THE UNIVERSITY OF NEW MEXICO

U-RISE
Summer Research Symposium

August 25th, 2023
3:00 - 6:30 pm

Schedule

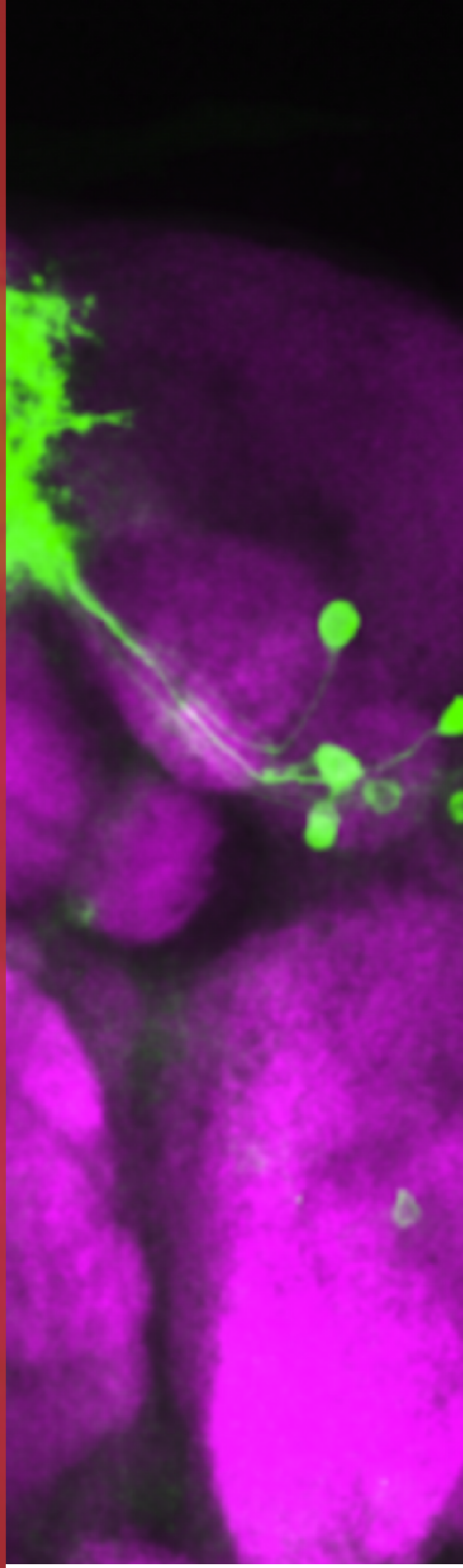
3:00 PM	Opening Remarks
3:15 PM	Alexa Gonzalez
3:30 PM	Brandi Hess
3:45 PM	Brenda Ramos Villanueva
4:00 PM	Alan Ibarra
4:15 PM	Marelessis Palomino
4:30 PM	Courtyard Reception
5:00 PM	Ellie Larence
5:15 PM	Lidia Appell
5:30 PM	Ariana Pritha
5:45 PM	Adina Abudushalamu
6:00 PM	Brendan Sanders

About U-RISE

Our program offers a two-year research experience with the mentorship of a UNM faculty member. With additional support, our scholars develop skills to enter a field in the biomedical sciences.

This experience is essential to preparing a successful application to the top Ph.D. programs in the country.

Program Director -
Dr. Cristina Takacs-Vesbach



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U-RISE
UNIVERSITY OF NEW MEXICO

Our Scholars

Alexa Gonzalez

Hi, my name is Alexa Gonzalez. I am from Arrey, New Mexico and a first-generation college student. I am currently pursuing a major in Spanish and Biology at UNM. In my studies, I have been particularly interested in understanding the molecular mechanisms involved in neural and brain development and function. This summer I was studying as an intern in the Summer Research Opportunities at Harvard Program in a neuroscience lab. Here, I investigate the role of nonclassical MHC genes in determining state-dependent mice behavior. Outside of academics, I like to read, try new dishes, and especially enjoy spending time with loved ones.

