Isabela Alesna
Independent Research-AHSSRP

Isabela Alesna is a sophomore at Macalester College majoring in biology, a minor in anthropology and a concentration in community and global health. The firstborn of two Filipino migrants, her future goals are to obtain a career in the biological sciences or in medicine. Isabela is currently a volunteer EMT at Macalester's Emergency Medical Services, and will be volunteering at Fairview Hospice. She enjoys drawing, painting and playing ultimate frisbee.

Institution: Macalester College
Faculty Mentor/Department: Dr. Mark Schleiss/Pediatrics
Poster Title: **Characterizing gp138.1 and 138.2 in Guinea Pig Cytomegalovirus Infection**

Abstract: Human cytomegalovirus (HCMV) infects 0.5-2% of neonates worldwide and has been declared a public health priority because of its severe long-term sequelae, including sensorineural hearing loss and mental retardation. Due to the species specificity of betaherpesviruses, HCMV cannot be studied in animal models; however, guinea pig cytomegalovirus (GPCMV) crosses the placental barrier and infects the fetus, providing a superior rodent model for studying congenital CMV. Not all gene products of the >200 kbp GPCMV genome are known, and recently a 1.6 kb locus encoding GP129, GP131 and GP133 was discovered to form part of a pentameric complex essential for infection *in vivo*. Our purpose was to characterize genes gp138.1 and gp138.2 in ATCC GPCMV strain 22122 near this 1.6 kb locus. Since gp138.1 and gp138.2 have no homologs in HCMV, we investigated if gp138.1 and gp138.2 encode gene products that are expressed in natural GPCMV infection. Genes were successfully cloned to construct recombinant plasmid vectors, which were then used to transfect guinea pig lung fibroblast cell lines. RNA blots indicated the transcription of the genes 72 hours post-infection. Lack of successful antibody binding using immune sera from infected animals during immunoblotting in addition to predicted protein structures and functions suggested that these gene products are not membrane proteins or glycoproteins, and are likely not targets of immune response during natural infection. Further study is needed to discern the roles of these genes during infection, but results lend to a better understanding of the GPCMV genome.

Shaughn Anderson
Heart, Lung, & Blood

Shaughn Anderson is a junior at Whitman College, with a major in Biochemistry, Biophysics and Molecular Biology (BBMB). He is interested in doing molecular and biochemical research

with medical applications. He is a dual citizen of the US and Italy. He enjoys running, reading, sports and film.

Institution: Whitman College
Faculty Mentor/Department: Dr. Alexandra Sobeck/Biochemistry
Poster Title: **Mapping of the CtIP Interaction Domain on the *Fanconi anemia* Protein FANCD2**

Abstract: *Fanconi anemia* (FA) is an inherited genetic disorder characterized by bone marrow failure and increased cancer susceptibility. FA is caused by mutations in any one of 18 functionally interconnected FA genes. Classically, the FA pathway is known to promote the repair of DNA interstrand crosslinks (ICLs)[1]. In addition, we recently found that the central FA pathway member, FANCD2, functions during the DNA replication stress response. FANCD2 cooperates with the key DNA repair protein, CtIP to mediate the restart of stalled replication forks[2]. Although FANCD2 and CtIP were previously shown to directly interact *in vitro*[3], the interaction domain on FANCD2 remains unknown. In this study, we are seeking to identify this domain. Due to the instability of recombinant human FANCD2 protein, we are taking advantages of the *Xenopus laevis* system to generate recombinant *Xenopus* FANCD2 (xFANCD2) and CtIP (xCtIP) proteins[4]. We are using full-length xFANCD2 as well as seven (T1-7) truncated xFANCD2 protein fragments for interactions with xCtIP *in vitro*. Our goal is to identify the minimal CtIP-interaction site on FANCD2, to then ask whether the FANCD2-CtIP interaction is crucial for replication stress recovery and ultimately, for the maintenance of genome stability and anti-cancer protection.

Ted Bebi
Independent Research-AHSSRP

Ted Bebi is a rising junior at Macalester College majoring in biology and special interest in biomedical research. His future plans include a combination of medicine and public health. He grew up in Albania where he spent the first 18 years of his life and recently moved to the US to pursue higher education. Ted enjoys writing, reading, music, traveling and foreign languages. As of now, he does not drink coffee.

Institution: Macalester College
Faculty Mentor/Department: Dr. Cindy Martin/ Medicine, Cardiology
Poster Title: **Sox7 Regulates Vascular Development by Modulating the Notch Signaling Pathway**

Abstract: Cardiovascular development is crucial to the survival of vertebrate animals. Understanding the intricate molecular pathways involved in normal development could facilitate future therapies for cardiovascular

disease. Sox7 is a transcription factor that has a known role in endothelial cell development. Sox7 null mice die between e10.5 and e12.5 of cardiovascular defects. *In vitro* overexpression of Sox7 leads to increased levels of endothelial progenitor cells. An RNA-Seq experiment to identify Sox7 target genes indicated that overexpression of Sox7 modulated several signaling pathways, including the Notch signaling pathway. The Notch signaling pathway is known to play an important role in vascular development. Among the identified genes, Hey2 and Dll4 were shown to be upregulated by Sox7 overexpression. Dll4 is a known target gene of Sox7 in arterial-venous specification. Hey2 is a downstream target gene of the Notch signaling pathway contributing to arterial fate determination of endothelial cells as well as ventricular septation in the heart. We hypothesize that a mechanism by which Sox7 controls vascular cell specification is through transcriptional activation of Hey2. Gene expression analysis by RT-qPCR demonstrated that Hey2 and Sox7 have a similar expression pattern in wild type mouse embryonic bodies (EBs). Doxycycline-induced overexpression of Sox7 showed temporal upregulation of Hey2 in mouse EBs. We identified a Sox7 binding sequence in the proximal promoter of the Hey2 gene and experiments are underway to determine whether Sox7 can activate the Hey2 promoter. Our preliminary data indicate that Sox7 regulates endothelial development by interacting with the Notch signaling pathway.

Brionna Bennett
Heart, Lung, & Blood

Brionna Bennett is sophomore at Winthrop University majoring in biology and minoring in chemistry. After completing her undergraduate studies she plans to attend graduate school. Her career goals are to obtain a PhD in microbiology with a concentration in immunology and infectious diseases. She dreams of working in a lab as a microbiology researcher. Brionna enjoys reading, gymnastics, and playing the violin. She is currently an intern at a local pregnancy clinic.

Institution: Winthrop University
Faculty Mentor/Department: Dr. Sabita Roy/Department of Surgery
Poster Title: **Methamphetamine and Its Effects on the Intestinal Epithelial Barrier**
Abstract: Methamphetamine(METH) is a highly abused and addictive stimulant, with an estimated 35 million users worldwide. METH usage elicits a plethora of consequences in the body including the brain, lung, and liver. Recent studies by our lab suggest that METH increases bacterial translocation within the liver, lung, and mesenteric lymph node. Bacteria can be translocated by two mechanisms—an imbalance within the microbiome or an impaired epithelial barrier. Therefore, in the present study, we analyzed the effect of METH on epithelial barrier function using

two cell lines—IEC-6 and CaCo-2. We hypothesize that METH will modulate the barrier function by inducing cell death, and disrupting the epithelial tight junctions. We found that high dosage METH at 100 μM causes a significant reduction in cell viability at 24h and 48h in IEC-6 and CaCo-2 cells using CCK8 assay, however, low dosage of METH at $10\mu M$doesn't have the same effect. We also found that $100\mu M$ METH caused a significant increase in cell apoptosis in IEC-6 cells. Furthermore, using Electric Cell Impedance Sensing(ECIS) we show a disruption in barrier integrity. In summary, this study show that METH treatment in high doses disrupts epithelial barrier function by modulating tight junction integrity and epithelial cell viability.

Mariah Berner
Heart, Lung, & Blood

Mariah Berner is a freshman double majoring in Biology, Society, and Environment and Psychology at the University of Minnesota. Her plans are to go into the field of medicine and research, with a focus in hematology/oncology. Mariah is part of variety of groups on campus including Pre-meds in Action, Students Today Leaders Forever, Human Rights Advisory Board, and Colleges Against Cancer. Apart from time spent in school and with clubs, she enjoys outdoor activities and playing the viola.

Institution: University of Minnesota - Twin Cities
Faculty Mentor/Department: Dr. Colin Campbell/Pharmacology
Poster Title: **Creating a Plasmid Vector to Investigate the Cellular Response to DNA-Protein Crosslinks**

Abstract: DNA-protein crosslinks (DPCs) form when protein becomes covalently attached to DNA. DPCs interfere with DNA replication, and can cause replication fork collapse. Because replication fork collapse is associated with DNA double-strand breaks, we seek to test the hypothesis that DNA double-strand break repair contributes to DPC repair. To test this hypothesis we constructed a substrate in which a site-specific DPC can be repaired *via* either non-homologous DNA end-joining (NHEJ) or homologous recombination (HR). Error-prone NHEJ-mediated repair substrate can activate a dysfunctional mCherry gene leading to red fluorescent protein production. In contrast, HR-mediated repair activates a defective green fluorescent protein gene. This substrate will be transfected into wild type and repair-deficient Chinese hamster cell lines and the respective levels of red and green fluorescent proteins will be monitored. We anticipate that both the NHEJ and HR pathways contribute to DPC repair. However, because HR is largely error-free, whereas NHEJ typically results in insertions or deletions at the site of repair, we predict that clones deficient in HR will display an enhanced reliance on error-

prone NHEJ to repair DPCs. In contrast, we propose that inactivation of the NHEJ repair pathway will have a minimal effect on the frequency of HR-mediated DPC repair.

Maren Bettermann
Heart, Lung, & Blood

Maren Bettermann is currently a junior at Macalester College double majoring in Biology and Religious Studies. Her future career plans include a career that combines medicine and biomedical research. She has a special interest in public health, and is a certified HIV educator through the Minnesota AIDS Project. She enjoys being a member of both the cross country and track and field teams at Macalester College.

