that serves as a model to understand soft matter effects. Upon photoactivation, the chromophore retinal isomerizes from 11-cis to all-trans within the rhodopsin orthosteric site, triggering conformational changes into several intermediates, including preactive metarhodopsin-I and active metarhodopsin-II states. We previously demonstrated that the activation equilibrium is sensitive to dehydration of the protein interior and lipid-protein interactions as explained by the flexible surface model [1,2]. Here, we test this sponge model of rhodopsin activation using osmolytes to probe the effects of dehydration on the binding of transducin mimetic peptides to rhodopsin in various lipid environments [1]. UV-visible spectroscopy was used to measure rhodopsin activation, and a binding model was employed to calculate the dissociation constant of the peptides. We hypothesize that binding and release of the transducin G-protein is coupled to rhodopsin hydration cycling. Notably, we discovered that osmolytes affect rhodopsin activation, with small osmolytes penetrating the binding pocket and increasing activation. Furthermore, we found that osmolytes do not compete with binding of these mimetic peptides as the binding constant is unaffected by small osmolytes. We also observe that rhodopsin in unsaturated recombinant lipids has a higher binding affinity to these peptides than its native membrane, hinting that curvature forces are involved in G-protein binding. Unveiling water and lipid effects on transducin peptide binding to rhodopsin offers medical applications, from pharmaceutical drug development to deciphering disease mechanisms. [1] U.Chawla et al. (2021) Angew. Chem. Int. Ed. 60, 2288. [2] S.D.E.Fried et al. (2022) PNAS 119, e2117349119.

Maxwell Maloney – Biochemistry #31

Research Faculty Advisor: Nathan Cherrington, Pharmacology & Toxicology

MASH Impairs Renal Anion Transporter Function and Alters Cefazolin Disposition in Mice

Metabolic dysfunction-associated steatohepatitis (MASH) is a progressive form of liver disease known to alter the expression and function of both hepatic and renal transporters. These changes may influence the renal disposition of numerous pharmaceutical compounds. The objective of this study was to investigate the effects of this phenoconversion on the pharmacokinetics of cefazolin (CEF). MASH was induced in mice using a fast-food diet/thioacetamide (FFDTH) model, which has been shown to accurately model the renal transporter changes seen in human MASH. Histological assessment revealed characteristics of the MASH phenotype including steatosis and inflammation. Expression of the renal transporters Oat3 and Oat5 was significantly reduced in FFDTH mice compared to controls. Oat3 expression decreased to 101.6 ± 44 pmol/mg protein from 203.9 ± 52.3 pmol/mg protein (Figure 3B). Similarly, expression of the apical transporter Oat5 was reduced by approximately 80%, from 30.60 ± 4.30 pmol/mg protein in healthy mice to 6.21 ± 2.30 pmol/mg protein in FFDTH mice (Figure

3C). Hepatic transporters remained unchanged in response to the FFDTH model, despite this plasma concentrations of CEF increased to 183% in FFDTH mice compared to controls. Similarly, hepatic accumulation increased to 180% of control. In contrast, there was no difference between FFDTH and healthy mice for CEF concentration in the kidneys or in the urine. Glomerular filtration rate (GFR) was significantly reduced in FFDTH mice (0.34 mL/min/g) compared to controls (0.76 mL/min/g), Collectively, these results imply that reduced GFR, in combination with decreased OAT transporter expression, impairs renal clearance and contributes to increased systemic exposure of CEF. Overall, these findings suggest a risk of increased systemic exposure to CEF as well as higher burden in the liver in MASH patients, including those undergoing surgery. As such, it would be beneficial to explore potential dose adjustments to balance efficacy and safety in this patient population.

Mara Lluvitza Navarro Perez – Biochemistry, Microbiology #34

Research Faculty Advisor: Gayatri Vedantam and Farhan Anwar, Animal & Comparative Biomedical Sciences

The Prevalence and Diagnostic Characterization of Clostridioides difficile in Southern Arizona (2024-2025)

Clostridioides difficile is a rod-shaped, spore-forming anaerobic bacteria and a common cause of antibiotic- and healthcare-associated diarrhea. The Centers for Disease Control and Prevention estimated C. difficile infection (CDI) caused 223,900 hospitalizations and 12,800 deaths in 2019 alone. Our CDI surveillance program and ribotyping of clinical isolates are performed to document strain diversity and endemic prevalence. This study evaluated 371 de-identified, to-be-discarded stool specimens collected from Southern Arizona hospital patients with diarrheal symptoms in 2024 - 2025.

The aim of this study was to quantify the C. difficile prevalence rate in Southern Arizona. Out of 371 specimens, 313 were analyzed based on their respective GDH/Toxin diagnostic test results, wards, and date of collection information. The frequency of positive test results (GDH+/Tox+) accounted for 14.4% (n=45), whereas the combined frequency of GDH+/Tox EIA- and false negative (GDH-/Tox EIA-/ C. difficile +) results was 21.4% (n=67). Of the 371 specimens evaluated, 153 C. difficile strains were isolated (41.2%). Notably isolates were recovered from all but two of the GDH-positive specimens. While ribotyping is an ongoing effort, the isolation rates and diagnostic profiles highlight the endemic prevalence of C. difficile in Southern Arizona and the potential for misdiagnosis.

Brynn Nichols – Biochemistry, Neuroscience, Classics #19

Research Faculty Advisor: Juliana Sacoman, Chemistry and Biochemistry

Beyond Memorization: Promoting Active Learning Using Case Studies

Undergraduate science education often relies on passive learning strategies, such as memorization, which can limit students' ability to apply complex biochemical concepts to real- world scenarios. Active learning approaches, particularly case-based learning, provide opportunities for students to engage in higher-order thinking by integrating scientific knowledge with ethical reasoning in various real-case scenarios. This project aimed to promote active learning through the implementation of case studies in an upper-division undergraduate course, focusing on the intersection of biochemistry, bioethics, and culturally relevant contexts. Three case studies were developed, with themes involving patient confidentiality/autonomy, genetically modified organisms, and germline genome editing. Each case requires students to evaluate real-world scenarios using biochemical principles while also considering ethical trade- offs related to public health, environmental impact, and societal implications. These case studies encouraged students to engage with topics such as healthcare access disparities, ecological risks, and genetic equity, fostering deeper conceptual understanding and critical thinking beyond memorization of facts. Therefore, by connecting biochemical mechanisms to culturally and socially relevant issues, this approach enhances student learning, engagement, and promotes the development of transferable skills such as scientific reasoning and communication.