Examining State Dependent Changes of the Vomeronasal Organ in Mice

Alexa Gonzalez[1], Mustafa Talay[2] and Catherine Dulac[2]

[1]Biology Department, University of New Mexico, Albuquerque, NM
[2]Department of Molecular and Cellular Biology, Center for Brain Science, Harvard University, Cambridge, MA

Social interactions among animal species play critical roles for survival and overall well-being in response to their environments. Parenting behavior, encompassing nurturing actions like feeding, nest building, grooming, and protection, ensures the survival and development of offspring for eventual reproduction and species success. In mice, *Mus musculus*, parental behavior is observed in sexually experienced and naïve females, as well as father males, whereas sexually naïve males exhibit aggressive and infanticidal behavior towards pups when exposed. The vomeronasal organ (VNO), responsible for detecting pheromones, has been linked to drive infanticide in naïve males, as its ablation induces parenting behavior in sexually inexperienced males. Interestingly, sexually experienced (mated) males show reduced VNO activity when exposed to pups. This study aims to understand the molecular distinctions in the VNO between sexually naïve and mated males. Employing single-cell sequencing, we identify candidate genes with differential expression in VNOs obtained from aggressive virgin males and nonaggressive fathers. To validate these candidates, we utilize sequence-specific probes and employ *in situ* hybridization to quantify the expression levels of the identified genes.

Brandi Hess

My name is Brandi Hess, and I am a senior undergraduate studying biochemistry at the University of New Mexico. On top of being a full-time student and researcher, I am also the mother of a two-year-old boy, and when I am not working on class work or research, my time is spent exploring the world with him. In the spring of 2024, after graduating with my B.S., I will be starting graduate school while expanding on what I have learned throughout my bachelor's. Currently, I am using electrophysiology to look at spreading depolarizations (SD) in mild traumatic injuries (mTBI) and how SD's could explain the behavioral deficits seen in mTBI. I have also have experience using two photon microscopy to look at *in vivo* cell signaling before, during, and after mTBI.

Cortical Network Dysfunction Following Spreading Depolarizations

Brandi R. Hess[1], Natalie J Pinkowski[1], Carissa J. Mehos PhD[2], Russell A. Morton PhD[1]

[1]Department of Neurosciences, University of New Mexico Health Sciences Center, Albuquerque, NM

[2]Center for Brain Recovery and Repair, University of New Mexico Health Sciences Center, Albuquerque, NM

Concussions are considered a mild traumatic brain injury (mTBIs) and are diagnosed by the signs and symptoms of neurological impairment such as loss of consciousness, dizziness, nausea, headaches, and inability to concentrate. However, the underlying mechanisms of the neurological impairment remain unclear. Our previous work in mice has demonstrated that mTBIs can initiate a Spreading Depolarizations (SD). SDs are slowly propagating waves of tissue depolarization that result in the suppression of neuronal firing for multiple minutes. The presence of bilateral SDs in our mTBI model is tightly associated with acute behavioral deficits that last hours. We hypothesize that SD's induce cortical dysfunction and disrupts coherence between the hemispheres of the brain. Using in-vivo electrophysiology, we will investigate cortical network function while inducing SDs via optogenetics. The purpose of these studies is to measure cortical network coherence across hemispheres prior to, during, and after sham or optogenetic induced SDs. Our data suggests that unilateral SDs show a rapid recovery of coherence where bilateral SDs are associated with prolonged disruptions in coherence. These data provide a mechanistic insight into cortical function following mTBI induced SDs.

Brenda Ramos Villanueva

My name is Brenda, and I am from Oaxaca, Mexico. I am currently studying Biology at UNM while conducting avian malaria research in the Witt Lab. During the summer, I participated in the Microbial Friends and Foes program at Cornell University and did cancer immunotherapy research in the Dongre Lab. My main project focused on finding out if cross protection in heterogeneous breast cancer happened as a systemic or local mechanism. Upon completion of my bachelor's degree, I am hoping to participate in a PREP program and eventually apply to a PhD program. During my free time, I enjoy reading, birding and sewing.