Institution: Macalester College
Faculty Mentor/Department: Dr. Jill Siegfried/Pharmacology
Poster Title: **Activation of the Fibroblast Growth Factor Receptor through the Estrogen Receptor Pathway in Non-Small Cell Lung Cancer**

Abstract: It has become clearer that estrogen signaling pathway is involved in the development and progression of non-small cell lung cancer (NSCLC). For example cytoplasmic detection of the Estrogen Receptor-1 (ERβ-1) has been identified as a negative prognostic factor for lung cancer supporting epidemiological evidence about role of estrogen in lung cancer. Our lab has provided evidence for the interaction between ERβ and the Epidermal Growth Factor Receptor (EGFR/HER-1), which contribute to NSCLC growth, protection from apoptosis and angiogenesis. It is thought that other receptor tyrosine kinases such as the fibroblast growth factor receptor (FGFR) might be interacting as well with ERβ. Here we show immunohistochemical and immunoblotting evidence in regards the interaction between these pathways. We observe an early activation of the FGFR-1 pathway in several NSCLC cell lines with the addition of estrogen ligand. This demonstrates that there is an interaction between the ERβ and the FGFR-1 pathway—contributing to NSCLC proliferation, growth and development.

HannahSofia Brown
Neuroscience

HannahSofia Brown is a freshman at Vanderbilt University, majoring in Medicine, Health, and Society with a focus on health disparities in America. HannahSofia plans to attend medical school, and is interested in pursuing a career in rural family medicine. She is also interested in health care policy and reform. HannahSofia hopes to speak Spanish fluently by the time she graduates from Vanderbilt, and intends to study abroad in Latin America sometime in the next

three years. She currently tutors children in transition housing. HannahSofia enjoys swimming, running, and traveling.

Institution: Vanderbilt University
Faculty Mentor/Department: Dr. Wensheng Lin/Neuroscience
Poster Title: **The Role of the ATF6 Pathway in Oligodendrocytes**
Abstract: Oligodendrocytes are myelin producing cells in the central nervous system (CNS). The myelin sheath is an enormous plasma membrane structure that wraps around axons to insulate them. ATF6 activation in response to disruption of ER homeostasis stimulates the expression of ER chaperones, restoring the function of the ER. Evidence suggests that ATF6 is activated in oligodendrocytes under normal and disease conditions. Out study investigates if the ATF6 pathway is involved in the myelinating function of oligodendrocytes, using a genetic approach. CC1 staining was used to compare the number of oligodendrocytes in wild type versus ATF6 knockout mice, and MBP staining was used to compare myelination in wild type versus ATF6 knockout mice. We found that knocking out the ATF6 pathway did not change the number of oligodendrocytes or the degree of myelination in the CNS of young, developing mice and adult mice. This finding implies the minimal role of ATF6 in the myelinating function of oligodendrocytes.

Paola Caballero-Leon
Heart, Lung, & Blood-MSTP

Paola Caballero-Leon is a sophomore at the University of Puerto Rico - Cayey majoring in chemistry with a special interest in biochemistry. Her future plans include a career that combines medicine and biomedical research. Paola's passion for chemistry has encouraged her to become the treasurer of the ACS Student Chapter at the University, where she is able to provide tutoring to other students. Spanish is her native language, though she manages to speak English with high proficiency and is currently learning a third language: French. She is an avid reader and dancing is her main hobby.

Institution: University of Puerto Rico - Cayey
Faculty Mentor/Department: Dr. Jakub Tolar/Pediatrics
Poster Title: **The Role of Matrix Metalloproteinases in the Outcome of Bone Marrow Transplant for Junctional Epidermolysis Bullosa**
Abstract: Severe generalized junctional epidermolysis bullosa (JEB) is a lethal skin disease that is caused by loss-of-function mutations in genes that encode for the protein laminin-332 (*LAMA3, LAMB3, and LAMC2*). Retroviral gene therapy and induced pluripotent stem cell therapy have been proposed to treat lethal diseases such as JEB. Nonetheless, these therapies have not been widely accepted due to the fact they are both viral-based

and have potential toxicities. Bone marrow transplantation (BMT) is an alternative therapy that has proved effective in another variant of EB called recessive dystrophic epidermolysis bullosa (RDEB). Clinical trials have started with JEB patients, but the results are not as promising as for RDEB patients. BMT cross-corrected the genetic deficiency in JEB patients with the *LAMA3* mutation but not in patients with the *LAMB3* mutation. We hypothesized that this is due to unwarranted cleavage of the β3 chain by upregulated pro-migratory enzymes such as matrix metalloproteinases (MMPs). To study this, we used fibroblasts from healthy (control), *LAMA3* deficient (JEB 1), and *LAMB3* deficient (JEB 2) patients and extracted RNA to determine changes in gene expression of candidate MMPs via real-time qPCR. Our data shows a tendency in downregulation of our candidate MMPs in the LAMB3 deficient JEB 2 patient and upregulation in the *LAMA3* deficient JEB1, except for MT5-MMP. BMT outcomes could be potentially subject to this difference in gene expression between the two variants of JEB. Our study clearly shows that the basement membrane zone of the JEB patients is altered and unusual quantities of its components might be interfering with the process of laminin-332 production and retention.

HeeJin Cheon
Heart, Lung, & Blood-MSTP

HeeJin Cheon is currently a junior at Cornell University, majoring in Biological Sciences. She is interested in immunology and its applications in medicine. Though she is a citizen of the United States, she was born and raised in South Korea until she was 13. She enjoys public speaking and learning about diverse traditions and cultures. She also has a desire to travel around the world one day. Her hobbies include swing dancing and watching documentaries. She is planning on pursuing a dual MD/PhD degree.

Institution: Cornell University
Faculty Mentor/Department: Dr. Bryce Binstadt/Pediatrics
Poster Title: **Role of IL-17A and IL-17F on B Cell Isotype Switching and Activation in the Pathogenesis of Rheumatoid Arthritis**

Abstract: Rheumatoid arthritis, inflammation of the joints due to autoimmune causes, affects about one in 150 Americans. Studies using arthritic mouse models have suggested that the interleukin (IL)-17 family of cytokines is heavily involved in the pathogenesis. Although targeting the IL-17 pathway for treating other autoimmune diseases such as psoriasis and psoriatic arthritis has been successful, efforts to target the IL-17 pathway for treating rheumatoid arthritis have shown little to modest benefit in clinical studies. Autoantibodies produced by B cells are critical in driving rheumatoid arthritis, but how the IL-17 family of cytokines affects B cells has not been elucidated. Therefore, we stimulated wild type murine B cells

with varying concentrations of IL-17A, IL-17F, and combinations of IL-17A and IL-17F to observe their effects on B cell isotype switching and activation. We observed increased IgG1 production with IL-17A, but not with IL-17F. IgG2b, IgG2c, and IgG3 antibodies were not detectable. We observed upregulation of MHCII in all our samples treated with anti-CD40 and anti-IgM antibody, and this effect was independent of type of cytokine added. We did not observe any significant effects of IL-17A and IL-17F treatment on CD80 expression. Additional insight on the IL-17 system will help find suitable treatment for rheumatoid arthritis and clarify the discrepancies found between animal models of arthritis and clinical studies.

Austin Cole
Independent Research-BTI

Austin Cole is currently a senior at the University of Minnesota enrolled in two degree programs; Biology and Statistics. His research focuses on evolutionary prediction, specifically using *Escherichia coli* chemotaxis. He is also involved in Student Government, Jazz performance, and campus politics. After completion of the LSSURP, Austin will attend graduate school at UT Austin's PhD program in Cell and Molecular Biology.

Institution: University of Minnesota - Twin Cities
Faculty Mentor/Department: Dr. Michael Travisano/Ecology, Evolution and Behavior
Poster Title: **Molecular Convergence in Experimentally Evolved Populations of *Escherichia coli* Under Chemotaxis Selection**

Abstract: Evolutionary convergence is a well-documented phenomenon, but the combination of selection and genotype-phenotype relationship facilitating it is poorly understood. Under 1500 generations of experimental evolution with *Escherichia coli* (*E. coli*) selected for both chemotaxis efficacy and specificity in glucose-rich minimal media soft agar, we document remarkable molecular convergence across ten populations. Physiological tuning of all ten evolved populations was consistent with expectations derived from analytical and computational models. Adaptive mutations of separately evolved lineages frequently occurred within the known network governing chemotaxis, but notably, all populations qualitatively exhibited at least one of three types of gene duplication. Despite the consistency of duplication, the amplified regions don't contain genes anticipated to be adaptive in the experimental environment. The repeatability of duplication and location of adaptive mutation suggests evolution is somewhat predictable under selection on a well documented genotype- phenotype map, though caution should be taken to restrict predictions appropriately.

Alexander Dash

Independent Research-AHSSRP

Alexander Dash is a sophomore at Macalester College majoring in Biology, with a minor in Chemistry, a concentration in Global Health. He is interested in pursuing an MD/PhD. Alexander wants his future career to be a Physician Scientist or in Pharmacology where he explores some sort of chronic disease--whether that be cancer, heart disease, or neurodegenerative disorders. He is an only child from New York City and his hobbies include playing baseball for Macalester, giving tours, and occasionally working for the local chapter of Habitat for Humanity.