Sofia Orrantia – Biochemistry, Molecular and Cellular Biology #40

Research Faculty Advisor: Jennifer S. Carew, Cancer Center

Novel Brain-Penetrant Bifunctional Small Molecule Inhibitors for Glioblastoma Therapy

Gli+I37oblastoma multiforme is the most common and aggressive brain malignancy in adults. New strategies are urgently needed to improve outcomes for patients with glioblastoma multiforme. Histone deacetylase Inhibitors (HDACi) are a class of anticancer agents that inhibit the activity of HDACs, leading to cell cycle arrest, apoptosis, and differentiation in cancer cells, making HDAC inhibitors promising anticancer agents. Several HDAC inhibitors are FDA-approved but display limited efficacy due to resistance mechanisms such as induction of autophagy. We sought to develop dual HDAC and autophagy inhibitors with improved anticancer efficacy. To this end, we have developed H6, which we hypothesize will disrupt both HDAC activity and autophagy and display significant anticancer efficacy against GBM models. The data indicates that H6 significantly reduces GBM cell viability across multiple GBM models. Increased sub-G_o/G_1 populations confirm that H6 induces apoptosis. H6 binds to HDAC1 through in silico analysis. H6 also was found to bind to other HDACs and various lysosomal proteases. Western blotting confirmed that H6 increased acetylated histone H3 levels and p21 expression, consistent with HDAC inhibition, while also leading to p62 stabilization indicating impaired autophagic flux. Future studies will include HDAC activity assays to confirm on-target inhibition and relative potency of H6 and will evaluate the potential toxicity of H6 in normal brain cells.

Ronald Palmenberg – Biochemistry, German Studies #14

Research Faculty Advisor: Matthew Cordes, Chemistry and Biochemistry

Key mutations associated with venom recruitment in a recluse spider toxin

Recluse spiders in the Sicariidae family have toxic bites that often cause loxoscelism, characterized by dermonecrotic and sometimes hemolytic syndromes. Their venom is predominantly comprised of phospholipase D (PLD) enzymes, known to degrade important membrane lipid substrates into cyclic products. Sequence comparison of ancestral recluse spider PLDs before and after venom recruitment reveal three consistent mutations distributed between two lipid binding sites, the enzymatic active site and an additional lipid-binding site with allosteric functions. We hypothesize that these mutations are primarily responsible for the changes in enzyme activity and substrate preference observed with venom recruitment. Three mutated venom ancestors were created: active site mutation M246I, allosteric site mutations T225W/S233T, and combined active and allosteric mutations T225W/S233T/M246I. These mutants represent pre-venom recruitment PLD active site and/or allosteric site with no extrinsic changes. The M246I mutant showed a significant increase in enzyme activity and change in substrate preference towards sphingomyelin (SM) over ceramide phosphoethanolamine (CPE). The T225W/S233T showed no significant increase in enzyme activity nor change in substrate preference, but liposome pulldown showed a 10-20% increase on SM membrane binding. The T225W/S233T/M246I mutant showed significant increase in enzyme activity and a stronger shift towards SM beyond that of the M246I mutation alone. A decrease in catalytic activity upon venom recruitment may seem surprising, but these mutations are coupled with the loss of an inhibitory C-terminal domain, which may be compensatory. Additionally, the shift in substrate preference may reflect the universality of CPE in spider prey. These studies prove the mutations are important for substrate binding, preference, and catalysis, providing the scientific community with an understanding of the exact amino acid residues responsible for venom-expressed PLD toxicity. This could lead to safe and FDA-approved loxoscelism treatments or other practical applications.

Delaney Petruzelli – Biochemistry #02

Research Faculty Advisor: Thomas Gianetti, Chemistry and Biochemistry

Pendent Arm and Bridging Atom Variation in Carbenium Ions: Synthesis, Properties, and Applications

Triaryl carbenium ions, such as AzaDiOxaTriAngulenium (ADOTA) and DiMethoxyQuinAcridinium (DMQA), are stable carbocations that have demonstrated unique applications in red-light-mediated photoredox catalysis and symmetric organic redox flow batteries. The photophysical and electrochemical properties of these carbenium cores can be tuned through various modifications. Herein, we explore different variations of pendant arms around the carbenium core, as well as different bridging atoms (i.e. nitrogen vs. oxygen). We successfully introduced a pyrene pendant arm and a bipyridine pendant arm. The pyrene arm unlocks access to an extended conjugated system via ring closure with the core, while the bipyridine arm enables metal binding. We present their synthesis, photophysical and electrochemical properties, and discuss their potential applications in future research.

Aidan Prahl – Biochemistry #25

Research Faculty Advisor: Brett Colson, Cellular and Molecular Medicine

Developing a Fluorescence Lifetime-Based Assay to Characterize Small-Molecule Binding to N-terminal Cardiac Myosin-Binding Protein C

Cardiac Myosin Binding Protein-C (cMyBP-C) is encoded by the MYBPC3 gene and plays an essential role in modulating cardiac muscle contraction through interactions with actin and myosin. Mutations in MYBPC3 are a significant cause of familial hypertrophic cardiomyopathy (HCM), however, the structural and conformational mechanisms involved in cMyBP-C regulation and therapeutic targeting are largely not understood. The N-terminal domains of cMyBP-C (C0-C2) are of prominent interest due to their actin and myosin S2 head dual-binding capabilities and regulation by protein kinase A (PKA)-mediated phosphorylation. The present study investigates domain-specific structural changes in N-terminal cMyBP-C induced by small-molecule binding utilizing a high-throughput fluorescence lifetime (FLT) assay. A total of five C0C2 constructs of cMyBP-C with differential engineered cysteine residue sites generated via site-directed mutagenesis and labeled with a tetramethylrhodamine (TMR) probe for site-specific perturbations. Protein constructs were expressed, purified, and analyzed in both phosphorylated and non-phosphorylated states. A library of 60 candidate compounds, that showed previous evidential binding to N-terminal cMyBP-C, was screened in a 384-well plate, and FLT changes were statistically quantified using Z-score analysis to interrogate local structural protein alterations due to small-molecule incubation. Results proved site-dependent variability in probe accessibility, with cysteine 184 showing the highest labelling efficiency. Phosphorylation produced detectable FLT changes, consistent with structural perturbations. Small-molecule incubation and screening demonstrated differential, site-specific structural responses, with consideration for differences dependent on both probe location and phosphorylation state. Such findings establish FLT as a high-resolution method for detecting domain-specific structural changes in cMyBP-C and provides a scalable application for therapeutic screening. This work outlines the foundation for identifying compounds that regulate cMyBP-C structure and function, progressing drug discovery efforts for HCM.

Soleil Robinson – Biochemistry #17

Research Faculty Advisor: Marielle Hegetschweiler, Chemistry and Biochemistry

Investigating the Role of the C-terminal Tail in mitochondrial human Heat Shock Protein 60 (mtHsp60)

Molecular chaperones play a critical part in maintaining protein homeostasis under conditions that challenge cellular stability. They facilitate the folding of imported proteins, refolding misfolded proteins, and preventing aggregation. One such chaperonin system that has been proven to be present under cellular stress is the mitochondrial 60-kDa heat shock protein 60 (mtHsp60). The mtHsp60 functions along with its co-chaperon, heat shock protein 10 (Hsp10) in an ATP-dependent manner. Unlike mtHSP60, its bacterial homolog GroEL is rather well studied. It has been seen in GroEL that its C-terminal tail assists in protein folding. We wanted to study the

role of the C-terminal tail in human mtHsp60 structure and function. I mutated the wt mtHsp60 to remove the C-terminal tail (ΔC mtHsp60).Then, I overexpressed, purified, and studied the ΔC mtHsp60 mutant alongside the wt. Here, we compare them in terms of assembled state, protein refolding, and ATP hydrolysis. Here, we show that ΔC mtHsp60 mutant is seen to be less functional. Our study underscores the importance of the C-terminal tail in the mtHsp60 activity.