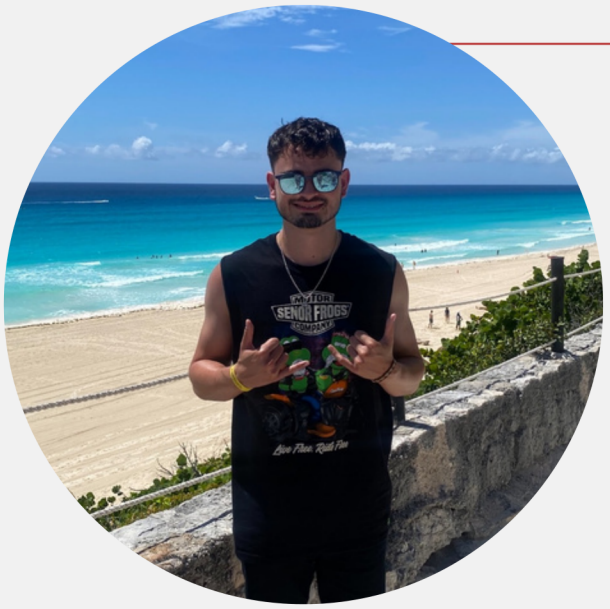
CD73-driven cross protection in heterogeneous breast tumors

Brenda L Ramos Villanueva[1] and Anushka Dongre[2]

[1]Department of Biology, University of New Mexico, Albuquerque, NM
[2]Department of Biomedical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY

The epithelial to mesenchymal transition (EMT) is a cellular process in which epithelial cells acquire quasi-mesenchymal characteristics, gain metastatic potential and drive resistance to anti-CTLA4 immune-checkpoint blockade therapy (ICB). While epithelial carcinomas are infiltrated by CD8+ T-cells and respond well to anti-CTLA4 ICB, quasi-mesenchymal tumors recruit immunosuppressive cells to their tumor microenvironment and are resistant to anti-CTLA4 ICB. Most importantly, in mixed tumors comprised of both cell types, a minority fraction of quasi-mesenchymal cancer cells can cross-protect their epithelial neighbors and drive resistance to anti-CTLA4 ICB in a CD73-dependent manner. CD73 is an adenosine-generating ectoenzyme. However, the underlying mechanism by which it drives cross protection remains unclear. Given that epithelial cancer cells express adenosine receptors, whether they themselves express CD73 to drive such cross-protection is elusive. To address this, we asked whether paracrine factors secreted by quasi-mesenchymal cancer cells could alter the immune-modulatory properties of nearby epithelial cancer cells. Epithelial cells were incubated in control media or conditioned media (CM) derived from quasi-mesenchymal cells. Strikingly, epithelial cells showed an increase in the expression of immune-suppressive CD73 only when cultured in conditioned media from quasi-mesenchymal cancer cells while also remaining epithelial. These results suggest that cross-protection observed in heterogeneous breast tumors may likely be due to the ability of epithelial cancer cells to gain CD73 expression in response to signals received from mesenchymal cancer cells.

Alan Ibarra



My name is Alan Ibarra, and I am a junior majoring in Pharmaceutical Sciences. I am originally from Chihuahua, Mexico, and I am the first member of my family to enroll at an American university, with aspirations to pursue a doctoral degree at prestigious institutions. During my free time, I enjoy exercising, reading, spending time with friends, and cooking. I recently joined Dr. Todd Thompson's laboratory within the Department of Pharmaceutical Sciences. During my time there, I undertaken research in areas of toxicology, cancer biology, and pharmacogenomics. My research primarily centers on the exploration of Astemizole, an antihistamine medication, and its underlying mechanisms that trigger necrotic cell death in cancerous cells through lysosomal dysfunction. I also spent some time working with a Pharmacy student on his project, where we studied Dronabinol as a therapeutic agent to control drug induced neuropathy caused by Paclitaxel treatments by activation of CB1 receptors and inhibition of CYP2J2.