Institution: Macalester College
Faculty Mentor/Department: Dr. Robert Kratzke/Medicine, Hematology, Oncology, Transplantation Office

Poster Title: **Role of JAK/STAT Pathway in Mediating Sensitivity of Non-Small Cell Lung Cancer Cells to Vesicular Stomatitis Virus Expressing Human Interferon-β (VSV-hIFNβ)**

Abstract: Vesicular Stomatitis Virus (VSV) producing human interferon-β (hIFNβ) is a potent oncolytic virus with limited toxicity to healthy human cells. IFNβ is key to sensing viral infection in human cells and results in activation of the JAK/STAT pathway, among many other genes. In combination with the JAK/STAT inhibitor, Ruxolitinib, it is hypothesized that VSV-hIFNβ replication will be stimulated in cancer cells with an intact interferon pathway. Five different non-small cell lung cancer cell lines were treated with escalating concentrations of Ruxolitinib and multiplicities of infection (MOIs) of virus. Western blots of JAK/STAT proteins were preformed to determine the effects of VSV-hIFNβ in combination with Ruxolitinib on the signaling pathway. Cells were seeded and treated in ninety-six well plate to measure cell viability (CCK-8 assay). Viral titers were measured by using the limiting dilution assay to determine the tissue culture infective dose 50% (TCID50). In the cell lines with a partially functional interferon pathway (H838, A549, H2009, H2030), VSV-hIFNβ amplified production of both total and phosphorylated STAT1. This effect was abrogated when combined with Ruxolitinib, which is corroborated by the increase in viral mediated killing and viral titer in all of the cell lines at lower MOIs. Therefore, our results indicate that Ruxolitinib can sensitize cancer types with an intact interferon pathway to VSV-hIFNβ.

Ernie Diaz-Rivera
Heart, Lung, & Blood

Ernie Diaz-Rivera is currently a sophomore at the University of Puerto Rico - Ponce. He is part of the Student Honor Program and an Adjunct member of Ponce Research Initiative for Scientific Enhancement Program. His current goals are to finish his biomedical major and continue postgraduate studies in graduate or professional school. Ernie is exploring oncology

and/or cancer research as possible career options. He likes to participate in volunteer activities like giving food and clothes to the homeless, visiting children and the elderly, and providing workshops. Some of his pass time activities include lifting weights, staying up to date with the world of technology, going to the movies and spending time with friends and family.

Institution: University of Puerto Rico - Ponce
Faculty Mentor/Department: Dr. Ameeta Kelekar/Lab Medicine and Pathology
Poster Title: **Investigating a Putative Glycolysis Regulating Interaction between GAPDH and Mcl-1**

Abstract: Cancer metabolism has been an increasingly researched topic in recent years. The Kelekar Laboratory has focused on human Noxa's role in altered cancer cells metabolism. Noxa, an apoptosis regulator, is recruited into glucose sensitive, multiprotein complexes (A and B) in leukemia cells, and the lab has demonstrated that Noxa overexpressing cells exhibit higher metabolic rates until, but not past, the Glyceraldehyde-3-Phosphate Dehydrogenase (GAPDH) catalyzed step of glycolysis. They have also determined that GAPDH and Myeloid cell leukemia 1 protein (Mcl-1) are components of both complexes, and are able to interact with each other. They hypothesize that this interaction regulates glycolysis by disrupting GAPDH's catalytic function. Although we have evidence of the proteins' interaction, the binding site is unknown. The region of interaction was narrowed to a hydrophobic finger at the carboxy (C)-terminus of GAPDH. Deletion of this sequence abrogated binding, but the loss of binding could be due to misfolding of the protein rather than loss of the interaction site. To resolve this issue, GAPDH's size and integrity was preserved creating single point mutants within the hydrophobic finger at the C-terminus. These mutants were translated, incubated with purified recombinant His-Mcl-1 and subjected to a His protein pull-down assay. Testing the binding ability of GAPDH deletion and point mutants to Mcl-1 will help identify the specific residues that may be required for the interactions. Future studies will focus on the disruption effect caused on GAPDH's catalytic function and later determine whether this interaction regulates metabolic rates, particularly glycolysis, in leukemia.

Sylvia Edoigiawerie
Neuroscience

Sylvia Edoigiawerie is currently a freshman at the University of Maryland - Baltimore County. Due to her interest in the science behind learning and memory and her appreciation for combining knowledge from seemingly divergent fields of study like biomedicine and art, she is majoring in biology and Interdisciplinary Studies with a neuroscience focus. After her undergraduate studies, she aspires to pursue a career as a physician-scientist. In addition, Sylvia enjoys writing and classical oil painting and values both hobbies because of the creative perspective they give her in regards to science.

Institution: University of Maryland - Baltimore
Faculty Mentor/Department: Dr. Marija Cvetanovic/Neuroscience
Poster Title: **The Role of NF-kB in SCA1**
Abstract: Spinocerebellar Ataxia type 1 (SCA1) is an autosomal dominant poly-glutamine disease that is caused by an expanded CAG tri-nucleotide repeat in the ATAXIN1 (ATXN1) gene. Most studies on SCA1 have focused on degeneration of Purkinje neurons in the cerebellum that is observed in this disease. Our focus is on glial cells, and we have recently shown that they are activated early in SCA1. This study aims to examine the effect of glial cell activation on SCA1 and establish their role in the inflammatory process that may contribute to neuron degeneration. We focus on Bergmann Glia, a specific subset of cerebellar astrocytes that are closely linked to the Purkinje neurons in the cerebellar cortex and on NF-kB, a key transcriptional regulator of inflammation. We have examined NF-kB activation in ATXN1[82Q] mice, an animal model of SCA1 that expresses mutant ATXN1, a gene with eighty-two repeats designed to replicate the cerebellar atrophy and motor incoordination seen in humans. We have found increased NF-kB activation by immunohistochemistry and western blotting in some cerebellar samples from ATXN1 mice compared to wild type littermate controls between the ages of one and six months. We hope to use this knowledge to test the potential of modulation of NF-kB pathway as a possible form of therapy for SCA1.

Tia Eskridge
Heart, Lung, & Blood

Tia Eskridge is a sophomore in Macalester College majoring in chemistry with a potential minor in physics and mathematics. She plans to pursue a career in chemistry after attaining her PhD. Tia wants to find a career that involves both chemistry and physics such as the one that researches alternative sources of energy through chemical means. She is interested in the energy interaction within molecules. Tia enjoys reading, anime, and singing during her free time.

Institution: Macalester College
Faculty Mentor/Department: Dr. Lisa Peterson
Poster Title: **The Interaction of Two Compounds Found in Tobacco**
Abstract: The aim is to examine if there is an interaction between two chemicals found in tobacco products: acetaldehyde and N-nitrosonornicotine (NNN). NNN is a known human carcinogen that causes cancerous tumors in the esophagus; while acetaldehyde is a possible human carcinogen. Our hypothesis is that animals receiving both compounds will have an increased rate of tumor formation than animals receiving only one of the compounds based on three potential pathways: 1) acetaldehyde may

inhibit repair of NNN-derived DNA damage; 2) acetaldehyde may act as a co-carcinogen by increasing cell proliferation in the esophagus; 3) acetaldehyde may form DNA adducts that are mutagenic. We test the interaction of NNN and acetaldehyde by using F344 male rats that consume water containing these chemicals. There are six treatment groups in total: control (tap water), 4 ppm NNN, 8 ppm NNN, 3000 ppm acetaldehyde, 3000 ppm acetaldehyde plus 4 ppm NNN, and 3000 ppm acetaldehyde plus 8 ppm NNN. These dosages are comparable to the relative amounts of these chemicals found in tobacco products. The focus of my research was to analyze the NNN and acetaldehyde concentrations in the water in order to ensure the rats are receiving the intended dosages. A high performance liquid chromatography instrument (HPLC) with UV detection was used to quantify the concentrations by an external standard curve. NNN has a UV absorbance so it is injected directly onto the instrument. However, acetaldehyde does not, so it is reacted with excess 2,4-dinitrophenylhydrazine (DNPH) to form the corresponding hydrazone. The reaction attaches DNPH where the aromatic moiety in DNPH can be read by the UV detector and give the concentration of acetaldehyde. The concentrations of NNN and acetaldehyde throughout my research have been within ten percent of the expected amount for each treatment group. This shows that the dosages of NNN and acetaldehyde are what we expect for each treatment group.

Madison Glass
Neuroscience

Madison Rose Glass was born in Montana, but raised in Ohio, the south eastern part that looks very much like the landscapes in Calvin and Hobbes. She is currently a junior at Haverford College and hopes to contribute to neuroscience of development, learning and memory that will one day help educators accommodate diverse learning needs. Along with pursuing her career goals, she enjoys event planning, martial arts, teaching, and talking to other people about books. This past year she lived in a community house called a Nerd House.

Institution: Haverford College
Faculty Mentor/Department: Dr. Robert Meisel/Neuroscience
Poster Title: **Visualizing Nucleus Accumbens Afferents Active during Female Syrian Hamster Sex Experience**

Abstract: All addictions share the mesolimbic reward pathway with motivated behaviors such as feeding and sexual behavior. Studying naturally motivated behaviors to understand the components of this reward pathway could lead to better treatments for addictions. The nucleus accumbens is part of this pathway, but its afferents that are active during female Syrian hamster sex experience, a model motivated behavior, are unknown. To

visualize these neurons, immunohistochemistry was used on brain tissue harvested from two groups of female Syrian hamsters, a group that had sex experience and a control group that did not. Previously, both groups were ovariectomized and given stereotaxic injections of the retrograde tracer cholera toxin B in the nucleus accumbens of the right hemisphere. Cholera toxin B was stained to determine nucleus accumbens afferents, and cFos was stained to determine which cells were recently activated. The cholera toxin B injection included the nucleus accumbens, and cFos staining in the accumbens was responsive to sex experience. The medial prefrontal cortex was trending significance for active afferents, and may help drive nucleus accumbens activity during sex. No significantly active afferents were in the basal medial amygdala. This study describes an approach to examining the neural circuitry that mediates motivated behaviors.