Michaela Ryan – Biochemistry #15

Research Faculty Advisor: Mark Manary, Professor of Pediatrics at Washington University School of Medicine in St. Louis, Missouri

Effects of Egg Supplementation on Intestinal Inflammation and Regeneration Markers in Moderately Malnourished Children in Sierra Leone

Environmental enteric dysfunction (EED) is a chronic inflammatory condition of the small intestine that affects millions of children living in low-resource settings and contributes to impaired growth. Fecal messenger RNA (mRNA) transcripts provide a non-invasive method to evaluate intestinal inflammation and epithelial repair. Eggs contain highly bioavailable amino acids, choline, and bioactive peptides that may promote intestinal healing. This study is a secondary analysis of a randomized controlled trial conducted in Sierra Leone that evaluated whether 24 weeks of daily whole egg powder supplementation altered fecal mRNA markers of intestinal regeneration and inflammation in children with moderate acute malnutrition (MAM). Seven transcripts were quantified using droplet digital PCR (ddPCR): CDX1, MUC12, REG1A (regeneration domain), and CD53, HLA-DRA, S100A8, TNF (inflammation domain). Among 192 children included in this substudy (138 with week 24 samples), egg supplementation did not significantly alter any individual transcript or composite domain score at 6, 12, or 24 weeks compared with corn flour. Inflammatory transcripts were strongly correlated with one another, while regeneration transcripts correlated inversely with intestinal permeability. These findings suggest that egg supplementation does not modify mucosal inflammatory or regenerative transcriptional pathways in children with MAM.

Erin Schuette – Biochemistry #39

Research Faculty Advisor: Dawn Coletta, Medicine

Characterization of MALAT1 Expression and Genetic Associations in Inflammation and Metabolic Dysfunction

Metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) is a highly expressed long noncoding RNA implicated in inflammatory signaling and metabolic disease, with emerging evidence linking its expression to genetic variation in the ectodysplasin receptor (EDAR) pathway. This study aimed to characterize MALAT1 expression across tissues and determine its associations with metabolic phenotypes and genetic variation. We first performed single-cell RNA sequencing (scRNA-seq) on skin biopsies from genotype-stratified participants to evaluate cell type–specific MALAT1 expression. MALAT1 was broadly expressed across cell types, with higher levels observed in T cells and vascular endothelial cells from EDAR370A carriers. To assess systemic relevance, we analyzed whole-blood transcriptomic data from the Arizona Insulin Resistance registry, which similarly showed elevated MALAT1 expression in EDAR370A carriers. To investigate inflammatory regulation, human keratinocytes (HEKa) were treated with lipopolysaccharide (LPS), confirming induction of inflammatory markers alongside modest increases in MALAT1. Associations between MALAT1 expression and metabolic phenotypes, including insulin resistance, glycemic control, and adiponectin levels, revealed significant relationships. Genetic analyses in El Banco por Salud participants identified single nucleotide polymorphisms within MALAT1 and adjacent transcripts associated with multiple metabolic phenotypes, with distinct glycemic and lipid-related clusters. MALAT1 expression was evaluated across peripheral metabolic tissues, during C2C12 differentiation, and in human skeletal muscle following acute exercise, demonstrating dynamic, context-dependent regulation. Finally, expression of MALAT1, EDA, EDAR, and EDA2R in skeletal muscle varied by EDAR370 genotype and body composition. Together, these findings position MALAT1 as a multi-tissue regulator linking inflammation to metabolic dysfunction.

Jordan Schultz – Biochemistry #35

Research Faculty Advisor: Chi Zhou, Animal & Comparative Biomedical Sciences

The Effect of Maternal Obesity on Cellular Senescence

Maternal obesity is a prominent risk factor for pregnancy complications and is associated with adverse perinatal and long-term fetal outcomes, such as small or large gestational age and during pregnancy and increased risk of cardiovascular diseases in the offspring. Although placental dysfunction is known to contribute to these maternal obesity-associated adverse fetal outcomes, the underlying mechanisms remain elusive. We hypothesize that maternal obesity dysregulates the regulation of cellular senescence in the placenta and contributes to the dysregulated fetal development during pregnancy.

To test our hypothesis, female CD-1 mice were treated with control (CT) or western diet (WD) for 30 days pre-pregnancy through gestational day (GD) 18.5 with daily weight measurement. Fetal and placental weight of individual fetuses from each litter were measured at GD 18.5. The expression levels of cell proliferation markers and cell senescence markers are examined in GD 18.5 placenta tissues using reverse transcription quantitative polymerase chain reaction (RT-qPCR).

Results demonstrated that placental efficiency is decreased in fetuses from WD dams. Fetal and placental weight distributions were similar between CT and WD groups, with the majority of samples falling within the 11-89th percentile range across both sexes. Although average placental efficiency was lower in the WD group compared to CT, this difference did not reach statistical significance. Gene expression analysis revealed that Cdkn1a (P21) expression level was the primary factor influencing target gene expression, while treatment group and fetal sex had no significant effect. A significant association was observed between Cdkn1a and Tert expression ($p=0.008$), suggesting a relationship between cellular senescence and telomere regulation, whereas no significant relationship was found between Cdkn1a and Casp3, Dkc1, or Polr2a expression.

Sanjana Shahreen – Biochemistry #18

Research Faculty Advisor: Marielle Hegetschweiler, Chemistry and Biochemistry

Unraveling the Molecular Mechanisms of PFAS Binding to Malate Dehydrogenase (MDH)

Per- and polyfluoroalkyl substances (PFAS) are persistent environmental contaminants associated with metabolic and mitochondrial dysfunction; however, the molecular basis of these effects remains poorly defined, particularly regarding direct interactions with metabolic enzymes. Here, we investigate whether PFAS interact with cytosolic malate dehydrogenase (MDH1), a key regulator of cellular redox balance via the malate–aspartate shuttle. We utilize an MDH enzymatic activity assay and 19F NMR spectroscopy to assess effects on activity and potential binding interactions. We show chemical perturbations indicating binding to MDH, along with a clear reduction in NADH turnover by MDH1. Together, these results demonstrate that PFAS can directly associate with MDH1 and modulate its catalytic function by altering the local chemical environment, possibly interfering with substrate access or cofactor interactions. This work provides evidence for enzyme-level PFAS–protein interactions, offering insight into how PFAS may influence cellular redox metabolism and disrupt metabolic function, while establishing a foundation for future studies investigating PFAS–MDH1 interactions.

Meghan Sheehan – Biochemistry, Pharmaceutical Sciences #30

Research Faculty Advisor: Gregory Thatcher, Pharmacology & Toxicology

Vacor Analogues as Inhibitors of NAMPT in Cancer Metabolism

Altered cellular metabolism is a defining feature of aggressive cancers, with many tumors relying on the salvage pathway to produce nicotinamide adenine dinucleotide (NAD+) for sustained adenosine triphosphate (ATP) production and proliferation. Nicotinamide phosphoribosyltransferase (NAMPT), the key enzyme of this pathway, is frequently upregulated and represents a promising therapeutic target. This study evaluates a series of vacor-derived analogues designed to inhibit NAMPT while reducing toxicity associated with vacor itself. Recombinant human NAMPT was expressed and purified, and compound efficacy was assessed with an enzyme activity assay

alongside a fluorescence polarization (FP) displacement assay to measure binding affinity at the enzyme's rear channel.