Alan Ibarra, Todd Thompson, *et al.*

[1]Department of Pharmaceutical Sciences, School of Pharmacy, University of New Mexico, Albuquerque, NM

Drug-induced toxicity refers to the harmful effects and adverse reactions that occur as a result of the administration of certain medications. Understanding the mechanism of adverse drug responses is critical in drug analysis and remains a challenge in the development of useful pharmaceuticals. Astemizole, marketed as a second-generation antihistamine, was withdrawn from the market in 1999 due to QTc interval prolongation. It is incompletely understood how drugs such as astemizole are cytotoxic. We have found that astemizole induces a necrotic cell death in several different type of cancer cells. Our goal was to understand the underlying mechanism involved in necrotic cell death induced by astemizole. These studies were performed using A549, PC3, and THP-1 cell lines. MTT viability assays were performed to assess the cytotoxicity of astemizole. An abrupt decrease in cell viability from astemizole was observed with a TD50 ranging between 10 to 20 μM. Biomarkers of apoptotic cell death were not observed. However, an increase in cell volume and fragmentation was observed using flow cytometry, consistent with a necrotic cell death mechanism. In addition, increases in LC3-II protein and autophagic vesicle production were found, indicating the induction of autophagy. A critical step of autophagic pathways is the production of autolysosomes. Astemizole was found to disrupt autolysosome production leading to the production of enlarged autophagic vesicles. Astemizole also inhibited acid sphingomyelinase activity, essential for lysosomal activity. These results support that astemizole produces cytotoxicity through lysosomal dysfunction resulting in necrotic cell death.

Marelessis Palomino

Marelessis Palomino is a senior majoring in chemical engineering with a concentration in material processing. At UNM she works in Dr. Diane Lidke's cancer and immune cell research lab where her research focuses on understanding the fceR1 signaling mechanisms that control the allergic response at a molecular level. This summer she participated in the Summer Honors undergraduate Research Program (SHURP) at Harvard Medical School where she worked in Dr. Don Ingber 's Lab. During her time in the Ingber lab she worked on "Shrilk", a bioinspired plastic from crustaceans and insect exoskeleton cuticles. The motivation behind this project was to create a biodegradable alterative to plastic. She hopes to obtain a PhD in biological engineering and work in pediatric cancer medication development. In her free time, she enjoys traveling, watching movies with her fiancé and building Legos.

Biomimetic Plastic: From Bench Top to Large Scale Manufacturing

Marelessis Palomino[1], Emily Stoler PhD[2], Raghavan Sivaraman[2], Adama M Sesay PhD[2], Donald Ingber PhD[2]

[1]The University of New Mexico Department of Chemical and Biological Engineering

[2]Wyss Institute for Biological Inspired Engineering at Harvard University

Petroleum plastic is resistant to biodegradation meaning that once manufactured, the only way to break down plastic is by using harsh chemical treatment or dumping it in landfill. These methods have contributed largely to the global issue of plastic pollution including the floating ocean of plastic. Additionally, microplastics are becoming a concern as many plastics contain harmful chemicals and additives and can be accidentally consumed harming biological life. Due to the urgency of finding a solution to plastic pollution, our lab has been creating a bioplastic Shrilk using two naturally sourced biopolymers. The advantage of bioplastics is that they are biodegradable, cost-effective, and non-toxic making them a great alternative to plastic. Our biopolymer plastic design is inspired by chitin polysaccharides and protein laminates that make up crustacean and insect exoskeleton cuticles. This complex is formed naturally through non-covalent bonds and requires little to no chemical modification. Previous data suggest that building multilayer films of a 1:2 weight-by-weight ratio of chitosan and silk fibroin has unusually increased strength to nearly that of industrial plastics. Due to this observed strength, our lab is replicating these multilayer films but due to the limitations of the benchtop method our lab is experimenting with using alternative naturally sourced protein, green processing methods, and roll-to-roll manufacturing methods. Our hope is to optimize laminate methods to take Shrilk from a benchtop to a manufacturable product prototype.