Elizabeth Gordon
Heart, Lung, & Blood

Elizabeth Gordon is currently a freshman at the University of Florida majoring in biotechnology and a minor in anthropology. Her future goals are to graduate with a doctorate degree and participate in medical research that incorporates both biology and medical anthropology. Elizabeth has a twin sister, but she claims they look nothing alike. Elizabeth enjoys playing percussion for her university, reading, being outside, and watching The Office. She currently volunteers in a hematology/oncology lab through a local hospital.

Institution: University of Florida
Faculty Mentor/Department: Dr. Li-Na Wei/Pharmacology

Poster Title: **Synergistic Effect of IL-4 and Retinoic Acid on Arginase-1 Production in the M2 Macrophage**

Abstract: Retinoic acid (RA), the active metabolite of vitamin A, has important roles in innate and adaptive immune cells as vitamin A deficiency increases susceptibility to various types of infection. Macrophages are main players in innate immunity, and they can be activated, mainly, as pro-inflammatory (M1) or anti-inflammatory (M2) macrophages in response to different stimuli. Arginase-1 is a marker of the M2 macrophage and is an enzyme that is essential for collagen synthesis and tissue repair. The functional role of RA in macrophages is not well established. In this study, we examined the role of RA in M2 activation. We found that the addition of Interliuken-4 (IL-4) and RA create a strong synergistic effect that results in dramatic increase in arginase-1 production and activity. We also investigated the molecular mechanism that results in this synergistic effect using mouse macrophages cell line.

Ayantu Hamid
Independent Research-BTI

Ayantu Hamid is a senior at the University of Minnesota majoring in biochemistry. She is interested in helping underserved communities with a career in medicine and also wants to participate in scientific discovery of cell structures and mechanisms through biomedical research. Ayantu plans to go back to her country to contribute to improve the health care system in Ethiopia. She has five siblings and four of them are also in the biomedical field profession. Ayantu's hobbies are reading, swimming, and learning the ukulele.

Institution: University of Minnesota Twin Cities
Faculty Mentor/Department: Dr. Burckhard Seelig/Biochemistry

Poster Title: **Using *Escherichia coli* to Identify and Modify Novel Enzymes from Synthetic Peptide Libraries**

Abstract: Protein catalysts found in nature are subjected to many evolutionary selections to give rise to enzymes used in cells today. However, there are many other possible amino acid sequences that are not utilized in nature. In our study, we examined potential de novo enzymes derived from two protein libraries. The first protein library is based on the thermostable GDPD enzyme of T. maritime with seven of the eight loops on the catalytic face randomized. The other protein library contains unstructured, randomized variants that are 80 amino acids long. Potential de novo enzymes are able to rescue auxotrophic *E. coli* strains that are missing an essential gene and do not grow on minimal media. These protein variants are examined for catalytic function, its mechanism, and whether supplementing the missing enzyme. *In vivo* selection has identified five protein variants from the two libraries capable of rescuing auxotrophs in minimal media. Variants that rescue the auxotrophic strains are further analyzed for catalytic activity or upregulation of multicopy supressor genes to rescue the strain. Three multicopy supressor genes are removed form the auxotrophic strains and will aid to show that the rescue is not due to upregulation. Following this we use directed evolution to increase the activity of variants to decrease amount of time needed to rescue. Directed evolution will also be useful to detect catalytic activity *in vitro*. We expect these variant to function as the enzyme coded by the genes serB and metC of the auxotrophic strains but at a slowed rate.

Nicole Haywood
Neuroscience

Nicole Haywood is a sophomore at Oakwood University majoring in Biology with a plan of pursing a MD/PhD program after graduation. Her goal is to become a neurosurgeon or anesthesiologist. Nicole is one of seven children raised in the rough neighborhoods of south central Los Angeles, CA. In her spare time Nicole enjoys volunteering for low income student schools, symphonies, military air shows, and visiting museums. She hopes that through her career she can help bring science programs to children of a lower socioeconomic status.

Institution: Oakwood College
Faculty Mentor/Department: Dr. Lucy Vulchanova-Hart/Neuroscience

Poster Title: **Conditional Deletion of VGF in Sensory Neurons to Study the Function of VGF Nerve Injury-Induced Hypersensitivity**

Abstract: In the pain pathway, nociceptor neurons -whose cell bodies reside within the dorsal root ganglia (DRG)- detect noxious stimuli via their peripheral processes and transmit these signals via their central processes into the superficial spinal dorsal horn where they synapse with second-order neurons. Subsequently, these neurons transmit the signal to the thalamus in the brain. Damage to peripheral nerves causes changes to neurons of the pain pathway, leading to chronic neuropathic pain. Hypersensitivity to mechanical stimuli is one of the distinguishing features of neuropathic pain. At the onset of peripheral nerve damage it has been observed that there is an upregulation of the neuropeptide precursor protein VGF (non-acronymic). VGF is proteolytically cleaved into bioactive peptides that are thought to play a role in mechanisms of chronic pain. Therefore, we sought to determine if VGF participated in maintaining hypersensitivity following peripheral nerve injury using AAV-mediated conditional deletion of VGF. The objective of this project was to determine Cre-recombinase (Cre) and VGF expression in spinal cord and DRG tissue through anatomical analysis after treatment with AAV9 vectors carrying the gene for Cre under the control of the cytomegalovirus (CMV) or synapsin (Syn) promoters. Mice with a floxed VGF gene were subjected to spared nerve injury (SNI), a model of peripheral nerve damage. Seven weeks after injury the mice were treated with AAV9.Syn.Cre-GFP and AAV9.CMV.Cre-GFP to conditionally knockout VGF. DRG and spinal cord tissue were harvested 8 weeks after vector treatment. A double-labeling method of immunofluorescent staining was used on spinal tissue to identify location, number, and types of cells transduced with AAV9.Syn.Cre-GFP and AAV9.CMV.Cre-GFP. Glial markers were also used to determine whether the virus caused neuroinflammation. DRG were also analyzed for number of transduced neurons and number of VGF-positive neurons. In the spinal cord, more cells were transduced in the AAV9.Syn1.Cre treatment group compared to AAV9.CMV.Cre. GFAP labeling suggested the presence of gliosis in vector-treated mice, consistent with potential neuroinflammation. In DRG, the transduction rate was similar between the two vector treatments. This was unexpected in comparison to previous work, which resulted in greater transduction rates using AAV9.CMV.GFP. The number of VGF-positive DRG neurons

was decreased in vector-treated mice, indicating successful Cre-dependent deletion. This observation was in agreement with transient decrease in hypersensitivity in vector-treated mice.

Jason Kelly
Heart, Lung, & Blood

Jason Kelly is a junior at Austin Peay State University majoring in physics with a math minor. He has been highly involved in music ever since the sixth grade. Jason plays the saxophone and is currently learning how to play the piano. At Austin Peay he is involved in the marching band, pep band, jazz band, and symphonic band. Jason is also a member of Phi Mu Alpha, the national men's music fraternity. In the future he wants to attend graduate school for biomedical engineering.

Institution: Austin Peay State University
Faculty Mentor/Department: Dr. Paul Iaizzo/Surgery

Poster Title: **Highlighting Coronary Vasculature of Perfusion-Fixed Hearts using Magnetic Resonance Imaging**

Abstract: Magnetic resonance imaging, or MRI, of perfusion-fixed hearts is highly effective at obtaining high quality images of soft tissue and fat. Computed tomography, or CT, scans are preferred for imaging vasculature of the perfusion-fixed hearts. CT scans, however, have a much lower resolution than MRI, which can show details as small as 0.1mm. Gadolinium-based contrast agents can be used in MRI to brighten images at the location of delivery. A method for obtaining a model of both the tissue and vasculature of ex-situ perfusion-fixed human hearts using a single imaging method, MRI, is needed.

Balloon catheters were inserted into the coronary sinus and coronary arteries to fill the vasculature with various solutions.

The gadolinium-based contrast resulted in an image that fluoresced well, but the contrast leaked from the vasculature into the tissue, thus the delivery method needed to be improved. After the vasculature was properly occluded, solutions including fish oil, graphite microspheres, and mineral oil were explored with varying success. More agents are being explored in an attempt to optimize the imaging of coronary vasculature, myocardium, and adipose tissue using a single imaging modality.

Anna Kim
Heart, Lung, & Blood-MSTP

Anna Kim is currently a junior at Boston College majoring in biology with an interest in computer science. Her future goals include the pursuit of an advanced degree with a focus on research in biomedicine or biotechnology. In her spare time, Anna enjoys listening to music, drawing, and playing video games. Other hobbies include playing viola with the Boston College Symphony Orchestra, friendly discussions on life, ethics and philosophy, and volunteering when able to particularly in the arts.

Institution: Boston College
Faculty Mentor/Department: Dr. Dan Kaufman/Medicine

Poster Title: **The Role of the Aryl Hydrocarbon Receptor Pathway on Early Human Hematoendothelial Development**

Abstract: Human embryonic stem cells (hESCs) are an ideal platform to understand the basic developmental mechanisms that facilitate the production of early hematopoietic stem and progenitor cells. The aryl hydrocarbon receptor (AHR) pathway has emerged as a key regulator of HSC expansion *ex vivo*. The pathway had previously been studied for its normal functioning in the detection and metabolism of xenobiotic aryl hydrocarbons as a transcription factor for two members of the cytochrome P450 family. However, its antagonist StemRegenin1 (SR1) was found to cause expansion of umbilical cord blood hematopoietic stem cells (HSCs). Here, we evaluated the role of AHR on human hematopoiesis at an even earlier stage of development. We hypothesized that AHR signaling plays a role in hESC differentiation from hematoendothelial cells into early hematopoietic progenitor cells. To better elucidate the role of AHR, we treated hESCs with the *AHR* antagonist SR1 and the agonist 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and quantified the decrease and increase of expression of downstream targets *CYP1A1* (0.32±0.12 and 9.07±1.24; n = 3) and *CYP1B1* (0.19±0.02 and 9.19±2.23; n = 3) using quantitative reverse transcriptase PCR (qRT-PCR). qRT-PCR data has also demonstrated that *AHR* expression increases as hESCs differentiate, indicated by the increased expression of the downstream targets *CYP1A1* (329.32±226.95; n = 4), *CYP1B1* (453.19±339.83; n = 4), and *AHRR* (42.75±23.63; n = 4) by Day 15 of differentiation relative to undifferentiated hESCs. We then generated AHR gene knockouts in hESCs using the CRISPR/Cas9 gene editing system and optimized primers to produce PCR products of the altered region. Successful clones were identified using the optimized PCR products with the Surveyor assay. Future work includes the quantification of altered expression of hematoendothelial differentiation genes in the *AHR* knockout clones. By disrupting this pathway and subsequently increasing hESC differentiation into hematopoietic stem and progenitor cells, we can be better understand factors that play key roles in HSC differentiation to potentially improve the use of hESCs and human induced pluripotent stem cells (iPSCs) for the production of HSCs suitable for use in patients.