Twenty-three analogues were screened, and five compounds demonstrated enhanced inhibitory potency and binding affinity compared to vacor. Compound BK-1-149 exhibited the lowest EC50 and IC50 values, indicating strong enzyme inhibition and rear channel binding. These findings suggest that structural modification of vacor can yield more potent NAMPT inhibitors with potentially improved therapeutic profiles. Future studies will assess cellular toxicity and efficacy to further evaluate these compounds as candidates for targeting NAD+ metabolism in cancer.

Sophia Shomper – Biochemistry #41

Research Faculty Advisor: Christopher Frost, BIO5 Institute

Balancing the Blend: Context-Dependent Variation in Green Leaf Volatile Ratios

Green leaf volatiles (GLVs) are plant compounds produced through a conserved biosynthetic pathway and released rapidly following tissue damage. Ecologically, GLVs play critical roles in plant-herbivore and plant-plant interactions, modulating plant priming and indirect defenses. Despite their importance, the extent of variability in GLV emission ratios under different stress conditions remains poorly understood.

This study investigates the variability and reproducibility of GLV ratios in corn (Zea mays) through the lipoxygenase pathway. By analyzing volatile profiles across distinct damage treatments, we investigate the stability and reproducibility of GLV ratios. Volatile emissions were collected following chemical inhibition with phenidone, cryogenic tissue disruption, mechanical wounding, or herbivore feeding by beet armyworm (Spodoptera exigua).

Phenidone-treated tissues produced no GLVs, indicating that these compounds are synthesized immediately in response to damage. Furthermore, among damage treatments GLV profiles varied significantly. Cryogenically disrupted tissue emitted primarily cis-3-hexenal, with no production of downstream products. In contrast, herbivore-induced emissions were dominated by product cis-3-hexenyl acetate. Mechanically damaged plants produced intermediate profiles but showed no production of cis-3-hexenyl acetate, suggesting that it is specifically herbivore induced.

These findings demonstrate that while GLV ratios remain relatively consistent within a given condition, they vary substantially across damage types. This highlights the context-dependent regulation of the lipoxygenase pathway and suggests that GLV composition reflects distinct responses to different forms of stress.

Jordan Singleton – Biochemistry #46

Research Faculty Advisor: Jon Chorover, Agriculture Life & Environmental Sciences

The Iron Redox Continuum: How Hillslopes Shape Soil Chemistry

Although Fe(III)-oxide reductive dissolution to dissolved Fe(II) is recognized as an important redox process in soils, its mechanistic control at the system-scale remains poorly understood. Thus, we asked: What processes are limiting Fe (III) reductive dissolution across hillslopes and depth gradients, and how do these processes contribute to the distribution of Fe at the landscape scale? To investigate this, soils from each depth and position of a hillslope (derived from pinal schist bedrock that underlies a mixed conifer forest) were incubated in an anoxic environment for 30 days. The samples were periodically assessed for Fe (II) release, aqueous total organic carbon content and composition, pH, and CO2 flux. Furthermore, mineralogical data such as extractable iron, clay%, and iron enrichment were recorded.

Results revealed three major patterns: (1) Clay and iron appeared to exhibit a decoupling down the catena, where accumulation of clay-sized particles does not translate to Fe retention. (2) Extractable Fe alone was not a reliable predictor of Fe (II) release, suggesting that mineral protection, hydrologic connectivity, and microbial activity interact to control reductive dissolution at each position. (3) Transport-limited and reaction-limited regimes coexist and vary by element, forming a redox continuum across the ridge-to-toe gradient that challenges traditional single-framework models.

Coltrin Sparks – Biochemistry #22

Research Faculty Advisor: Guang Yao, Molecular & Cellular Biology

Profiling Quiescence

Cellular quiescence is a reversible, non-proliferative state that plays a critical role in development, tissue maintenance, and cancer progression. Many cancer therapies preferentially target actively cycling cells, leaving quiescent populations capable of re-entering the cell cycle and contributing to relapse. To better understand and potentially manipulate quiescence, it is essential to distinguish cell populations not only by cycling status but also by quiescence depth.

This study develops a method to distinguish distinct cell populations with varying quiescence levels within both isolated and mixed samples. Using E23T cells expressing an E2F1 promoter-driven GFP reporter, combined with DNA content staining and autofluorescence measurements, flow cytometry data were visualized using four-dimensional graphing techniques. According to one study, "Senescence-associated-β galactosidase (SA-β-gal) activity" is correlated with an increase in autofluorescence within their cell population (Bertolo et al 2020). This trend is also generally observed in quiescence, supporting the idea that growth to senescence is a continuum. According to another study, "qNSCs (quiescent neural stem cells) display an enrichment of autofluorescence localizing to a subset of lysosomes which can be used as a graded marker of NSC quiescence" (Morrow et al 2024). With all of these markers, including autofluorescence, cells subjected to 2–3 days of starvation, 8–10 days of starvation, and standard growth conditions were compared.

Our findings demonstrate that distinct starvation durations produce separable phenotypic profiles, and that these populations remain distinguishable even within mixed samples. This approach provides a framework for parsing heterogeneous cellular populations and may have future applications in cancer biology and tissue analysis.

Lane Vazquez Luna – Biochemistry #43

Research Faculty Advisor: Brian McKay, Ophthalmology & Vision Science

Interactions of Proteins Linked to Glaucoma

Previous research has suggested that there are at least six genes/proteins linked to glaucoma when a mutation occurs on the gene: TGR6 (myocilin), WDR36, optineurin (OPTN), tank-binding kinase 1 (TBK1), and caveolins 1 (CAV1) and 2. Investigation into age-related macular degeneration (AMD) has shown that Levodopa (LD) activates the GPR143 receptor, which influences the release of myocilin on extracellular vesicles (EVs) through the endosomal pathway. How a mutation on myocilin leads to glaucoma is still unknown, but the interaction with GPR143 illustrates a connection between GPR143, pigmentation or race, and myocilin function. Given that glaucoma exhibits a pigmentation bias similar to AMD, we hypothesize that glaucoma pathogenesis is linked to a defect in the endosomal pathway governed by GPR143. This study explored the interactions of CAV-1, OPTN, and TBK1 with GPR143 and LD activation by examining isolated EVs of porcine trabecular meshwork (TM) and retinal pigment epithelium cells (RPE). EVs were isolated via differential ultracentrifugation, quantified by nanoparticle tracking analysis, and examined for linked proteins via western blot. EV concentration (particles/mL) and mean size (nm) were compared using paired t-tests. OPTN was detected in both TM and RPE EV's regardless of LD activation. LD treatment induced no significant change in EV size (RPE: +2.64%, $p = 0.29$; TM: +5.64%, $p = 0.21$). However, LD activation resulted in a significantly increased EV concentration in RPE cells by 55.50% ($p = 0.010$), while the 19.93% increase in TM cells was not statistically significant ($p = 0.19$). These findings suggest that CAV1 and TBK1 do not share the same endocytic process as myocilin, whereas OPTN may be a potential therapeutic target within this pathway. Further investigation of CAV1 and TBK1 pathways should be explored to develop more personalized POAG diagnostic and treatment tools.