Ellie Larence

Ellie Larence is a rising senior at the University of New Mexico (UNM). She has a bachelor's degree in biology and worked as a molecular biologist at the New Mexico Department of Health before returning to UNM to study computer science. Previous research projects Ellie has worked on include characterizing morphological variation in American marten with Dr. Joe Cook and evaluating the performance of deep learning models with Dr. Nilah Ioannidis at the University of California, Berkeley. Currently, she is working with Dr. Melanie Moses at UNM to develop an agent-based model of SARS-CoV-2 infection, using supercomputing resources to simulate the effects of the innate immune response on viral infections. She is also an active member of UNM's supercomputing team, participating in national competitions involving building, running, profiling, and optimizing HPC applications on multiple supercomputing systems. Ellie is broadly interested in applying computational techniques to challenging biomedical research questions and is particularly excited by machine learning applications to genomic data. She plans to pursue a Ph.D. focusing on problems in computational genomics.

Investigating the Impact of DNA Methylation on a Neural Network's Prediction of CTCF Binding

Ellie Larence[1], Ayesha Bajwa[2], and Nilah Ioannidis[2,3]

[1]Department of Computer Science, University of New Mexico, Albuquerque, NM
[2]Department of Electrical Engineering and Computer Sciences, University of California Berkeley, Berkeley, CA
[3]Center for Computational Biology, University of California Berkeley, Berkeley, CA

As genomic data have increased in complexity and magnitude, the analytical tools needed to process and derive insights from those data have adapted. Machine learning algorithms, which detect patterns in complex data, are well equipped for this challenge, and recently several deep learning models have been developed to process genomic data with goals ranging from genotype-phenotype associations to charting biochemically active genomic regions. Enformer is one such model which incorporates self-attention and a large receptive field (100 kb) to predict gene expression from an input DNA sequence.

This project evaluates Enformer's predictive performance by first determining regions where Enformer's predictions differ from two laboratory techniques - Directed Methylation with Long-read Sequencing (DiMeLo-Seq) and chromatin immunoprecipitation (ChIP) with sequencing, both of which map protein-DNA interactions along the genome. Once disparate regions are identified, we investigate possible causes of divergence, specifically assessing whether the methylation state of a DNA sequence impacts Enformer's performance by quantifying methylation.

Understanding the effect of methylation on Enformer's performance will inform how best to improve deep learning models. If input DNA's methylation state explains Enformer's predictive mistakes, incorporating methylation data into a model's input data may be a promising avenue to improve Enformer and similar models. By improving our ability to predict expression from sequence, we gain a parsimonious understanding of transcriptional regulation, the key mechanism by which cells regulate the conversion of DNA to RNA and consequently determine gene activity.

Lidia Appell

Hello! My name is Lidia Appell. I'm from Jemez Springs, New Mexico. I'm a junior majoring in psychology at the University of New Mexico. My research at UNM this summer focused on the computational analysis of intestinal organoids exposed to the cytotoxin EspP. I am interested in acute injury in the gut and dysregulation of the cell cycle, specifically with regards to small RNA post transcriptional regulation. In my free time, I enjoy writing short fiction, dancing, and spending time with my family.

