Minority Health Disparities (MHD)

Coordinator: Holly Lopez

Sponsors: The University of Arizona, the University of Arizona Graduate College, Western Alliance to Expand Student Opportunities (WASEO), Building Undergraduate Infrastructure Leading to Diversity: Southwest Consortium of Health-Oriented Education Leaders and Research Scholars (BUILDing SCHOLARS)-University of Texas, El Paso.

MINORITY HEALTH DISPARITIES SUMMER RESEARCH PROGRAM (MHD)

SHERLEE ALVAREZ

Functional Genomics of the Compass-Like Complex in Pancreatic Neuroendocrine Tumor Malignancy

University of New Hampshire, Biochemistry, Molecular and Cellular Biology
Mentors: Brenna Rheinheimer and Ron Heimark

Abstract

Pancreatic neuroendocrine tumors (PanNETs) are the second most common epithelial neoplasm of the pancreas with a mortality rate of 60%. The incidence of PanNETs has increased through routine diagnostic imaging; however, our current understanding of the molecular pathology of PanNETs is insufficient to standardize clinical management of the disease. Non-functional PanNETs grow silently; therefore, patients often present with an asymptomatic mass or symptoms of abdominal pain. Though surgical resection is the treatment of choice for PanNETs, many patients present with unresectable tumors or substantial metastatic disease, rendering the current treatment plan ineffective. Currently, there is inadequate information of PanNETs to predict prognosis of patients diagnosed with these tumors or to develop new diagnostics and treatments for improvement of disease management.

Previous genomic studies have shown four common genetic mutations in PanNETs including the gene MEN1. MEN1 is a tumor suppressor gene consisting of 10 exons and is located on chromosome 11q13. The MEN1 gene encodes the protein Menin, a 610-amino acid protein with two nuclear localization signals on the C-terminal end to allow for localization in the nucleus. Menin acts like a platform to regulate gene transcription by coordinating chromatin remodeling and is one component of the MLL/SET1-like histone methyltransferase complex. Though MEN1 is mutated within 44% of PanNETs, and analyses have revealed candidate loci for genes involved in the progression of PanNETs, MEN1 has not been examined through functional analyses. Therefore, we propose that PanNETs containing MEN1 mutations are more aggressive tumors that lead to decreased overall survival.

CLAUDIA AYALA

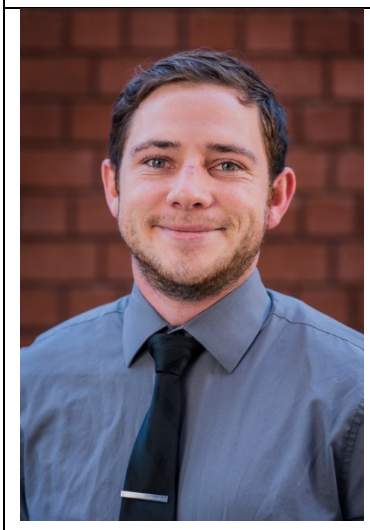
A Model System for Cells Deficient in Mitochondrial MAVS

University of Texas, El Paso, Biology
Mentors: Dominik Shenten and Marvin O'Ketch

Abstract

The detection of microbial ligands by pattern recognition receptors (PRRs) and the resulting inflammatory response is a critical regulatory step for the generation of adaptive immune responses by T and B cells. The function of membrane-bound PRRs such as Toll-like receptors in this process is clearly recognized. In contrast, the role of cytosolic PRRs in the regulation of adaptive immunity is still poorly understood.1 Rig-I-like Receptors (RLRs) are a family of PRRs that recognize microbial RNAs in the cytosol. RLRs rely on the signaling adaptor MAVS (also called IPS-1) for the activation of downstream transcription factors that drive the transcription of pro-inflammatory cytokines and interferons, thereby producing an antimicrobial response.

MAVS localizes to two organelles, namely mitochondria and peroxisomes. Previous work has shown that the mitochondrial and peroxisomal pools of MAVS induce