Audrey Winkle – Biochemistry, Molecular and Cellular Biology #33

Research Faculty Advisor: Kerry Cooper, Animal & Comparative Biomedical Sciences

One Health Approach to Studying Campylobacter jejuni Antibiotic Resistance and Virulence

Campylobacter is the leading cause of bacterial gastroenteritis worldwide, responsible for an estimated 550 million cases annually. Most infections are caused by Campylobacter jejuni, with poultry and other birds serving as major reservoirs that results in undercooked poultry as the primary route of human infection. Campylobacter jejuni can cause either watery diarrhea or bloody/inflammatory diarrhea, with strains associated with the latter being more invasive resulting in more severe clinical outcomes. However, very little is known about the role of wild birds in the transmission of C. jejuni strains and human disease overall, in fact, currently, there is very limited research regarding the possibility of Campylobacter associated with wild birds being transmitted to humans. Due to the interconnectedness between animals, humans, and the environment, this subject requires a One Health approach. Using a genomic methods (whole genome sequencing (WGS)) approach, we compared the genetic similarities and potential transmission routes between animals (e.g. cattle, wild bird, etc.) and environmental (e.g. water, soil, etc.) Campylobacter strains associated with a feedlot during the grow out period for beef cattle. Analysis of sequencing results for the three source types (bird, environmental, cattle) allows us to assess the genomic variability, virulence and antibiotic resistance potential of these different Campylobacter strains associated with several different major reservoirs. Together, these novel results will provide key information on the transmission dynamics of C. jejuni associated with a cattle feedlot, and the potential for Campylobacter strains associated with wild birds to potentially cause disease in humans.

Charlie Woodring – Biochemistry #47

Research Faculty Advisor: Laura Meredith, Natural Resources & the Environment

Design and matrix optimization of a biofilter system for hydrogen leak mitigation

Hydrogen (H_2) is an important non-direct pollutant to mitigate for its associated atmospheric effects such as interactions with methane and ozone causing indirect greenhouse gas effects. The creation of a hydrogen biofilter to siphon H_2 from industrial pollution, will help mitigate the increasing hydrogen concentrations therefore reducing the interaction with greenhouse gases. The goal of this study is to find the most optimal organic matrix containing optimized soil and hydrogenases for the biofilter to efficiently absorb the H_2 that would be released into the atmosphere. The biofilter matrix will support and enhance biological processes to siphon these H_2 emissions leading to a decrease in the indirect greenhouse effect associated with atmospheric H_2.

To establish a baseline for the functionality of a biofilter prior to the bioaugmentation with Mycobacterium smegmatis, a known H_2-oxidizing bacterium, different inorganic and organic matrix materials were tested for their ability to support an elevated H_2 adapted NY soil in a closed system at 500 ppm H_2. Using a trace gas analyzer, our optimized matrix containing an inorganic PP sponge, a moisturized coconut husk, and the adapted soil was created and tested within a PVC shell biofilter to result in a hydrogen uptake of -0.55 μ mol/m^2/s. These findings establish a strong baseline for the further bioaugmentation of the matrix with M. smegmatis through a hydrogel to stimulate the oxidizing activity and further optimize the functionally of the biofilter as it pertains to the ability of oxidize H_2 and have a new solution to mitigate H_2 leaks into the atmosphere contributing to a cleaner H_2 economy.

RESEARCH PRESENTATIONS

Elliott Brumbaugh – Biochemistry #55

Research Faculty Advisor: Jianqin Lu, Pharmacology & Toxicology

Development and Characterization of Liposomal Nanomedicines for Colorectal Cancer

Camptothecin (CPT) is a Topoisomerase I (TOP I) inhibitor that stabilizes the TOP I cleavable complex (TOP Icc), resulting in the accumulation of TOP I–DNA–protein crosslinks (TOP I–DPCs) and subsequent DNA damage. Because TOP I is highly expressed in many cancer cells, CPT has demonstrated strong anticancer activity in both preclinical and clinical studies. However, its clinical translation remains limited due to lactone instability, proteolysis, off-target toxicity, and poor pharmacokinetic properties. In previous work, we synthesized a sphingomyelin (SM)-conjugated CPT and formulated it into Camptothesome, which significantly improved drug stability and therapeutic performance. Compared with free CPT and Onivyde, Camptothesome better stabilized the active lactone form, reduced systemic toxicity, prolonged circulation time, enhanced tumor accumulation, and markedly improved anti-colorectal cancer efficacy. Recent studies have further shown that cancer cells can repair TOP1–DPCs via the neddylation-dependent ubiquitin– proteasome pathway, thereby decreasing CPT sensitivity. Pevonedistat (PEV), a NEDD8- activating enzyme inhibitor, can effectively block this repair mechanism. To further enhance CPT efficacy, we developed a synergistic co-delivery liposomal platform based on Camptothesome (C-pevosome) to simultaneously deliver CPT and PEV. This system aims to inhibit TOP1–DPC repair while enhancing DNA damage, enabling optimized drug ratio delivery to tumor sites. Ultimately, C-Pevosome is expected to maximize antitumor efficacy while minimizing off-target toxicity, improving safety, efficacy, and patient compliance in colorectal cancer therapy.

Ava Grote – Chemistry #50

Research Faculty Advisor: Rebecca Schomer, Plant Science

Evaluating the impact of Phosphorus Acid (H3PO3) treatment on beneficial soil microbe viability

Ralstonia Solanacearum is a destructive soil-borne pathogen that causes bacterial wilt in hundreds of critical food crops such as tomatoes and potatoes. This pathogen is categorized as a high priority threat in agriculture, for the European Union, United States, and Canada (Álvarez 2022). Due to its persistence in soil and water, Ralstonia Solanacearum is adaptable and resistant in diverse environments, with climate change possibly contributing to further geographical spread. As the agriculture industry seeks sustainable methods in controlling this soil borne pathogen, phosphorus acid (H3PO3) deems a promising treatment. This study evaluates) the impact of phosphorus acid on the growth and stability of six beneficial soil microbes (Paraburkholderia phytofirmans PsJN, Paraburkholderia bryophila 376MFSha3.1, Herbaspirillium seropedicae SmR1, Acidovorax sp. GW101-3H11, Streptomyces griseus, and Pseudomonas putida F1), to determine if the non-target microbial viability is preserved during treatment. Strains were cultured in a rich media (LB or CPG), rich media + H3PO3, a minimal media (MSB), and minimal media+H3PO3. The growth rates in each of the four conditions were observed over a 21 day period using serial dilution plating. Colonies were counted on the 0, 1st, 2nd, 7th, 14th, and 21st day. Across all tested species, phosphorus acid treatment demonstrated a neutral to positive effect on microbial viability; specifically, the treatment either maintained standard growth cycles or, as seen in the Paraburkholderia species, actively stabilized population density over time in the rich media.

Chloe Ha – Biomedical Science, minor: Biochemistry #52

Research Faculty Advisor: Edward Gelmann, Medicine

Inhibition of DYRK1B Kinase Enhances NKX3.1 Stability in Prostate Cancer

NKX3.1 is a prostate-specific homeobox gene located on locus 8p21 that regulates epithelial proliferation, differentiation, and mediates DNA repair. NKX3.1 is haploinsufficient, and reduced protein levels diminishes tumor suppression, predisposing to prostate cancer development. NKX3.1 has a half-life of approximately 30 minutes, and steady-state turnover is regulated by DYRK1B phosphorylation of serine 186. DYRK1B phosphorylation results in protein polyubiquitination and proteasomal degradation of NKX3.1. Therefore, we hypothesized that DYRK1B inhibition would prolong NKX3.1 half-life and increase levels of this haploinsufficient suppressor in order to reverse the neoplastic process.
We therefore tested a large panel of DYRK1B inhibitors for the property of prolonging NKX3.1 half-life in order to find compounds that could be tested in a preclinical prostate carcinogenesis model.