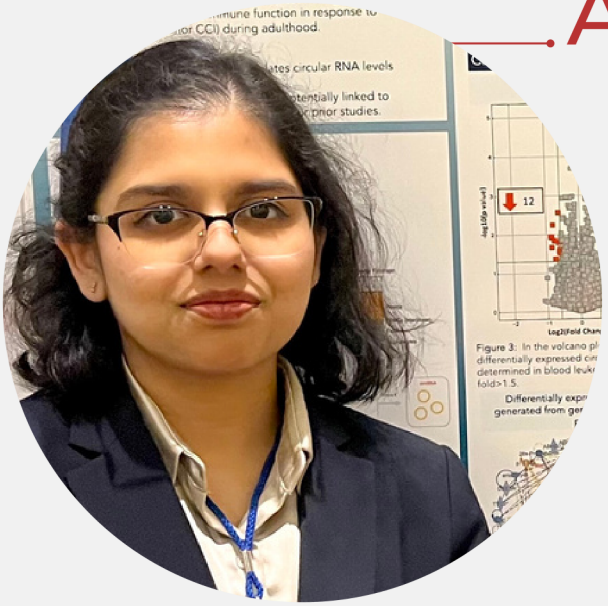
Proteomic and Transcriptomic Analysis of Human Intestinal Organoids Exposed to Bacterial Cytotoxin

Lidia L. Appell[1 2], David P. Scieszka[3], Roger Atanga[2], Julie G. In[2]

[1]Department of Psychology, University of New Mexico, Albuquerque, NM
[2]Division of Gastroenterology and Hepatology, Department of Internal Medicine, University of New Mexico Health Sciences Center, Albuquerque, NM
[3]Department of Pharmaceutical Sciences, College of Pharmacy, University of New Mexico, Albuquerque, NM

EspP is a potent bacterial cytotoxin secreted by enterohemorrhagic *Escherichia coli* (EHEC). EHEC is the most common strain of foodborne *E. coli* found in severe intestinal infection. This toxin was recently shown to cause significant colonic epithelial damage and induce activation of the WNT signaling pathway. WNT signaling is involved in proliferation, differentiation, and regeneration. Disruption of the WNT signaling cascade has been linked to cancer, however the mechanisms between host-pathogen interactions and oncogenesis are not well characterized. Our objective is to elucidate how EspP induced injury can promote colorectal cancer through disruption of the WNT pathway. Human intestinal organoids, derived from colonic biopsies, were dissociated into single cells and processed for droplet-based single cell RNA sequencing (scRNA-seq) or tandem mass spectrometry and iTRAQ. The scRNA-seq data was analyzed using the Seurat pipeline, integrated with the proteomics dataset, then applied to G:Profiler and Cytoscape. The merged datasets show enriched pathways involved in WNT signaling, cyclin degradation, and transcriptional oncogenic regulation are upregulated upon EspP induced injury. Collectively, these pathways are associated with promoting oncogenesis. These computational results suggest that there is a potential link between bacterial cytotoxins and oncogenesis through transcriptional modification. Additionally, several WNT ligands were differentially expressed in the EspP condition, specifically in the transitory progenitor cells. Determining the function of these specific WNT ligands will help elucidate the role that they play in promoting oncogenesis and the link between bacterial cytotoxins and intestinal epithelial dysfunction.

Ariana Pritha



Ariana Pritha is a junior aiming for a dual degree in BS Biochemistry and BS Psychology with a minor in Health, Medicine, and Human Values. Ariana was born in the States but lived in Sweden and Bangladesh (her ethnic origin) prior to immigrating to Canada - and in present-day, has lived in Albuquerque to pursue her undergraduate degree. With hopes to matriculate into a MD/PhD program after graduation, Ariana has been working on her project on identifying and validating circRNAs as potential blood biomarkers for the diagnosis of prenatal alcohol exposure (PAE) in adolescence at the Noor Lab under Dr. Shahani Noor. She is also involved in projects with PhD candidate Andrea Pasmay working with morphine's effects on chronic pain in PAE populations. This past summer, she presented her work at the Research Society on Alcoholism as an Undergraduate Diversity Travel Awardee. Ariana's research interests are in neurodegenerative conditions, biomarkers, diagnostic tools, and patient population trends. In her free time, Ariana enjoys reading classics, listening to music, roller-skating, and acrylic painting.