	overlapping but distinct sets of genes including pro-inflammatory cytokines and interferons (IFN). For example, mitochondrial MAVS induces type I IFNs, while peroxisomal MAVS induces type III IFNs.2 There are also kinetic differences between the two pools of MAVS that depend on the cell type involved. We and others have previously shown that MAVS is important for the B cell-driven antibody response to West Nile Virus (WNV).3 However, the relative functions of mitochondrial and peroxisomal MAVS in the regulation of this response is currently unknown. Here, we establish an in vitro system that allows us to differentiate between the mitochondrial and peroxisomal pools of MAVS during WNV infection. This system will also form the basis for subsequent generation mice carrying a deletion of mitochondrial MAVS in order to dissect the role of mitochondrial and peroxisomal MAVS in vivo.
	**DAMIESHA BRYANT** *Analysis of Mice Kidney Reveals an Effect of High Fat Diet on Protein Expression* Philander Smith College, Biology Mentor: Lawrence Mandarino **Abstract** As obesity remains prevalent in the western world, it is critical to understand how excess visceral adiposity alters the function of other organs. Obesity increases the risk of developing degenerative conditions such as hypertension and Chronic Kidney Disease. Dysfunctions in the regulation of the renin-angiotensin-aldosterone system has been shown to promote renal disorders. High fat diet induced obesity has also been shown to lead to hypertension due to both excessive visceral adipose tissue surrounding the kidneys and the elevation of renal tubular sodium reabsorption. Not only does a high fat diet promote hypertension, but also it encourages an increased fatty acid oxidation and decreased fatty acid synthesis. In this study, using mass spectrometry based proteomic analysis, we evaluated the effects of high fat diet (HFD) induced obesity on the mouse kidney protein expression to gain insight into how obesity may lead to Chronic Kidney Disease.
	**BRENT CARRILLO** *Genome-Wide Association of Vasodilator Drug Response in Pulmonary Arterial Hypertension* New Mexico State University, Chemistry Mentor: Jason Karnes **Abstract** Introduction: Pulmonary arterial hypertension (PAH) is a deadly disease characterized by an increased pressure in pulmonary vasculature. Vasodilators are used to treat PAH but are only effective in some individuals suggesting that genetic differences exist. The objective of our study was to identify genetic influences on vasodilator drug response in PAH. Methods: Samples from PAH cases were collected from over 28 US institutions. Mean pulmonary arterial pressure (mPAP) was measured at rest and after acute vasodilator

	administration. Patients were considered vasodilator responsive if mPAP decreased by more than 10 mm Hg with preserved cardiac output. Logistic regressions were performed in Caucasian and adjusted for age, gender, and principle component 1. Genotyping was done using the Illumina GWAS platform OMNI5 Results: After quality control (removing individuals with missing data) a total of 23 PAH cases and 411 controls remained. Frequency and genotyping pruning resulted in 2,548,842 SNPs. Several associations were identified above the suggestive association threshold ($p=5x10^{-6}$) in chromosomes 1, 3, and 9. Clustering of associations were observed in chromosome 6, falling just below the suggestive association threshold. QQ-plots suggest that population stratification was adequately controlled Conclusion: The results identify several genetic variants that are potentially associated with vasodilator responsiveness in PAH. These results require replication of signals in an independent cohort.
	**HASSAN-GALAYDH FARAH** *Brainstems, Breathing & Babies: How Developmental Nicotine Exposure Impacts the Respiratory System* The University of Arizona, Physiology Mentor: Ralph Fregosi **Abstract** Developmental Nicotine Exposure (DNE) is defined as the administration of nicotine to prenatal organisms and is shown to have a profound impact on the developing respiratory system. By investigating a number of histological methods, immunohistochemistry provides a detailed illustration of specific receptors within the Hypoglossal Motor Nucleus (XIIMN). The XIIMN resides in the brainstem and it was collected from postnatal rats ranging from neonatal age and onward. Once isolated, the brainstem was sectioned into 7-15 slices ranging between 70-100µm in thickness and mounted onto superfrost slides. The slides then underwent two separate steps to illustrate structure: Nissl staining for macroillustration and Immunohistochemistry for specific structures such as the XIIMN. Throughout the past 10 weeks in the Fregosi lab, I have successfully managed to achieve consistent and clear Nissl stains as well as a better understanding of IHC protocol. With more time, additional results from IHC analysis can be provided.

GISSELLE GONZALEZ

Poly (Lactide-co-glycolide) + Human Epidermal Growth Factor-containing Soymeal Electrospun Scaffolds

The University of Arizona, Biomedical Engineering
Mentors: Marv Slepian and Daniel Palomares

Abstract

Diabetes is on the rise worldwide, affecting over 422 million people. The consequences of Diabetes, e.g. accelerated arterial disease and peripheral neuropathy, increase risk of foot ulceration, infection and potential amputation. A need exists for therapeutics able to locally deliver agents – e.g. biologics, Epidermal Growth Factor (EGF) – to treat ulceration. Fiber/mesh constructs formed through electrospinning are effective for biologic delivery. Genetically modified soybeans, producing human EGF, have the potential to serve as therapeutic source materials for fibers. We hypothesized that soybeans engineered to produce human EGF provide usable material for electrospinning into therapeutic fiber constructs. We examined the efficacy of soymeal + polymer blends to be electrospun into fibers to form scaffold constructs, and physically characterized soymeal-PLGA composite fibers over a range of soy concentrations.