Elizabeth Lezama
Heart, Lung, & Blood

Elizabeth Lezama is currently a sophomore at the Milwaukee School of Engineering majoring in Biomedical Engineering with a special interest in Medicinal Cardiology and the testing of medical devices. Her future plans include a career that combines medicine and biomedical research such as an MD/PhD. She will be the first in her family to get a college degree and go on to higher education. Elizabeth's hobbies include Zumba, volunteering, hanging out with friends, traveling, and sleeping.

Institution: Milwaukee School of Engineering
Faculty Mentor/Department: Dr. Colin Campbell/Pharmacology
Poster Title: **Constructing pGL3 Donor Plasmid to Test Homologous Recombination in Response to DNA-Protein Crosslinks**

Abstract: DNA-protein crosslinks (DPCs) form when proteins become covalently attached to chromosomal DNA. Known causative agents include numerous cancer chemotherapeutic drugs, ionizing radiation, and cigarette smoke. DPCs exhibit cytotoxic and mutagenic characteristics. They interrupt chromosomal replication and can cause mutations that can cause cancer. Currently, it is not known how cells recognize and respond to drug-induced DPCs. In order to gain insight into this question, a plasmid containing a 700bp deletion in the luciferase gene was constructed. This 700bp deletion causes the luciferase gene to be inactivated. A site specific DPC, already constructed, will be co-transfected with the plasmid containing the inactivated luciferase gene into Chinese hamster cells and a host cell reactivation assay performed to quantify DNA repair. We hypothesize that cells utilize homologous recombination to repair DPCs. To test this hypothesis we will co-transfect an undamaged homologous recombination donor plasmid along with the DPC-containing plasmid. It is believed that homologous recombination with the donor plasmid enhances DPC repair and will yield more plasmids with an active luciferase gene. We anticipate that insight gained into the pathways that contribute to DPC repair will enhance efforts to understand how tumor cells develop resistance to certain anti-cancer therapeutics, and may aid in the development of novel anti-cancer drugs.

Sue Heidi Loperena-Medina
Heart, Lung, & Blood

Sue Heidi Loperena-Medina is a senior at the University of Puerto Rico - Aguadilla majoring in Biomedical Sciences. From a very young age, she enjoyed reading fiction novels, drawing, painting, and admiring nature. Sue Heidi likes going to the beach, riding her beautiful green bike,

and including her little sister in her academic activities to ignite her scientific interest. With her free time she gets involved in volunteer and extracurricular work in her community and university. Sue Heidi's dreams and plans are to contribute to the scientific world as a researcher, explore different countries, ride in a hot air balloon and watch the Aurora Borealis.

Institution: University of Puerto Rico - Aguadilla
Faculty Mentor/Department: Dr. Stephen Jameson/Lab Medicine and Pathology
Poster Title: **ARTC2.2 Expression Determines Phenotypic Change of T Cell Subsets in Lymphoid and Non-Lymphoid Tissues**

Abstract: Preservation of full functionality and phenotype of T cells during cell preparation is critical in various experimental settings and for their potential use in therapies. The ADP-ribosylation pathway is one way that phenotype and functionality of T cells may be affected. Through this pathway, the ARTC2.2 enzyme utilizes excess NAD^+ released by tissue injury to ADP-ribosylate the ionotropic receptor P2X7. This permits downstream effects that eventually change membrane integrity, which could lead to cell death. Previous publications have shown that inhibition of ARTC2.2 enzyme with a camelid nanobody improves recovery of Tregs and NKT cells in spleen and liver, respectively. However, the expression of ARTC2.2 and P2X7 on different T cell subsets in various tissues is elusive. In the present study, we investigated the expression of ARTC2.2 and P2X7 on conventional CD4 and CD8 T cells as well as iNKT and Tregs in thymus, spleen, liver and lymph nodes. We confirmed that ARTC2.2 is highly expressed in all T cell subsets in peripheral organs while its expression is almost absent in thymus. Furthermore, the expression of P2X7 is detected with higher frequency on CD4 T cells in all tissues, but its expression appears to be negligible on CD8 T cells. Future experiments are aimed at determining the physiological significance of the expression of ARTC2.2 and P2X7, and at studying other non-lymphoid tissues such as the gut. Additionally, we want to examine what regulates the expression of this enzyme and further translate this research to ARTC2.2$^{-/-}$ and P2X7$^{-/-}$ mice.

Michael Montgomery
Heart, Lung, & Blood

Michael Montgomery is a junior microbiology student at California State University - Long Beach. He conducts undergraduate directed research on how defects in intracellular nucleocapsid movement affect the viral infection's ability to spread from cell-to-cell. In addition to his academic endeavors, Michael participates in Division 1 athletics as a pole-vaulter on the university Track & Field team. After graduation, he plans on pursing a Ph.D. to assist him in his

quest to discover novel ways to combat multi drug-resistant strains of bacterial pathogens as well as advancing technology in the realm of viral infectious disease treatment.

Institution: California State University
Faculty Mentor/Department: Dr. Deepali Sachdev/Medicine
Poster Title: **IGF-I Regulates Metastasis through HIF-1a and XBP1 Pathway in Triple Negative Breast Cancer**

Abstract: The insulin-like growth factor (IGF)/insulin system has been proven to play a role in metastasis and survival of circulating cancer cells in triple negative breast cancer (TNBC) through activation of downstream pathways such as PI3K. The Sachdev lab has previously demonstrated that angiogenesis is inhibited in xenograft tumors expressing functional type I insulin-like growth factor receptor 1 (IGF1R) by blocking the receptor with an antibody. The objective of this study was to examine the relationship between IGF-I signaling via IGF1R, and various transcription factors in the progression, as well as metastasis, of triple negative breast cancer in a model of high risk metastatic breast cancer using the triple negative MDA435/LCC6 metastatic cancer cells. Herein we hypothesize that activation of IGF1R regulates metastasis by affecting levels of molecules within the HIF-1 pathway and the unfolded protein response (UPR) pathway, which have been implicated in the progression of TNBC. Activation of downstream signaling pathways was verified through recruitment of molecular methods including BCA assay, SDS-PAGE, and western blot. LCC6 cells with functional IGF1R (LCC6-WT) and LCC6 cells with a truncated, dominant negative IGF1R (LCC6-DN) were treated with IGF-I and transcript levels of HIF-1/VEGF and XBP1 pathway were quantified by RNA isolation, cDNA synthesis, and qRT-PCR. Our very preliminary data suggests that IGF1R activation in LCC6-WT cells may upregulate XBP1 spliced variant while LCC6-DN cells show a trend towards decreased XBP1 levels. It was also observed that IGF1R activation did not affect levels of HIF-1a transcript under the normoxic conditions used in the experiments. This data suggests that IGF-I signaling via IGF1R may regulate metastasis through interaction with the UPR and HIF-1a pathways.

Carlos Nowotny
Heart, Lung, & Blood-MSTP

Carlos Nowotny is currently a junior at San Diego State University majoring in Chemistry with an emphasis in Biochemistry and a minor in Neuroscience. He plans to attain an MD/PhD degree in Neurological sciences, with the goal of treating and researching neurodegenerative diseases.

Carlos was born in La Paz, Bolivia and is a first generation college student. When not in lab he enjoys going to concerts, spending time with family, and exercising.

Institution: San Diego State University
Faculty Mentor/Department: Dr. Gregory Vercellotti/Department of Medicine
Poster Title: **Heme Binding in Recombinant Murine Hemopexin**
Abstract: Sickle-cell Disease (SCD) is characterized by hemolysis, which releases hemoglobin and heme into the blood stream. Heme binds to TLR4 promoting oxidative damage, inflammation and vaso-occlusion. In SCD, heme detoxification and clearance becomes paramount. Heme can be cleared by haptoglobin (Hp) and hemopexin (HPX). The clearance of heme-HPX complexes occurs via hepatic CD91 receptors. In SCD patients Hp and HPX levels are low due to chronic hemolysis. In mouse models of SCD, supplementation of Hp and HPX prevents Hb or heme mediated vaso-occlusion. We hypothesize that gene therapy with HPX would protect murine SCD models from oxidative stress, inflammation and vaso-occlusion. In preliminary studies gene therapy with WT HPX inhibited inflammation and vaso-occlusion. In these studies we will examine functional assays detailing the specific residues responsible for this heme binding utilizing spectroscopy. We designed, and expressed rat FLAG-tagged hemopexin in Chinese Hamster Ovary (CHO) cell lines. Using site-directed mutagenesis we also expressed two heme-binding site mutants, and a CD 91 receptor mutant. A double His to Ala mutant at positions 235 and 291 had no functional loss of heme binding. Hemopexin overexpression inhibits heme-induced vaso-occlusion in murine models of SCD. A triple His to Ala mutant at positions 149, 235, and 291 had a significantly diminished heme-binding capacity. An Arg150Ala and Glu152Ala mutation at the putative CD91 receptor-binding site had not functional loss of heme binding. Future studies will look to test the important role of HPX in murine sickle cell disease models; utilizing these proteins to further understand the importance of heme clearance.