CIRC-VOPP1 MAY ACT AS NOVEL REGULATOR OF LONG-TERM PERIPHERAL AND CENTRAL NERVOUS SYSTEM IMMUNE DYSFUNCTION DUE TO PRENATAL ALCOHOL EXPOSURE

Ariana Pritha, Andrea Pasmay, Michela Dell'Orco, Joshua J. Sanchez, Jacob E. Sanchez, Suzy Davies, Daniel D. Savage, Nikolaos Mellios, Erin. D. Milligan, Shahani Noor

Department of Neurosciences, School of Medicine, University of New Mexico, Albuquerque, NM, 87131, USA.

Previous studies suggest that prenatal alcohol exposure (PAE) is a risk factor for developing pathological touch sensitivity (e.g., allodynia) following minor peripheral nerve injury that is thought to be due to heightened proinflammatory peripheral and spinal glial immune activation. However, spinal gene expression profiles underlying neuroimmune dysregulation and proinflammatory immune function due to PAE are poorly understood. Non-coding circular RNAs (circRNAs) are novel modulators of mRNA expression. Here, we hypothesized that dysregulation of circRNAs caused by PAE may play regulatory roles in neuroimmune function in response to challenge during adulthood. In rats with unilateral minor sciatic nerve injury that developed a clear unilateral allodynia or sham controls, circulating peripheral immune cells were isolated and analyzed for circRNA expression changes induced by PAE by employing a circRNA microarray platform (Arraystar Inc) with 14,145 probes. Our microarray data identified 18 circRNAs that were significantly differentially expressed in blood leukocytes from adult PAE rats compared to control (Sac) rats ($p < 0.05$) with a >1.5-fold change. Utilizing the Ingenuity Pathway Analysis (IPA), a bioinformatics software program, "top networks" of the genes associated with these differentially expressed circRNAs were analyzed. Distinct canonical pathways including NF-κB pathway, which is activated by innate immune receptor TLR4 signaling, were identified. Following nerve injury, endogenous immune molecules bind to TLR4 receptor inducing NF-κB-mediated transcription of proinflammatory cytokines. Interestingly, our microarray data identified the circRNA, circ-Vopp1, which originates from the *Vopp1* gene and is involved in regulating transcriptional activity of NF-κB. Therefore, using real-time PCR and melting curve analysis, we have designed specific primers for detecting circ-Vopp1 to validate our findings and to identify a specific primer that identifies the circular, and not the linear *Vopp1* mRNA levels. Together, our results suggest that circRNA dysregulation could be a potential underlying mechanism of PAE-induced neuroimmune dysregulation, and that circ-Vopp1 may be a novel circRNA involved in TLR4 pathway sensitization due to PAE. Further research may identify circ-Vopp1 as a clinical biomarker for fetal alcohol spectrum disorder (FASD) and may determine a circ-Vopp1 as a new target to mitigate immune dysfunction consequent of PAE.

Adina Abudushalamu

Hi! I'm Adina Abudushalamu, and I'm currently a senior. I was raised in Las Cruces, New Mexico and moved up to Albuquerque to study Biochemistry here at UNM. This summer, I continued my research project in the Gulisija Lab at UNM on how genetic interactions affect adaptation of organisms in temporally changing environments. In my free time, I like spending time with friends and family as well as listening to music or reading.

Genetic Interactions Modulate Levels of Genetic Diversity in Changing Environments

Adina Abudushalamu[1 2], Eve Rowland[3], and Davorka Gulisija[3]

[1]Department of Biochemistry and Molecular Biology, [2]Department of Computer Science, University of New Mexico, Albuquerque, NM
[3]Department of Biology, University of New Mexico, Albuquerque, NM