Soybeans engineered to produce human EGF1 were manually ground to a meal (powder) and added to 7.5% w/v poly(L-lactide-co-glycolide) acid (PLGA) (ratio M/M% = 95/05) in dichloromethane. Soymeal-PLGA blends were electrospun and fibers were characterized via SEM and contact angle. Soymeal-PLGA was successfully blended and electrospun into fibers, yielding scaffold meshes. Increasing soy concentration resulted in fibers with increasing irregularities or “beads,” with thinner fibers associated to beading. With increasing soymeal concentration, scaffold hydrophilicity more closely approximated soymeal hydrophilicity.

Conclusion: PLGA + soymeal composite scaffolds are readily formed and robust. These constructs warrant further development as cutaneous drug delivery therapeutics for diabetic foot ulceration. Future work will focus on optimization of parameters for electrospinning, i.e. working distance, voltage, polymer MW; and will examine the biocompatibility and cell proliferative potential of polymer + hEGF soymeal constructs in vitro utilizing epidermal cell cultures.

CHRISTINA HARRIS

Education and the Public Health Workforce

The University of Arizona, Public Health
Mentor: Doug Taren

Abstract

It is well established that, in general, public health employees score low regarding budgeting skills and policy development and relatively high regarding communications. This study examines whether the amount of college education impacts public health employees' performance in these three domains. For 763 entry level employees (Tier 1 employees) and 325 middle manager employees (Tier 2 employees) from 9 Arizona public health departments, the study compared education level to self-rated performance on budgeting, policy and communications. Questionnaire items were on rated on a Likert-type scale, ranging from 1 to 5, where 1 was "Not at all competent", 3 was "Competent", and 5 was "Expert". Communications had 7-9 items (depending on Tier), Financial Planning had 8-9 items and Policy Development had 15 items. A mean score ≥ 3 was counted as competent. The questionnaire items came from the Council of Linkages 2010 competency assessment instrument. The results indicated that each of the skills was positively associated with amount of education; and this was the case for both Tier 1 and Tier 2 employees. While amount of education was also positively associated with financial planning and policy development, the amount of the association differed by Tier. More specifically, the association of education with finance and policy development was greater for Tier 2 employees with a Bachelor's degree or a post-baccalaureate degree than for Tier 1 employees with the same amount of college education. Using the same process, the relationship between education level and performance can also be investigated in other domains.

DANIELLE JOHNSON

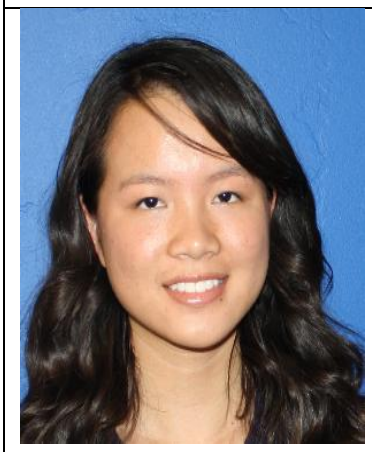
Nuclear Localization of the ErbB Protein Family in Triple-negative Breast Cancer Cells

Tennessee State University, Chemistry & Biochemistry
Mentors: Joyce Schroeder and Dawn Geiser

Abstract

In normal breast tissue, the Epidermal Growth Factor Receptor (EGFR/ErbB1), a member of the ErbB protein family, undergoes several regulatory mechanisms with ligand binding (e.g., EGR and TGF□) such as, dimerization, phosphorylation, endocytosis, endosomal recycling, ubiquitylation, and lysosomal degradation which limits the expression and signaling of EGFR on the cell membrane. However, in triple negative breast cancer (TNBC), EGFR has been shown to have increased expression with altered subcellular localization and trafficking to the nucleus. Alterations in EGFR regulation and nuclear localization increases transcription of genes that promote cellular proliferation which leads to poor prognosis with TNBC diagnosis. Our previous research has shown that a mucin protein expressed on the cell membrane, MUC1, is a key contributor to the altered subcellular localization and function of EGFR, which requires further investigation of MUC1 interaction with the ErbB protein family. We