Natalia Olmeda-Viera
Heart, Lung, & Blood

Natalia Olmeda-Viera is currently a junior at the University of Puerto Rico - Humacao. Her future plans are to finish her bachelor degree in Industrial Chemistry, obtain a PhD in a pharmaceutical related science and working on behalf of others. She loves to work with the youth of her community by tutoring them and by offering various activities to help them grow as better people. Natalia enjoys reading, music and visiting new places.

Institution: University of Puerto Rico - Humacao
Faculty Mentor/Department: Dr. Timothy Walseth/Pharmacology

Poster Title: **Synthesis and use of Biotinylated NAADP's for the Affinity Purification of the NAADP Receptor**

Abstract: Nicotinic Acid Adenine Dinucleotide Phosphate (NAADP) is a messenger molecule that activates calcium release from intracellular acidic stores, such as lysosomes. Calcium release mediated by NAADP can result in both local and global signaling cascades within a cell, triggering other calcium signaling pathways. Calcium signaling is important in cellular functions such as fertilization, muscle contraction, and gene expression. Two-pore channels are a family of ion channels that have been identified in the NAADP pathway, however, the receptor protein for NAADP has not been identified. The purpose of this study is to synthesize biotinylated NAADP's for purification of the NAADP receptor by affinity chromatography. Four biotinylated NAADP's were successfully synthesized and characterized for their ability to mimic NAADP action in terms of binding and calcium release in the sea urchin system. All four biotinylated NAADP's were able to successfully compete with ^{32}P-NAADP in a competition binding assay with IC_{50} values 10 to 1000 fold higher than NAADP itself. These compounds also were able to release calcium with EC_{50} values 125 to 1200 fold higher than NAADP. These probes desensitized calcium release induced by 1μM NAADP with IC_{50} values similar to those observed in the competition binding assays. Immobilization of the biotinylated NAADPs on streptavidin-agarose for affinity purification of NAADP binding proteins resulted in the retention of several proteins compared to control columns. This approach might prove useful in the identification of the NAADP receptor.

Alejandra Osorio
Heart, Lung, & Blood-MSTP

Alejandra Osorio is currently a junior at the University of Central Florida majoring in biology with a minor in mathematics. Her special interest in biology is evolution, which she hopes to combine with her interest in medicine as a career in evolutionary medicine. Alejandra is Colombian-American and was the first person in her family actually born in the United States. She enjoys reading, watching movies, traveling, playing piano and volunteering in her spare time.

Institution: University of Central Florida
Faculty Mentor/Department: Dr. David Potter/Medicine, Hematology, Oncology, Transplantation

Poster Title: ***Cyp3a11*** **Gene Excision in SMM2 Ovarian Dependent ER+ HER2- Mouse Mammary Carcinoma Cells**

Abstract: The *Cyp3a* gene family in mice is of emerging interest in mammary cancer progression. Functional analysis of the *Cyp3a* gene family has

been difficult because of redundancy and homology between gene family members, but is important for study of Cyp3a function in mouse models. Cyp3a11 is homologous to human CYP3A4, shown in previous studies to promote cell proliferation and tumor growth in breast cancer through synthesis of epoxyeicosatrienoic acids. While studies measuring the effects of *CYP3A4* gene knockdown on human breast cancer growth have been published, the roles of its homolog *cyp3a11* in mouse mammary cancer remain to be determined and its excision could enable animal models. Application of CRISPR-Cas 9 technology was used in this project to excise *cyp3a11*, which encodes a homologous monooxygenase enzyme in mice. Excision of Cyp3a11 was accomplished using the CRISPR/Cas 9 system to target exons 2 and 3 in the mouse SSM2 cell line, which causes mammary carcinoma. Our results suggest CRISPR/Cas9 excised one allele, leading to the creation of a heterozygous cell line. The effect of this heterozygous knockdown can now be studied by measuring proliferation of these modified cells and testing the role of cancer cell intrinsic Cyp3a11 in mammary tumor growth.

Jessica Pantoja-Alvarado
Heart, Lung, & Blood

Jessica Pantoja is a junior transfer student at the College of the Sequoias, majoring in chemistry and biology. Her career goal is to become a clinical infectious disease research specialist who treats and provides therapy, using biological agents and drugs, to patients with rare diseases. She hopes to obtain a PharmD/PhD or an MD/PhD. Jessica's hobbies include volunteering in her community, hiking, and dancing. She has been a presenter and lecturer for the past four years in 4-10 grade young women and men STEM conferences, as well as at high school science clubs.

Institution: College of the Sequoias
Faculty Mentor/Department: Dr. Li-Na Wei/Pharmacology
Poster Title: **ERK Signaling Pathway is Involved in RA Induced –MMP-9 Expression in P19 Cells**

Abstract: Mouse derived P19 embryonic carcinoma cell lines are able to differentiate into any cell lineage. Understanding the mechanism behind this ability could lead to the development of novel stem cell-based therapies in the field of regenerative medicine. Multiple drugs can induce P19 differentiation, for instance, in the presence of retinoic acid (RA); P19 cells can mature into neuron cells. Additionally, previous studies have shown that matrix metalloproteinase-9 (MMP-9) plays a role in neuron differentiation. The goal of our lab is to ascertain if RA can induce P19 neuronal differentiation by up-regulating MMP-9 through the extracellular-signal-regulated kinase (ERK) signaling pathway. P19 cells were grown and then treated with RA. After treatment, a gelatin zymography was run to detect MMP-9 expression. A western blot was

also run to detect the activity of ERK. It was found that at 30 minutes, RA treated P19 cells can induce ERK phosphorylation. Also, for the gelatin zymography assay, RA was shown to induce MMP-9 expression in P19 cells. Further experiments will be performed to decipher if RA induces MMP-9 via the ERK signal pathway.

Ivory Paulk
Heart, Lung, & Blood

Ivory Paulk is a junior at University of Central Florida. She serves on the Student Government Association Scholarship committee where she is currently helping to create scholarships to support undergraduate researchers. She is passionate about building minority participation in scientific research and higher education. She wishes to get her undergraduate degree in Biotechnology and her PhD in Cancer Biology. She is interested in exploring the neurology of malignant brain tumors. Ultimately she aspires to become a tenured professor, while leading her research into a viable medical product.

Institution: University of Central Florida
Faculty Mentor/Department: Dr. Michael Olin/Department of Pediatrics
Poster Title: **Use of a CD200R Agonist as a Method for Targeting the CD200/CD200R Blockade for Central Nervous System Cancer Immunotherapy**

Abstract: Cancer immunotherapy has demonstrated promising results. However, to date, researchers have failed to overcome the complex interplay between the tumor and its surrounding microenvironment. The progression to a productive immune response involves passing a number of immunological checkpoints. Immunological checkpoints, such as cytotoxic T lymphocyte antigen 4 (CTLA-4) and programed cell death protein 1 (PD-1), which are barriers to the targeting of immunotherapies' due to the use of malignant self-cells, those that express similar surface antigens as the cells in which they have arisen. To overcome this limitation, the FDA approved two checkpoint inhibitors, ipilimumab (anti-CTLA-4) and pembrolizumab (anti-PD-L1)1. In our glioma model, we discovered an excess of soluble CD200 protein, which acts as an immunological checkpoint. CD200 is highly expressed in a variety of human central nervous system tumors2. We reported a significantly increased in concentration of CD200 in the sera of glioma patients as their tumors progressed and went off our clinical trial (Fig 1a)2. Moreover, we reported a direct correlation of serum CD200 concentration and increased lineage negative myeloid derived suppressor cell (MDSC) population in our patients (Fig 1b)1.

Gabriel Peirats
Neuroscience

Gabriel Peirats is currently a sophomore at the University of Notre Dame majoring in Neuroscience and Behavior. He is a 2013 High School Presidential Scholar and honor student. He is particularly interested in Puerto Rico politics, world affairs, human behavior and sports, having played basketball and soccer competitively. He has had extensive extracurricular activities and leadership positions in sports and science fairs. He is passionate about volunteer activities and has widespread experiences in this field in Latin America and in Puerto Rico. His future plans include attending medical school upon graduation and becoming a neurosurgeon.

Institution: University of Notre Dame
Faculty Mentor/Department: Dr. Jonathan Gewirtz
Poster Title: **Effects of Morphine Withdrawal on Glia**
Abstract: Chronic morphine treatment has been shown to increase activation of microglia *in vivo*. We think this activation may contribute to the affective signs of long-term opioid use withdrawal. However, it is currently unclear how morphine activates microglia and whether this activation differs between acute vs. chronic morphine treatment vs. withdrawal. Thus, we sought to determine whether markers of glial activity and cytokines are upregulated by morphine withdrawal using both *in vitro* and *in vivo* models. In addition, we wanted to assess whether toll-like receptor 4 (TLR4) is required for the effects of morphine withdrawal on microglia activation and cytokines expression. To test these hypotheses a cell culture of post-natal glial cells was cultured in glial medium with morphine and then underwent elicit withdrawal. The *in vivo* model consisted of dissected tissue from wild type and TLR4 knockout mice that underwent precipitated morphine withdrawal. The results of the *in vitro* model reveal that the TLRs and markers of glial activity were activated after chronic morphine use, but not withdrawal. However, there was no significant up-regulation of cytokines at any time point. The results for the *in vivo* model reveal that morphine treatment did not alter the expression of TLRs or markers of glial activity regardless of genotype. These findings suggest that unlike chronic morphine exposure, withdrawal does not lead to activation of microglia. Future research is needed to determine the mechanism by which microglia affect the emotional symptoms of opiate withdrawal.