Kia Hashemi– Biochemistry #67

Research Faculty Advisor: Douglas Loy, Chemistry & Biochemistry and Materials Science & Engineering

Fungal Degradation of Polyacrylamide Hydrogels: Utilization of White Rot Fungi to Degrade Hydrogels, A Novel Study

White rot fungi have been known to have two enzymes of concern present: peroxidase and amidase enzyme. Synthesis of polyacrylamide gels using free radical polymerization chemistry has been utilized in our group in order to monitor potential degradation aspects; does the resulting degradation products award monomeric states or polyacrylate polymers? Degradation products are being monitored with percentage of mass loss and Fourier Transform Infrared Spectroscopy (FTIR) methods. So far, with utilizing white rot fungi, no degradation has been currently observed.

Polina Klishina – Molecular and Cellular Biology, minor: Astrobiology #51

Research Faculty Advisor: Rebecca Schomer, Plant Sciences

The Role of CheY in Chemotaxis and Biofilm Formation in Ralstonia solanacearum

In this study, we investigate the role that homologous CheY receptor proteins play in chemotaxis and biofilm production in the plant pathogenic bacteria, Ralstonia solanacearum. Chemotaxis is the movement of bacteria up or down a chemical gradient. Chemotaxis is regulated by chemoreceptors-proteins embedded in the cell membrane. When chemoreceptors bind to ligands, they trigger a signaling cascade that results in the change in direction of flagellar rotation. Biofilm production is an important protective mechanism for bacteria, which contributes to their survival and in the case of Ralstonia, the bacterium's virulence on plants. In R. solanacearum, chemotaxis directs bacterial movement towards plant roots and biofilm production protects the bacteria against the host immune response and is essential in colonizing the plant xylem. Ralstonia has 3 genes annotated as cheY. that are also known by their locus tags, Rsp1409, Rsp1402, and Rsc0741. Our hypothesis was that the homologous cheY genes are either (1) redundant and all contribute at some levels to the control of chemotaxis, or (2) have diverse roles in controlling motility or biofilm production. To test these hypotheses, we created deletion mutations for each cheY gene (Rsp1409, Rsp1402, and Rsc0741). We then tested the behaviors of these deletion mutations with the following methods: a swim plate assay (to quantify bacterial motility and chemotaxis), growth rate assays (to test if the mutations affected the growth rate over the course of 48h), and a biofilm assay (to quantify each mutant's biofilm production compared to the wild-type). Our results show that only Rsp1402 influences chemotaxis, while Rsc0741 alone alters biofilm production. Interestingly, Rsp1402, which appears to be the main response regulator for chemotaxis, and Rsp1409 are nearly adjacent to each other on the plasmid, while Rsc0741 is located on the

chromosome, which may explain the differences in function of Rsp1402 and Rsc0741. However, the role of Rsp1409 remains unclear. These observations disprove our hypothesis that these three homologous CheY proteins have redundant functionality, but rather that they coordinate different separate processes. In the future, I am creating higher order mutations (double and triple cheY deletions) to investigate if there is any crosstalk among the three CheY homologs.

Nolan Knapp – Chemistry #63

Research Faculty Advisor: Jeffrey Pyun, Chemistry & Biochemistry

Circular Reprocessability of High Sulfur Content Polymer Infrared Optics

Elemental sulfur is a highly abundant byproduct of global petroleum refining, with production exceeding 84 million tons per year. Despite over 50 years of byproduct formation, there remains limited chemical use for elemental sulfur. The Pyun group aims to utilize this surplus sulfur supply alongside organic comonomers to create novel, sulfur-based inverse vulcanized polymers. Among these polymers is Inverse Vulcanized Glass (IVG), consisting of crosslinked polysulfide chains stabilized by norbornadiene organic crosslinkers. IVG's high sulfur content confers significant electronic polarizability, giving rise to a high refractive index, while polysulfide bonds exhibit lower vibrational absorption frequencies, enabling broadband infrared transparency. Alongside these properties, IVG exhibits facile melt reprocessability with minimal degradation of internal cross-linking and IR performance. IVG's reprocessable nature enables circular processability, where IVG optics can be reshaped, repaired, or recycled without loss of function; thereby reducing material waste and extending lifetime. IVG's ease of fabrication, low cost, and reprocessability offer clear advantages to traditional infrared optics, which are typically discarded upon scratching or fracture; furthermore, these characteristics make IVG an ideal material for use in aerospace optics and other robust applications.

Alexander Korolevich – Chemistry #64

Research Faculty Advisor: Jeffrey Pyun, Chemistry & Biochemistry

Photopolymerization for Fabrication of High RI Polymers

Photopolymerization enables rapid curing, low-energy processing, and precise control over polymer network formation, making it attractive for fabrication of advanced optical materials. Through sulfenyl chloride inverse vulcanization (SC-IV) in the synthesis of photocurable monomers we can benefit from the practical advantages of light-driven polymerization as well as developing high refractive index (RI) plastic optics. In this work, sulfur-containing photocurable resins were prepared to study how resin design and curing behavior affect the mechanical and optical properties of the polymers. This work helps guide the development of new sulfur-based photocurable materials for advanced optical applications.

Lee Lawson & Aiden Lindsay – Biochemistry #65

Research Faculty Advisor: Jeffrey Pyun, Chemistry & Biochemistry

Tunable High Refractive Index Inverse Vulcanized Polymers for Cost-Effective Infrared Optics

Infrared (IR) optical materials are critical for imaging and sensing technologies, yet commonly used materials such as sapphire, ruby, and germanium are often expensive and difficult to process. Elemental sulfur, an abundant byproduct of petroleum refining, offers a low-cost alternative feedstock for polymer-based optical materials. Here, we explore inverse vulcanized glass (IVG) systems as high refractive index materials for infrared optics. These polymers are synthesized using elemental sulfur and organic co-monomers, specifically the norbornadiene dimer, enabling scalable and economically viable production. We tune refractive index through compositional control, including selenium incorporation, while evaluating key properties such as infrared transmission, reprocessability,

and fabrication versatility. Our results demonstrate that IVG materials exhibit high refractive indices and strong infrared transmission, with selenium addition further enhancing optical performance. Importantly, sulfur purity is found to significantly influence optical clarity and homogeneity, with higher purity enabling improved transparency and reduced scattering. In addition, these materials are readily reprocessed and fabricated, offering practical advantages over traditional inorganic optics. Overall, inverse vulcanized polymers represent a promising, low-cost alternative for infrared optical applications, with the potential to expand access to high-performance materials through sustainable and scalable manufacturing.