	investigated the effect of MUC1 knockout (shMUC1) in a TNBC cell line, BT20, on nuclear localization of EGFR and two other ErbB proteins, ErbB2 (HER2/Neu) and ErbB3 (HER3). We treated overnight serum-starved shMUC1 BT20 cells and control BT20 cells expressing MUC1 (shControl) with ligand (20 ng/ml EGF) to study the internalization of the ErbB proteins in TNBC cells. We demonstrate through immunofluorescence, differential detergent fractionation, SDS-PAGE, and immunoblotting the localization of ErbB proteins. In the future, we will examine potential gene targets of ErbB proteins in the nucleus that regulate cellular proliferation utilizing ChIPseq technology.
	**Dominique Lund** *Using a Peripherally-Restricted Cannabinoid to Treat Cancer-Induced Bone Pain* The University of Arizona, Pre-physiology Mentor: Todd Vanderah **Abstract**
	**MELISSA MARTINEZ** *Testing the Specificity of AAV-CaMKII-Alpha-eNPAC 2.0 in the Prefrontal cortex and Hippocampus of the Rat* California State University, Northridge, Public Health Mentor: Jean-Marc Fellous **Abstract** The Prefrontal Cortex (PFC) and Hippocampus (HC) are two different regions found in the rat brain. The PFC is involved in decision making and working memory, among other functions. The HC is involved in episodic memory and spatial learning. In previous optogenetic experiments, we have injected in PFC and HC a virus (AAV-CaMKII-Alpha-eNPAC2.0) thought to be expressed specifically in neurons containing CaMKII-Alpha, a protein kinase involved in spatial learning and long term potentiation. However, this specificity has not been demonstrated yet. The first aim of this study was to detect CaMKII-Alpha and GAD67, an enzyme that synthesizes GABA in neurons of the HC and PFC. The second aim was to assess the specificity of the expression of AAV-CaMKII-Alpha-eNPAC2.0 in CaMKII-Alpha positive neurons in the HC and PFC. Using three different antibodies, CaMKII-Alpha Monoclonal 6G9, Monoclonal Anti-Glutamic Acid Decarboxylase GAD67, and Goat-Anti-Mouse IgG (H+L)-Alexa Fluor 405, we were able to detect positive immunoreactivity staining for CaMKII-Alpha or GAD67 in different population of neurons in the HC and PFC. Our preliminary results also show that, in both regions of interest, CaMKII-Alpha, but not GAD67 positive neurons expressed AAV-CaMKII-Alpha-eNPAC2.0. This study can be applied to assess the viral

	expression in CaMKII positive neurons in different cortical and subcortical brain regions such as the thalamus, visual, and somatosensory cortex.
	**MARIAH MURRAY** *Interaction of PRL1 and SH3* University of Illinois, Applied Health Science: Community Health Mentors: Wolfgang Peti and Senthil Ganesan **Abstract** The purpose of our experiment is to express and purify the proteins SH3 and PRL1 in preparation for NMR analyzation of the molecular basis and characterization of srcSH3-PRL1 interaction. SH3 Domain (SRC Homology 3) and PRL1 tyrosine phosphatase are two proteins actively involved in dysregulation leading to diseases. SH3 Domain is a small protein domain of 60 amino acid residues that interacts with tyrosine kinases. The domains are found in proteins of signaling pathways which regulate the cytoskeleton. Approximately 300 SH3 domains are found in proteins encoded in the human genome. The Rous sarcoma virus contains the v-src gene. The Src protein has protein tyrosine kinase activity which is essential for its oncogenic potential. PRL1 tyrosine phosphatase is one of three closely related small protein-tyrosine phosphatases, which are characterized by C-terminal addition of prenyl residues. They contain a C-terminal CAAX motif as the signal for protein farnesylation. They are involved in the regulation of cell increase in numbers and transformation. PRL1 mRNA is over expressed in human tumor cell lines. Mutant of PRL1 results in mitotic defects and over expression of PRL-1 in epithelial cells results in a transformed phenotype and the transfected cells are able to form tumors, tumorigenesis. Both proteins are expressed and purified before they can be analyzed using NMR. Protein Expression is the manipulation of gene expression in an organism such that it expresses large amounts of the recombinant gene to build the protein. Protein purification is a series of methods used to isolate a protein from a complex. This is vital for the study of the function, structure, and interactions of the proteins of interest. These methods are used to analyze the complex formed between SH3 and PRL1.
	**VY NGUYEN** *Artificially Engineered ELP-AMP Proteins for Broad-Spectrum Antimicrobial Materials* The University of Arizona, Biomedical Engineering Mentor: Minkyu Kim **Abstract** Antibiotic resistance is a rising concern worldwide because pathogenic bacteria continually evolve to develop resistance to available antibiotics. Each year in the U.S., two million people become infected with antibiotic-resistant bacteria, 23,000 of whom die of their infections. More than 2,600 antimicrobial peptides (AMPs) have been discovered and characterized, and positive *in vitro* susceptibility testing against microorganisms has made AMPs into promising antibiotic alternatives. However, AMPs tend to degrade in the blood stream; thus, high AMP dosage is necessary for