Zackery Perry
Heart, Lung, & Blood-MSTP

Zackery Perry is currently a sophomore at the University of Alabama - Birmingham majoring in chemistry and a focus in biochemistry. He has a budding interest in molecular biology. Zackery is a Sunday school teacher at his local church, and also a volunteer for the Nigerian Student Association on the UAB's campus. He enjoys swimming, strength training, listening to jazz, and singing.

Institution: University of Alabama - Birmingham
Faculty Mentor/Department: Dr. Sundaram Ramakrishnan/Pharmacology
Poster Title: **The Relationship Between miR-210 and IGF2 Under Hypoxia in Ovarian Cancer**

Abstract: Hypoxia is known to drive tumor progression in ovarian cancer. Increased demand and inadequate supply of oxygen leads to hypoxia conditions in tumor microenvironment. Cellular response to hypoxia is regulated through Hypoxia Inducible Factor 1 (HIF1). HIF1 allows cells to adapt to the hypoxic environment and regulate the expression of multiple genes, which are essential for metabolic regulation and angiogenesis. Recent studies have shown that miR-210, a small non-coding RNA, is up-regulated by HIF1 and involved in "fine-tuning" cells to the hypoxic response. Bioinformatics analyses predict Insulin like growth factor II (IGF II) as a potential target for miR-210. IGF II is a maternally imprinted gene and plays an important role in fetal development. Its expression is turned off after birth. In many cancer types, including ovarian cancer, IGFII expression is re-activated. IGF II has been shown to be a potential biomarker in ovarian cancer. The focus of present studies is to understand the relationship between IGF II and miR-210 in ovarian cancer cells under hypoxia.

Blake Petersen
Independent Research-BTI

Blake Petersen is currently a junior majoring in microbiology at the University of Minnesota. His plans include a career focusing on research studying microbes and possibly pathogens. He is currently a volunteer at Dr. Bond's biotechnology lab in St. Paul, and a member of the Microbiology Club and American Society for Microbiology (ASM). In his spare time, Blake enjoys listening to music, reading, movies and TV shows, and video gaming.

Institution: University of Minnesota - Twin Cities
Faculty Mentor/Department: Dr. Daniel Bond/Biotechnology Institute
Poster Title: **Does a Two-Component Regulatory System Control *Geobacter sulfurreducens* Growth Rate?**

Abstract: A recent transposon mutagenesis (Tn-Seq) experiment indicated that mutations in a sensor histidine kinase gene increased growth rate of *Geobacter sulfurreducens*. However, subsequent deletion of this gene did

not alter growth rate, but instead produced mutants that failed to grow in up to 40% of experiments. Closer inspection of Tn-Seq data revealed that only inactivation of an N-terminal domain of unknown function (DUF) in the sensor histidine kinase increased the growth rate of this organism, while mutations to the C-terminal domain were lethal. To study the effect of this N-terminal domain on growth, three variants of the sensor histidine kinase were reintroduced into the full-gene deletion mutant strain of *G. sulfurreducens*. We created a variant of the entire gene, one lacking the membrane-bound receptor domain, and one containing only the ATPase domain of the gene. We predict that cells expressing only the C-terminal ATPase domain will have accelerated growth rates. This shows the value of saturation level mutagenesis data, as it can reveal the effects of specific domains versus an entire protein. The activity of this novel sensor histidine kinase in controlling growth rate of *G. sulfurreducens* can be applied for further engineering of this organism.

Carla Rodriguez-Deliz
Neuroscience

Carla Rodriguez-Deliz is a junior at the University of Puerto Rico - Ponce majoring in biology. She is preparing herself for a career in research and academia. Her specific interests include neuroscience and epigenetics. Occasionally, she tutors peers and assists high school students with science fair projects. Coming from a small island such as Puerto Rico, she enjoys learning about different countries, cultures and languages. Carla spends her free time reading, writing, playing sports and playing guitar.

Institution: University of Puerto Rico - Ponce
Faculty Mentor/Department: Dr. Donald Simone/Diagnostic and Bio Science
Poster Title: **Pain Related Behaviors in Mice Expressing Sickle Hemoglobin: Modulation by Bivalent Ligands**

Abstract: Sickle cell disease (SCD) is a genetic disorder that produces abnormal hemoglobin, leading to vaso-occlusion. The hallmark of this condition is pain. Little is known about the mechanisms of SCD, therefore, treatment remains inefficient. Several types of transgenic mice have been developed in order to study therapeutic options for SCD, including the Townes mice. In this study, we aimed to characterize pain-related behaviors in the Townes sickle mouse, as well as identify the onset of SCD-associated hyperalgesia. In addition, we aimed to study the effect of a new drug, MCC22, on such pain-related behaviors. For this, we measured paw withdrawal frequency (PWF) and mechanical threshold, as well as grip force to compare between control (AA), sickle trait (AS) and sickle cell (SS) mice. MCC22 was administrated through intraperitoneal (IP) injection and the mechanical tests were conducted at 15, 30 and 60 minutes to evaluate its effects. We observed that sickle mice had significantly higher PWF and lower thresholds when compared to AA and AS mice. However, values for grip force were not significantly different

between groups. Administration of MCC22 reduced PWF in SS mice. Decreases in relevant pain behaviors suggest that MCC22 may prove to be a novel effective therapeutic in SCD.

Jonathan Rodriguez-Vega
Heart, Lung, & Blood

Jonathan Rodriguez-Vega is a sophomore at the University of Puerto Rico - Mayaguez, where he is currently pursuing a Bachelors degree in Biology with an interest in Cardiovascular Biology and Microbiology. One of his many hobbies includes being a Tae Kwon Do instructor, playing baseball and reading books. Jonathan is currently an active member of the Biology Honorary Society, Tribeta (BBB), which has helped him develop leadership skills, scientific curiosity, communication and interpersonal skills. After he finishes his Bachelors degree in Biology, Jonathan will pursue a PhD in Cardiovascular Biology.

Institution: University of Puerto Rico - Mayaguez
Faculty Mentor/Department: Dr. Kalpna Gupta/Medicine
Poster Title: **Mechanism of Hemin-Induced Mast Cell Activation**
Abstract: In sickle cell disease, free heme/hemin is released upon hemolysis. Our research has found that hemin induces a new and unique phenomenon in the presence of tumor necrosis factor α (TNFα), causing mast cell activation. We hypothesize hemin induces this activation via the interaction with high-affinity IgE receptor (FcεRI), which stimulates the tyrosine kinase Syk. Syk in return activates the Linker for Activation of T cells (LAT). This signaling pathway activates NOD-like receptors (NLRP3), inducing the activation of the inflammasome in mast cells. We examined the signaling pathways induced by hemin in TNFa-primed mast cells that lead to mast cell degranulation and the formation of the inflammasome. We further analyzed hemin-induced mast cell activation by analyzing cytokines that may be released upon mast cell activation.

Ashley Scott
Heart, Lung, & Blood-MSTP

Ashley Scott is a junior at the University of Maryland Baltimore County majoring in Biological Sciences with a specific interest in cardiology. She plans to pursue a Ph.D. or joint M.D./Ph.D. program in the fall of 2016. Ashley's long term goal is to work as a physician scientist in her own lab at a research university or hospital, developing new ways to answer complex questions related to health and disease. She also has a passion for reading, baking, and playing basketball.

In her spare time Ashley enjoys taking part in her hobbies, but she also believes in giving back to the community.

Institution: University of Maryland - Baltimore County
Faculty Mentor/Department: Dr. Lisa Schimmenti/Pediatrics
Poster Title: **Characterizing the Phenotype of TALEN Engineered slc26a4 Mutant Zebrafish**

Abstract: Two out of every thousand newborns is deaf or hard of hearing. More than half of all hearing loss in newborns has an identifiable genetic cause with Pendred syndrome being one of the most common. Mutations in slc26a4 are associated with Pendred syndrome, a disorder characterized by bilateral sensorineural hearing loss and occasionally accompanied by thyroid goiter. Patients with Pendred syndrome may have an enlarged vestibular aqueduct, a malformation of the temporal bone. It is not known how an abnormal protein product of slc26a4, Pendrin, modulates hearing loss and contributes to temporal bone malformations. We have created mutations in slc26a4 via transcription activator like effector nucleases (TALEN) to mimic mutations found in humans and further characterize phenotypic changes using zebrafish as a model organism. We hypothesize that the homozygous recessive mutant fish will respond less frequently, if at all, to sound stimulus and that the heterozygous mutants will not display an abnormal phenotype. In order to test for deafness, a hearing assay was performed in which fish were exposed to a 400Hz sound. This frequency was chosen as it can specifically stimulate the inner ear and not the lateral line. Bright field imaging was conducted to look at differences in inner ear morphology. In order to quantitatively study the differences between mutant and wild type ears, measurements of the otolith radius and outer ear circumference were taken and standardized to trunk length. Preliminary results show that heterozygous mutants have significantly smaller otoliths and ear areas when compared to wild type fish. Based on this we can conclude that heterozygous mutants display a change in phenotype.

Jamilisse Segarra-Villafane
Heart, Lung, & Blood

Jamilisse Segarra-Villafane is currently a junior at University of Puerto Rico - Rio Piedras majoring in Biology and a special interest in Immunology, Physiology and Endocrinology. Her future plans includes a career in medicine combine with biomedical research. She wants to study more about the implications that any alteration in endocrine system has in other systems in the human body and look for alternatives to control it. Jamilisse's hobbies include playing sports, acting, dancing, singing and community service. She also enjoys watching movies and TV series.