Elise MacKirdy – Chemistry #66

Research Faculty Advisor: Jeffrey Pyun, Chemistry & Biochemistry

Polymer Property Variance: A Look Into An Inverse Vulcanization System

In 2013, the Pyun Group published the first paper of a new polymerization method using sulfur with organic comonomer, termed Inverse Vulcanization (IV). Since then, the sulfur polymer field has expanded and has many applications, from environmental remediation to optical lenses. The main property of the polymer is dependent on which organic comonomer is mixed with sulfur however, sulfur-based systems (> 50 wt%) typically have desirable properties such as increased Infrared transparency, increased refractive index, and high processability. The base system discussed in this poster is termed Inverse Vulcanized Glass (IVG). It is sulfur with a norbornene dimer as the comonomer. The resulting polymer has transparency through the visible and IR spectrums and is processable, making it an ideal candidate to build off of. In this poster, multiple monomer systems will be discussed to create a foundation of desirable properties achieved by system tuning for further development in the inverse vulcanization field.

Cody Mitchell – Chemistry #68

Research Faculty Advisor: Vanessa Huxter, Chemistry & Biochemistry

Solvation Effects on Eosin Y as a Photoredox Catalyst

Eosin Y (EY) is an organic photoredox catalyst used in the desulfurization of thioamides into amides. The mechanism of this reaction has been proposed to be independent of the solvent, however, EY is known to behave differently according to solvent polarity, hydrogen bonding capability, and solubility properties. Using steady state UV-Visible absorption spectroscopy, fluorescence spectroscopy, and time-resolved time-correlated single photon counting (TCSPC), we have found that EY acting as a photoredox catalyst is influenced by both the solvent and the concentrations used under reaction conditions. Our measurements have shown that EY undergoes solvatochromic spectral shifts, modification of the relative intensity of vibronic bands, as well as changes in the lifetime of the excited state in different solvents used for the desulfurization of thioamides into amides. These solvent-dependent effects influence the dynamics of EY acting as a photoredox catalyst, suggesting that the current mechanism may not represent the full reaction.

Kaia Mount – Molecular and Cellular Biology, minor: Chemistry #58

Research Faculty Advisor: Andrew Capaldi, Molecular & Cellular Biology

Ksp1 Fine-Tunes Cellular Stress Responses Within the TORC1 Signaling Network

The Target of Rapamycin Complex 1 (TORC1) is a central regulator of cell growth and metabolism in eukaryotes, integrating nutrient availability and environmental cues to balance anabolic and stress response programs. When nutrients are abundant, TORC1 is active and promotes the synthesis of proteins, lipids, and nucleotides; under stress or starvation, TORC1 is inhibited, leading to reduced translation and activation of adaptive responses. Dysregulation of TORC1 signaling has been implicated in numerous human diseases, underscoring the importance of defining the molecular mechanisms through which TORC1 controls cellular physiology. Despite its broad influence on cell behavior, only a subset of TORC1 downstream effectors has been identified. Previous work identified Ksp1, a poorly characterized kinase, as a TORC1-interacting protein that phosphorylates translation initiation factors, suggesting a

potential role in regulating translation during stress. This project tests the hypothesis that TORC1 signals through Ksp1 to coordinate translational control and cellular stress responses during TORC1 inhibition. To address this, I examined changes in protein abundance, localization, and phosphorylation of downstream targets under nutrient stress and rapamycin treatment. I found that Ksp1 selectively regulates translation by promoting the clearance of eIF4G2 and the RNA helicase Ded1, while eIF4G1 remains unaffected, indicating that Ksp1 fine-tunes rather than globally represses translation. Additionally, Ksp1 does not mediate PKA-dependent translational changes, supporting pathway specificity. Analysis of Ksp1 regulation revealed that its protein levels and cytoplasmic localization remain stable during TORC1 inhibition, suggesting regulation occurs through functional modulation rather than changes in abundance or localization. I additionally show that Ksp1 modulates TORC1 activity itself: loss of KSP1 impairs TORC1 inactivation during nitrogen starvation and disrupts TORC1 reactivation following rapamycin treatment, while having no effect under glucose deprivation. Together, these findings define Ksp1 as a downstream TORC1-dependent kinase that both fine-tunes translational responses and contributes to feedback regulation of TORC1 signaling during nutrient stress. This work provides new insight into how TORC1 achieves specificity in coordinating growth and stress adaptation and establishes a framework for understanding conserved regulatory mechanisms across eukaryotes.

Marley Novak – Chemistry #62

Research Faculty Advisor: M. Leandro Heien, Chemistry & Biochemistry

Fast-Scan Cyclic Voltammetry for Single Electrode Methods of Measuring Dopamine Release

Dopamine is a neurotransmitter that plays a critical role in the central nervous system. It helps regulate movement, cognition, motivation, and reward pathways. Imbalances in dopamine levels are associated with neurological disorders such as Parkinson's disease and schizophrenia. Accurate measurement of dopamine could improve understanding of these conditions and support therapeutic development. Dopamine has two signaling modes: tonic and phasic. Tonic signaling refers to slow, baseline-level activity, whereas phasic signaling refers to transient, rapid bursts of activity. The current electrochemical techniques measure these two signaling modalities separately using carbon-fiber microelectrodes and fast-scan cyclic voltammetry (FSCV) for phasic signaling, or fast-scan controlled adsorption voltammetry (FSCAV) for tonic signaling. The goal here is to enable measurement of both tonic and phasic dopamine levels using a single electrode. The two modes are interconnected, so measuring their dynamic is important. This is approached by applying FSCAV in vitro, then switching to FSCV during dopamine injection. One focus was on evaluating how effectively the FSCAV waveform detects rapid phase changes. If proven effective, the technique could be optimized and extended to in vivo applications.

Jaden Peters – Chemistry, Pharmaceutical Sciences #60

Research Faculty Advisor: M. Leandro Heien, Chemistry & Biochemistry

Measuring Tonic Histamine Signals Using Carbon Fiber Electrodes

Histamine is a biomolecule that is released all over the body as an immune response. It is biochemically made from L-histidine and binds to 4 different histamine receptors in the body. In the brain, histamine is unique as it acts as a neurotransmitter. However, there are issues with gathering data with histamine. Measuring histamine causes it to clump together into a giant mass, and the voltage needed causes over oxidation of the probes. Therefore, to study more about histamine and its role in the brain, there needs to be a more efficient method of measuring histamine readings.

Kristen Porter – Biochemistry #59

Research Faculty Advisor: Thomas Tomasiak and Darpan Raghav, Chemistry & Biochemistry

Functional Analysis of Phosphorylation Sites Governing Cdr1-Mediated Drug Efflux in Candidozyma auris

In recent years, the medical community has grown increasingly alarmed by the rise of Candidozyma auris. Its impact is particularly pronounced in hospitals and long-term care facilities across the United States. Recent statistical data indicates a sharp increase in reported cases, rising from its first identification in 2016 to 6,304 new clinical cases in 2024. The persistence of C. auris is partly attributed to its ability to survive on dry surfaces for extended periods, as well as its resistance to commonly used antifungal agents. One of the key mechanisms underlying this resistance is the overexpression of the Candida drug resistance protein 1 (Cdr1), an ABC class transporter that actively expels antifungal compounds from the cell. Previous studies in Candida albicans, a closely related species, have shown that N-terminal phosphorylation can influence Cdr1 function. Building on this, the present study employed phospho-proteomics coupled with mass spectrometry to identify novel phosphorylation sites in wild-type C. auris Cdr1. Site-directed mutagenesis was used to substitute phosphorylated residues with alanine, and the functional consequences of these mutations were evaluated using cell survival assays in the presence of fluconazole and Nile red transport assays. Results from our MS analysis identified nine novel phosphorylation sites in C. auris Cdr1. Functional characterization of these mutants demonstrated that individual phosphorylation events are functionally redundant and play a minimal role in regulating substrate transport activity and that a total loss of phosphorylation may be necessary to induce a measurable shift in the transporter's conformational equilibrium or significantly attenuate its catalytic efficiency. These findings provide new insights into the molecular regulation of drug efflux and may support the development of improved strategies to combat antifungal resistance in this emerging pathogen.