effectiveness, but the high dosages can be histotoxic and cytotoxic. Recent studies have found that tethered AMPs on nanoparticles can reduce AMP degradation, which can be lower the AMP dosage. It is currently unfeasible for real-world implementation of AMP-incorporated nanoparticles because of the high cost of producing AMPs, as well as immobilization processing between AMPs and nanoparticles that typically leads to waste of unbound expensive AMPs. To overcome the present challenges, we developed an artificially engineered protein, genetically composed of AMP and elastin-like polypeptide (ELP), a thermoresponsive intrinsically disordered protein that can self-assemble into nanoparticles at body temperature and that can potentially prevent AMP degradation. AMP LL37 from the human innate immune system is selected because of their ability to kill a broad range of bacteria, viruses and fungi by disrupting cell walls. Initial antimicrobial susceptibility testing with ELP-LL37 has shown promising results. Our artificially engineered ELP-AMP protein is expected to effectively treat antibiotic resistant infections with low AMP dosage.

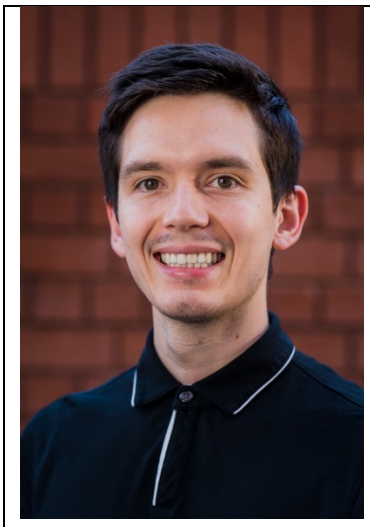
JANET OLIVAS

Zika Virus Replication in Humans is Dependent on Cholesterol Ester Synthesis

University of Texas, El Paso, Cellular and Molecular Biochemistry
Mentors: John Purdy and Liz Dahlman

Abstract

Zika virus (ZIKV) is an enveloped positive-sense RNA *Flavivirus.* A 2016 outbreak quickly spread internationally, mostly through mosquito transmission. ZIKV causes neurological disorders, such as Guillain-Barre syndrome, and birth defects including microcephaly. Prior to 2016, little was known about ZIKV and very few studies examined how it interacts with the human host. Although many labs have recently examined ZIKV-host interactions, most have not considered how ZIKV interacts with the host's lipid metabolism. In our lab, we have found that ZIKV infection increases cholesterol and cholesterol ester levels. Cholesterol esters consists of a cholesterol with a hydrocarbon fatty acid tail attached. The fatty acid can be various lengths and degree of saturation (carbon-carbon doubles). The host enzyme sterol o-acyltransferase 1 (SOAT1) catalyzes the formation of cholesterol ester. We hypothesize that ZIKV alters cholesterol and its esters in order to replicate. To test our hypothesis, we treated ZIKV infected cells with a SOAT1 inhibitor called Avasimibe. Our preliminary data demonstrates that SOAT1 is required for ZIKV infection, providing support for our hypothesis. A deeper understanding of ZIKV-host interactions can lead us to identifying possible antiviral treatments and therapies.



MATTHEW RANDAL

Proliferation Rates of K5+ Cells and Amylase+ Cells Following Radiation Damage and Restoration

University of Texas, San Antonio, Biochemistry
Mentor: Kirsten Limesand

Abstract

The purpose of our experiment is to express and purify the proteins SH3 and PRL1 in preparation for NMR analyzation of the molecular basis and characterization of srcSH3-PRL1 interaction. SH3 Domain (SRC Homology 3) and PRL1 tyrosine phosphatase are two proteins actively involved in dysregulation leading to diseases. SH3 Domain is a small protein domain of 60 amino acid residues that interacts with tyrosine kinases. The domains are found in proteins of signaling pathways which regulate the cytoskeleton. Approximately 300 SH3 domains are found in proteins encoded in the human genome. The Rous sarcoma virus contains the v-src gene. The Src protein has protein tyrosine kinase activity which is essential for its oncogenic potential. PRL1 tyrosine phosphatase is one of three closely related small protein-tyrosine phosphatases, which are characterized by C-terminal addition of prenyl residues. They contain a C-terminal CAAX motif as the signal for protein farnesylation. They are involved in the regulation of cell increase in numbers and transformation. PRL1 mRNA is over expressed in human tumor cell lines. Mutant of PRL1 results in mitotic defects and over expression of PRL-1 in epithelial cells results in a transformed phenotype and the transfected cells are able to form tumors, tumorigenesis. Both proteins are expressed and purified before they can be analyzed using NMR. Protein Expression is the manipulation of gene expression in an organism such that it expresses large amounts of the recombinant gene to build the protein. Protein purification is a series of methods used to isolate a protein from a complex. This is vital for the study of the function, structure, and interactions of the proteins of interest. These methods are used to analyze the complex formed between SH3 and PRL1.