Evolution can occur rapidly, resulting in notable genetic changes in populations within generations. In temporally varying environments, rapid evolution occurs by natural selection on existing gene forms (alleles), known as balanced genetic polymorphism. Balanced polymorphism arises when selection maintains multiple alleles in populations. However, only a few mechanisms are known to promote multi-locus (multiple genes) balanced polymorphism in temporally varying environments and typically require gene linkage (genes that are located close together on a chromosome) or sign epistasis. We propose that gene interactions (epistasis) can modulate the levels of balanced polymorphism in the absence of gene linkage. We model a multi-locus population-genetic model of subdivided Wright–Fisher populations and use forward-in-time computer simulations to examine conditions for multi-locus balanced polymorphism under the conditions of the spatial storage effect (a plausible natural mechanism). We model positive, negative, and sign epistasis between two unlinked loci under a broad range of selection coefficients, migration rates, and mutation rates in temporally varying environments. We find that both positive and sign epistasis increase diversity at the two selected loci under the spatial storage effect and population subdivision but negative epistasis decreases diversity, compared to diversity levels in the absence of epistasis. Overall, a novel mechanism for balanced polymorphism arises when genes positively interact under the spatial storage effect, expanding our understanding of multi-locus balanced polymorphism. We are currently examining the maintenance of multi-locus polymorphism under the spatial storage effect and epistasis in the absence of subdivision. This research helps elucidate the basis for rapid adaptation in temporally varying environments.

Brendan Sanders

Hi! I am Brendan Sanders, a rising senior majoring in biochemistry. This summer, I conducted research with Dr. Tamara Terzian of the Department of Dermatology at the University of Colorado Anschutz Medical campus. I worked on a human subject study to determine a quantitative method to replace recall bias of prior sun exposure and damage and subsequent Melanoma risk. Methods of cutaneous melanoma prevention still consist of generic widely used recommendations such as patient reported history of sun exposure, which can be subjective and shaped by recall bias. Here we are trying to find a quantifiable replacement of sun exposure history recall. For this, we tested the integration of 3 innovative ways of measuring sun-induced skin damage and compared this data to sun exposure history of numerous subjects. In my free time, I enjoy mountain and road biking, hiking, and reading.

Integrating Sun Damage Indicators in Melanoma Risk Modeling

Brendan Sanders[1 2], Ramiro Rodriguez[1], Logan Elwood-Digel[1], Andrew Lu[1], Andrew Nicklawsky[1], Robert Dellavalle[1], Lori Crane[1], Neil Box[1], Tamara Terzian[1]

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Melanoma presents a significant health issue in the United States, with a 477% increase over fifty years marking a more rapid incidence than any other cancer over that period. Ultraviolet (UV) radiation in sunlight is the main environmental melanoma risk factor. Our team has demonstrated that burning UV exposures interact with the genetic make-up of light skinned people to increase biomarkers of melanoma risk like moles and freckles. This study utilized subject's reported history of sun exposure, which can be subjective and shaped by recall bias. Here, we are trying to find a quantifiable replacement of sun exposure history recall by creating a composite score that integrates 3 innovative ways of measuring sun-induced skin damage. To assess the extent of Ultraviolet radiation (UVR)-induced damage, we used a survey of sun safe habits, and 3 non-invasive sun damage assessment tools on 102 human subjects born in 1998 who have been followed in a longitudinal study: The first sun damage assessment tool was Skin Microtopography, where a silicone cast takes an impression of the skin, removed, and analyzed by three researchers to quantify the lines and pores for sun damage in accordance to Beagley-Gibson skin grading scale. The second tool was SCINEXA, a scoring system that assesses and differentiates between intrinsic and extrinsic skin aging. The subject's face, neck and arms are examined by 3-4 researchers for components of intrinsic and extrinsic aging. Such factors included presence and size of nevi, sunburn freckling, and wrinkling of the face, shoulders, and hands. The third tool was the Canfield Visia camera, which analyses facial phenotypic factors associated with UV damage and melanoma risk. Frontal UV and cross-polarized photographs, wrinkling, nevus counts, spots (UV red, and brown) were analyzed. Preliminary data showed null or poor agreement between raters for the SCINEXA scoring. While preliminary results were inconclusive, we will continue working on understanding how the different measures of UV damage relate to each other and making a composite score that can replace recall of sun exposure history.

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Undergraduate Research Training Initiative for Student Enhancement

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