Laura Serikova – Biochemistry, Molecular & Cellular Biology #57

Research Faculty Advisor: Ingmar Riedel-Kruse, Molecular & Cellular Biology

Analyzing colloidal self-assembly patterns formed through synthetic cell-cell adhesion in E. coli during growth

Multicellularity of organisms on Earth enabled them to perform complex tasks through adhesion, division of labor, organization, and communication through various signaling pathways. Such property puts these systems above the technologies of today, owing to their ability to synthesize their own chemicals, self-replicate, and specialize. The development of a synthetic, genetically encoded inducible cell-cell adhesion toolbox with membrane-displayed nanobody-antigen pairs enabled the study of the biophysical characteristics of the self-assembly patterns of these microscopic colloidal structures (“core-shell aggregates”), revealing their ability to form different patterns by controlling the affinity and strength of the adhesins. However, little is known about the colloidal self-assembly patterns of these aggregates during growth. We address this gap by changing certain parameters, such as cell-to-cell ratios, adhesin type, cell density, and cell form factor, and growing the core-shell aggregates by reintroducing them into the growth medium. Confocal microscopy was used to image core-shell aggregates formed by mixing cells with complementary adhesins using the E. coli display system. The induced cell mixtures were imaged at 30-minute intervals for a total of 3 hours and were quantified using the ImageJ image processing program. Growth experiments showed visual changes to aggregates in size, and further quantitative analysis revealed a gradual growth of these aggregates over time. Understanding the growth patterns of these colloidal self-assembly structures holds promise in the field of ELM, which could utilize these building blocks in designing self-replicating systems with the ability to biosynthesize and so much more.

Ria Siddaiah – Molecular & Cellular Biology, minors: Biochemistry & Business Administration #56

Research Faculty Advisor: Pascale Charest, Molecular and Cellular Biology

Effects of wild-type versus oncogenic Ras on mTORC2 signaling

Directed cell migration is essential for embryonic development, maintaining multicellular organisms, and immune responses. Dysregulation of this process can contribute to diseases like cardiovascular disorders and cancer

metastasis. One key molecular player is the mechanistic Target of Rapamycin Complex 2 (mTORC2), which is implicated in abnormal cancer cell migration and invasion. The small GTPase Ras, mutated in approximately 30% of cancers, drives hyperactivation, increased proliferation, and migration of tumors in part through its association with the mTORC2 signaling pathway. However, its exact role in regulating mTORC2 to facilitate cell migration requires further investigation. To address this, we stably expressed wild-type and oncogenic Ras in MCF10A breast epithelial cells. We hypothesize that wild-type and oncogenic mutant Ras differently regulate mTORC2 activity and its function in the context of directed cell migration. To test this, we are conducting time-course EGF stimulation assays, 2D and 3D migration and invasion assays, and immunofluorescence microscopy. Together, these studies will define the relationship between Ras signaling and mTORC2 signaling.

Joanna Vargas – Chemistry #69

Research Faculty Advisor: Vlad Kumirov, Chemistry & Biochemistry

Measuring Methyl Exchange Rates in 4-X-N,N-dimethylbenzamides

This poster presents the analysis of different substituent groups on the compound 4-X-N, N-dimethylbenzamides. This was done through NMR Spectroscopy, more specifically using the CPMG Pulse sequence, where the exchange rate is measured by calculating the T2 relaxation. This was done with the purpose of comparing the results to the experimental origins of the categorization of different substituents, such as electron-withdrawing groups or electron-donating groups, which are widely used in organic chemistry textbooks. The experimental results presented a different categorization of the substituents of the halogens explored.

Dylan Weaver – Biochemistry #70

Research Faculty Advisor: Michael Taylor, Chemistry & Biochemistry

Perfluoro-alkyl Quinolinium Cation as a Radical Photocage for Post-Translational Modification

Described here, the synthesis of nonafluorinated quinolinium sulfonate ester then its utilization for post-translational modification of biomolecules via photodecaging. Upon absorption of visible light, the sulfonate ester is decaged via photolysis creating an electrophilic C-centered nonafluoro radical that is trapped by π-nucleophiles in biomolecules. Further experiments with this nonafluorinated quinolinium sulfonate ester involve post-translational modification of nanobodies and the study of protein-protein interactions with further implications in drug discovery.

Mandy Wu – Pharmaceutical Sciences, minor: Biochemistry #54

Research Faculty Advisor: Tally Largent-Milnes, Pharmacology

Is voriconazole an effective reversal treatment for photophobia?

Photophobia, or sensitivity to light, is a debilitating symptom commonly associated with migraine. Women are three times more likely to experience migraines and exhibit worser symptoms than men are. Migraines interfere significantly with productivity and quality of life; therefore, it is important to find targeted, safe, and effective treatments for photophobic behaviors related to migraine. Voriconazole is a broad-spectrum triazole used in the treatment of serious and invasive fungal infections. Voriconazole has been shown to inhibit TRPM3 channels, which are involved in nociceptive and sensory signaling, and to alter retinal activity, making it a candidate for modulating ocular pain and photophobia. In this study, we investigated whether voriconazole attenuates photophobic behaviors using a rat model of migraine with aura. Sprague-Dawley rats (N=20; female; 8 weeks-of-age) received an injection of potassium chloride (KCl) through a surgically implanted dural cannula to induce cortical spreading depression (CSD). An injection of either voriconazole (100 mg/kg, ip) or vehicle was given to rats 30 minutes after KCl administration. Light aversive and dark seeking behaviors were evaluated before and after (t=15, 60, 120, and 180 mins) KCl administration using a three-chamber light-dark assay. KCl-induced CSD resulted in increased light aversion and increased time spent in the dark chamber. There was no significant difference in light aversion and dark seeking behaviors between voriconazole and vehicle groups. These data indicate that voriconazole does not attenuate photophobic behaviors, suggesting differences between in-vivo and in-vitro models.

Jeffrey Yao – Biochemistry #53

Research Faculty Advisor: Shaowen Bao, Physiology

Dynamics of Microglial Morphology and Activation in the Auditory Cortex Following Noise Trauma

Disorders of central auditory processing are often linked to hearing loss. Previous studies have shown that noise exposure induces neuroinflammation in the auditory cortex and pathways. In this study, we investigated morphological changes in microglial cells in rodent models with noise-induced hearing loss. Our findings reveal that microglial activation occurs as early as one day after noise exposure, characterized by enlarged cell somas and reduced territorial coverage, an activated microglial state. This early activation may contribute to neuronal inflammation and the subsequent development of central auditory processing deficits and associated cognitive dysfunction.