CHYANN RICHARD

Altered Sleep Spindles in a LRRK2 Mouse Model of Parkinson's Disease

California State University, Long Beach, Psychology/ Neuroscience
Mentor: Stephen Cowen

Abstract

Altered Sleep Spindles in a LRRK2 Mouse Model of Parkinson's Disease
Parkinson's Disease is a neurodegenerative disorder found in 1-2% of the population 65 years and older and results from the substantial loss of dopaminergic neurons in the substantia nigra. Features of this disorder includes akinesia (impairment of movement initiation), bradykinesia (amplitude and velocity reduction of voluntary movements), muscular rigidity, and tremors. Aside from these primary motor impairments, non-motor features of the disease include sleep disturbances, depression and anxiety, cognitive impairment, and autonomic dysfunction. The most common genetic form of Parkinson's Disease is the protein mutation, LRRK2 G2019S. In a previous study using an alternative bacterial artificial chromosome mouse model, LRRK2 mice showed increase power in sleep spindles, cortical

	oscillations linked to memory consolidation. Using a newer, more specific LRRK2 knock-in mouse model, this study aims to investigate the changes in sleep-related brain activity in LRRK2 mice as well as investigate the effects of a LRRK2 inhibitor on these animals. In this study, mice will undergo 2 learning experiences; an environmental enrichment task and a rotarod task. It is hypothesized that LRRK2 knock-ins will show a difference in local field oscillatory activity (specifically LFP and EcoG) compared to wild type mice and that this difference may be rescued following LRRK2 inhibitor.
	**ANGELA RIVERA** *Purification of Pfu DNA Polymerase to Confirm Bacterial Mutations* The University of Arizona, Biochemistry, Molecular and Cellular Biology Mentor: Michael Johson **Abstract** Taq DNA polymerase is currently purchased for laboratory use. To mitigate these costs, the Johnson laboratory has decided to make a different DNA polymerase from Pyrococcus furiosus termed Pfu. If functional, Pfu will replace purchasing Taq polymerase. To express Pfu polymerase, we transformed a plsmid containing the gene for the protein in BL21 gold Escherichia coli cells for recombinant protein expression. Following the expression of Pfu in the E. coli, the cells were lysed via sonication, and the protein was purified via a nickel affinity chromatography column. As an added purity step, we also ran Pfu through a column that separates objects by size. We then tested the enzyme in a polymerase chain reaction, alongside Taq for a positive control, to verify the functionality of the Pfu for DNA amplification. Once we confirmed that Pfu was able to amplify DNA, we used it to confirm bacterial mutations in Streptococcus pneumoniae DNA. These results suggest that the Pfu is a viable polymerase for future use in laboratory.
	**ERIKA RODRIGUEZ-GUZMAN** *A Systematic Review of Physical Activity and Diet Interventions for Weight Management in Hispanic Adult Males* The University of Arizona, Public Health, Italian Language and Literature Mentor: David Garcia **Abstract** In the United States, Hispanic male adults suffer from the highest prevalence of overweight and obesity, yet there is a paucity of representation in lifestyle interventions. The purpose of this study was to 1) review existing physical activity and diet interventions published between January 2009 and June 2017 that included Hispanic adult males, and 2) identify culturally-sensitive intervention strategies for promoting successful weight loss. Included publications met the following criteria: (i) participant age 18 years and older, (ii) Hispanic population sample >10%, (iii) included any percentage of males, (iv) intervention addressed physical activity and/or diet, (v) published in English, and (vi) randomized control trial (RCT) study design. The primary search yielded 3,620 articles. Following duplicate removal, 2,679 articles underwent abstract and full-text article screenings. A total of 16 articles were considered eligible and subjected to data extraction. A total of 14 interventions were included as 3 papers

pertained to the same program; 7,703 participants were represented across these interventions. Across all programs 37.9% (n=2814) of participants were male and 26.6% (n=2055) were Hispanic. Of the interventions reviewed, 57% (n=8) specifically recruited Hispanics, 21% (n=3) were inclusive of Hispanics by providing culturally and linguistically appropriate materials, and 21% (n=3) did not make explicit efforts to recruit Hispanics. The data found demonstrates Hispanic males are vastly underrepresented in physical activity and diet interventions. Low participation of this population in weight management interventions will likely result in low generalizability of study results, underscoring the need for more culturally- and gender-sensitive weight loss interventions.

GABRIELLA ROMANO

Mechanical Design of a Transvaginal Salpingoscope

The University of Arizona, Biomedical Engineering
Mentor: Jennifer Barton

Abstract

Ovarian Cancer is the fifth deadliest cancer amongst women and is accountable annually for more deaths than any other cancer of the female reproductive system. Due to a lack of specific and noticeable symptoms associated with early stages of this disease, over a third of women affected will not receive a clinical diagnosis prior to metastasis. Currently, there does not exist an efficient and minimally invasive screening technique capable of detecting early stages of ovarian cancers. Visualization of reproductive organs through optical imaging may provide valuable information needed for these early diagnoses. This project focuses on the generation of the mechanical design of a flexible and steerable multimodal transvaginal salpingoscope, capable of utilizing two novel imaging techniques, Optical Coherence Tomography (OCT) and Multiphoton Microscopy (MPM) in order to image both the ovaries and fallopian tubes. Mechanical design of such an endoscope includes multiple constituents, these being a distal ferrule capable of holding in place optical components, a cover plate capable of forming a water tight seal at the distal tip while maintaining optical performance, and a flexible outer sheath capable of articulating 90 degrees in two directions. In addition to the imaging techniques, the endoscope must also contain two working channels for the introduction of saline and biopsy forceps. Moving forward, once the designs are finalized and constructed, the resulting salpingoscope will be tested for functionality in a ewe model. Funding for this project is provided by the National Institutes of Health/National Institute of Biomedical Imaging, grant number: 1R01EB020605.

VICTORIA ROSAS

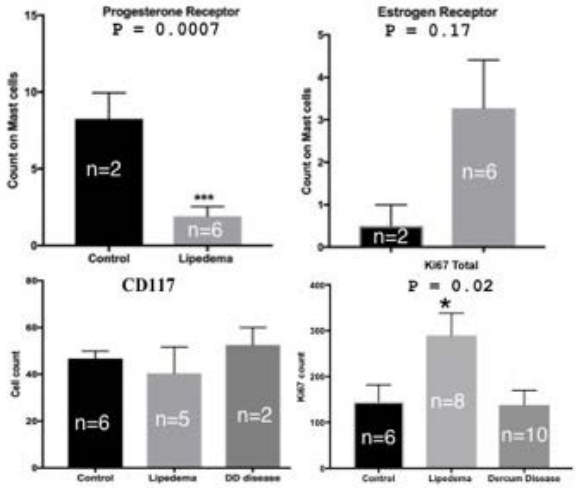
Lower Progesterone Receptors on Mast Cells Do Not Effect Blood Vessel Replication in Lipedema

University of Texas, El Paso, Microbiology
Mentor: Karen Herbst

Abstract

Lipedema (meaning edema in fat) and Dercum Disease (DD) are fat disorders in which accumulation of painful subcutaneous adipose tissue (SAT) affects more females than males, especially at times of female hormone change. Patients with both fat disorders are often misdiagnosed as obese. The purpose of this study was to determine if estrogen (ER) and progesterone receptors (PR) are different in lipedema versus DD in SAT and skin versus controls. We aim to determine levels of ER and PR in SAT and if blood vessels replicate at a higher rate in lipedema and DD versus controls to help further understand these conditions and work towards finding a cure. Immunohistochemistry was used to test for the presence of PR, ER, Ki67 (marker of replicating cells), and CD117 (marker of mast cells).

Lower numbers of PR in our data suggest mast cell secretions (histamine and others) could be higher inducing leakage from vessels and fluid collection in SAT. Fluid in the tissue should induce hypoxia and growth of more blood vessels. Despite higher PR on mast cells, lipedema blood vessels did not appear to be replicating at a higher level. With further research and additional samples, the relevance of elevated PR in lipedema tissue may become apparent.



IBRAHIM SALAMA

Expression Profile of Orai1 and Orai2 and Their Role in Pulmonary Hypertension

University of Texas, El Paso, Biology
Mentor: Jason Yuan

Abstract

Pulmonary arterial hypertension (PAH) is a fatal lung disease that predominantly affects women. It is characterized by increased pulmonary vascular resistance (PVR), which results in an increase in pulmonary arterial pressure (PAP) in patients with PAH. The major causes for increased PVR and PAP are sustained pulmonary vasoconstriction due to pulmonary artery smooth muscle cells (PASMC) contraction and excessive pulmonary vascular remodeling due to PASMC migration and proliferation. Increased Ca^{2+} is a vital stimulus for cell proliferation and contraction in PASMC. The stromal interaction molecule (STIM) and Orai channels are the essential components involved in Ca^{2+} signaling. STIM senses Ca^{2+} in intracellular stores, translocates to the plasma membrane, and recruits Orai to form store-operated channels (SOCs) that pump Ca^{2+} into the cell. Our goal was to do an expression profile of Orai 1 and 2 in different normal rat tissue samples (heart, lung, brain, kidney, liver, tongue, and pulmonary artery). RNA and protein were isolated from these samples and ultimately used in end-point reverse transcription polymerase chain reaction (RT-PCR) and to run Western Blot. The results showed that Orai 1 and 2 were highly expressed in kidney and tongue samples. Interestingly, they were not detected in lung samples but expressed at low level in pulmonary artery tissue. Since lung tissue is mainly composed of pulmonary artery endothelial cells, while the pulmonary artery tissue is primarily from PASMC. We concluded that Orai 1 and 2 are predominantly expressed in pulmonary artery and PASMC but not in PAEC and lung tissue.

ANTHONY SANTIAGO

Microvirus Spike Proteins and their Effects on Host Range and Specificity

The University of Arizona, Microbiology and Molecular & Cellular Biology
Mentor: Bentley Fane

Abstract

Microviruses encapsulate a circular, ssDNA genome within a tail-less, icosahedral capsid. They infect gram-negative bacteria, such as Escherichia coli and Salmonella typhimurium, utilizing lipopolysaccharides (LPS), a complex arrays of sugar molecules present on the surface of gram-negative cells, as a means of host identification. The attachment process begins when bacteriophage spike proteins, found on the exterior vertices of the capsid, and LPS collide. The spike proteins are jettisoned, exposing the coat proteins at the attached vertex to the LPS. Phage are only able to strongly bind to specific hosts possessing the required LPS configuration. Host species that display the required LPS constitute the virus's host range. Previous work has shown that spike, genome piloting, and coat proteins have a role in host attachment and therefore determine host range. However, the principle host determining protein has not been identified. We hypothesize that spike protein G may determine host range. G spikes decorate the external surface of the capsid at each vertex, thus they would likely be

	the first proteins to contact the host cell's surface. Two microvirus species, α3 and St-1, have very similar spike protein sequences but are not identical at the spike tips, the region which would likely be the first to encounter host LPS. Thus, α3 and St-1 have different hosts: E. Coli C and E. Coli K12, respectively. In order to test the proposed hypothesis, the viral G genes will be knocked out via site-directed mutagenesis which introduces premature amber stop codons into the G gene sequence. The mutant viruses will be propagated in amber suppressing cell lines, resulting in a mutant genome that is packaged into a wild-type capsid. These viruses can then be used in complementation tests involving the cloned St-1/α3 G genes. If the foreign G genes can complement the null-G mutants, then the host range of the resulting viral particles can be determined.
	**CHANDLER TURNER** *Program Development for Seed to Read: An Early Literacy and Healthy Eating Program for Low-Income Preschoolers* The University of Arizona, Arts & Letters Pre-Professional, Anthropology Mentor: Kate Speirs **Abstract** Program Development for Seed to Read: An Early Literacy and Healthy Eating Program for Low-Income Preschoolers Recent studies report kindergarten aged children begin school with improper literacy skills. These skills often referred to as emergent literacy skills set the tone for children's academic future. In order to combat this problem, the Seed to Read curriculum was established. Using Interactive Shared reading techniques and activates, the goal of Seed to Read is to improve the literacy skills of children age 3-5 years old. By focusing on children before they begin school the program hopes to enhance preschoolers literacy skill so they will not have to struggle in the future.
	**BREANNA VARELA** *Comparison of Adult and Neonatal Porcine Islet Alpha Cell Fractions* University of California, East Bay, Biological Forensics Mentors: Leah Stehyn and Klearchos Papas **Abstract** There are many new forms of treatment options for Type 1 Diabetes. One of these treatment options is islet transplantation, which is the replacement of the Islets of Langerhans within the pancreas. With islet transplantation as a new beneficial treatment option, there are limits to the access of human islet donors. This limitation makes xenografts or xenotransplantation using porcine islets a possible solution due to their insulin being biocompatible with humans as well as the increased availability of porcine donors. Nonetheless, there are still questions on the variation and function (insulin and glucagon secretion) within the different porcine age groups (adult or neonatal) that will influence the success of the transplantation. The APIs and NPIs were isolated under approved protocols and transported from the University of Minnesota and University of Alberta to the University of Arizona. On arrival the porcine islets are embedded in a mold using O.C.T. The slides with islet sections were

	then stained for DAPI (nuclear marker) and glucagon (alpha cell marker). Each sample had 20 images taken at 20x. Counting glucagon positive cells and dividing by the total cells on each image for each sample revealed the average percentage of glucagon positive cells. APIs (5.417± 0.492) appears to have lower glucagon positive cells as compared to NPIs (7.51± 0.536). This may be due to the neonatal porcine islets immaturity. The data also proposes that APIs have less variability between isolations making them perhaps more appealing to use as the age source for Porcine Islets Transplantation.