Institution: University of Puerto Rico Rio - Piedres

Faculty Mentor/Department: Dr. Paul Iaizzo/Surgery

Poster Title: **Effects of Laser-Cutting in Muscle Tissue Biomechanics**

Abstract: Laser technologies have advanced surgical procedures in precision and accuracy. However, the effects of laser cutting on tissue biomechanics of surrounding skeletal and smooth muscle are unknown. Our laboratory is interested in understanding how the modality of cutting affects the biomechanical properties of the muscle tissue and using this knowledge to predict tissue behavior and development. We are interested in determining whether the tensile strength of muscle is compromised after laser cutting. Muscle biopsies were taken from a York-X swine. Tissue samples were then cut using a carbon dioxide laser system or scalpel. Equal number of bundles were dissected for both the laser cut and hand dissected groups. After cutting, the tissue was pulled to failure with a tensile test to measure the biomechanical properties of the muscle fibers. The data showed that the rectus had an ultimate tensile strength of 0.0743 ± 0.0542 N/mm^2 and 0.0842 ± 0.0542 N/mm^2 ($p=0.30$) when using the laser and scalpel respectively. For the diaphragm values of 0.0728 ± 0.1028 N/mm^2 with the laser and 0.0625 ± 0.0473 N/mm^2 ($p=0.67$) with the scalpel were obtained. Finally, the esophagus had values of 0.3943 ± 0.2239 N/mm^2 and 0.4308 ± 0.1999 N/mm^2 ($p=0.52$) for the laser and scalpel. These results showed that there is an insignificant difference in the ultimate tensile strength per cross-sectional area in the muscle bundles when we change the modality of cutting for both smooth and skeletal muscle. Thus, laser technology is an effective tool for surgical procedures since they do not alter the tensile strength of the muscle fibers in the tissue.

Jasdip Singh
Heart, Lung, & Blood

Jasdip Singh is a junior at the University at Buffalo majoring in biochemistry. He plans on pursuing a PhD program in biochemistry or physiology and researching causes and treatments of disorders. During his spare time, you can find him tutoring students, or performing heart surgery on mice and running western blots in the laboratory. Jasdip was born in India, and moved to the United States in 1999. He has lived all around the USA, ranging from Portland, Oregon, to Houston Texas, and currently resides in Buffalo, New York. He is the first of his family to attend college.

Institution: SUNY Buffalo

Faculty Mentor/Department: Dr. Mark Schleiss/Pediatrics

Poster Title: **Role of Guinea Pig 116 & GP138.3 Genes in Cytomegalovirus *in vivo* Replication**

Abstract: Cytomegalovirus (CMV) is the most common congenital infection in the United States, infecting nearly 40,000 newborns each year. Approximately 10% of these newborns will have symptoms of the disease at birth, and

another 10-15% will develop hearing loss or mental retardation later in life. CMV is a species specific virus, and guinea pigs have proven to be a useful model for studying the disease because the virus is able to cross the placenta and infect the fetus, analogous to what happens in humans. In 2008, a CMV strain lacking a 1.6kb gene locus was identified. The 1.6kb locus was required for efficient growth *in vivo*, but not *in vitro*. GP 116 and GP 138.3 are two genes that flank the 1.6kb locus, and may play a role in propagation of viruses containing the 1.6kb gene locus. To better understand the role that the 1.6kb gene locus plays in virus propagation, the proteins produced by GP 116 and 138.3 and their functions must be better understood. Using techniques such as northern blotting, western blotting, topo cloning, and ELISAs, the function and structure of the proteins produced by GP 116 and GP138.3 can be elucidated. Proper targeting of the 1.6kb gene locus and its surrounding regions could serve as a method to hinder virus production *in vivo*.

Berliza Soriano
Independent Research

Berliza Soriano is a sophomore at the University of Puerto Rico - Mayaguez majoring in Industrial Biotechnology with a special interest in immunology. Her career goal is to include continuing graduate studies in a career that combines medicine and research in immunology. She is the first in her family to attend college and is a descendant of Dominican parents. Berliza speaks Spanish as her main language and is also proficient in English. She is involved in different types of community work such as reforestation, beach cleaning, tutoring children and much more.

Institution: University of Puerto Rico - Mayaguez
Faculty Mentor/Department: Dr. Michael Sadowsky/Soil, Water and Climate
Poster Title: **Identification of *Medicago truncatula* Genes Involved in Symbiosis with Sinorhizobium**

Abstract: Nitrogen is an essential soil nutrient for plant growth, but biologically useful nitrogen (e.g. nitrate, ammonia) is limited in the field. Some plants, specifically legumes, are able to obtain biologically-fixed nitrogen through symbiosis with rhizobia, where atmospheric nitrogen gas is converted to ammonia. There have been several studies done to understand this symbiosis for the enhancement of biological nitrogen fixation. Approaches have examined increasing the number of nodules formed, enhancing the efficiency of nitrogen fixation, or even modifying non-legumes to allow for symbiotic interaction with rhizobia. The *Medicago truncatula*/Sinorhizobium symbiosis has been used as a model to study legume/Rhizobium symbiosis. In the sequencing of 27 different *Medicago truncatula* genotypes numerous genes were identified that might have a

role in symbiosis formation. These candidate genes have been deleted through targeted genome modification. This study was done to determine the phenotypic changes in these genotypes, and included: chlorophyll content, the number of nodules formed, and nitrogen fixing activities in symbiosis between the modified Medicago and Sinorhizobium. Findings of this study will improve our understanding of *Medicago truncatula*/Sinorhizobium symbiosis and explore possibilities for increasing biologically fixed nitrogen to enhance crop production.

Ari Stoner
Heart, Lung, & Blood-MSTP

Ari Stoner is currently a junior at Indiana University -Bloomington majoring in Biotechnology and minoring in Chemistry and Mathematics. He hopes to pursue a combined career in medicine and biomedical research with a focus on infectious disease and immunology. He is fascinated by microorganisms and their impact on human health. Ari enjoys rock climbing, hiking, reading, and doing community service. He currently volunteers at a local shelter for homeless families.

Institution: Indiana University
Faculty Mentor/Department: Dr. Bryan Williams
Poster Title: **Antibiotic Resistance in *Pseudomonas aeruginosa* Agmatine Hypersecreters**

Abstract: *Pseudomonas aeruginosa* is a ubiquitous pathogen known to infect a broad range of organisms across multiple phyla. It is perhaps best known for its role in chronic infection in the cystic fibrosis (CF) lung, where its vast toolkit with which to develop multidrug resistance has left few effective antibiotics remaining in the arsenal of medical practitioners. A collection of clinical isolates from CF sputum were found to hypersecrete agmatine, a polyamine precursor, via a shared 11 base-pair deletion in *aguA*—a gene involved in polyamine anabolism. Previous work in the Williams lab has indicated that agmatine abundance is correlative with poor lung performance. The link between agmatine secretion in these mutants and their ability to regulate virulence and persistence in the CF lung remains to be elucidated. We hypothesize that agmatine renders the outer membrane of *P. aeruginosa* more positively charged, and thus repellant to cationic antibiotics such as aminoglycosides and colistin—antibiotics regularly used in the treatment of CF infections. To determine whether agmatine secretion induces antibiotic resistance to cationic antibiotics, disk diffusion and broth dilution methods were used. It appears that the *aguA* mutation confers antibiotic resistance in particular conditions for each of the clinical isolates. The mechanism by which agmatine secretion confers antibiotic resistance appears to be complex and warrants further experimentation. Understanding the link between this

mutation in bacterial metabolism and *P. aeruginosa* infection will help to develop better antibiotics and improve disease outcomes.

Ammanuel Taye
Independent Research-BTI

Ammanuel Taye is a junior at the University of Minnesota majoring in Biochemistry and Genetics, Cell Biology and Development. His career goal includes medicine and medical research. He is currently studying Bacterial microcompartment assembly in hopes of utilizing in synthetic biosynthesis. His hobbies include reading, learning about other cultures, listening to music and playing soccer. He is currently working as a research assistant in a biochemistry laboratory and as a part-time pharmacy technician.

Institution: University of Minnesota - Twin Cities
Faculty Mentor/Department: Dr. Claudia Schmidt-Dannert/Biochemistry
Poster Title: **Engineered Protein Nanobioreactors for Selective *in vitro* Biocatalysis**
Abstract: Spatial organization of enzymes is key to reaction efficiency. In nature, bacteria produce organelle-like specialized compartments called bacterial microcompartments (BMCs) to organize metabolic pathways. BMCs are used to encapsulate various pathway enzymes, selectively pass substrates to enhance reaction efficiency, and also prevent detrimental intermediates and byproducts from leaking into the cytosol. Substrate selectivity is provided by the highly-organized porous polyhedral protein arrays that form the outer shell of BMCs. BMCs could be engineered as protein nanobioreactors to selectively catalyze reactions of choice in a highly efficient manner. To achieve this goal, we designed and cloned a BMC shell protein pore variant that could selectively allow passage of polar substrates to the interior of the protein nanobioreactor. We expressed our engineered BMCs in a heterologous host and examined the formation of BMCs by fluorescent microscopy. These data confirmed that engineering the pore of a major shell protein does not disrupt BMC formation. Furthermore, we targeted a fluorescently labelled industrially relevant lipase to our engineered BMCs, and conducted assays to confirm its activity. *In vivo* data reveal that the fluorescently labelled lipase associated with BMCs. In addition, results from *in vitro* enzyme assay show that the lipase enzyme is active with a resorufin-based substrate. These data provide the basis towards designing and engineering protein nanobioreactors, which may be a promising tool to enhance metabolic efficiency in synthetic biochemical pathways.

Keaira Thornton

Heart, Lung, & Blood

Keaira Thornton is a junior at Norfolk State University. Through out her tenure at Norfolk State University, Keaira has achieved numerous awards and honors. Currently majoring in Biology, Pre- Professional with a minor in chemistry, Keaira is a scholar and leader on her campus. She has been initiated into numerous honor societies and holds multiple officer positions in various clubs and organizations. In her future, Keaira plans to conduct research in biomedical science and to become a pediatric doctor because of her interest in children's health. In her spare time, she loves to go paint balling and shopping.

Institution: Norfolk State University
Faculty Mentor/Department: Dr. Michael Georgieff/Pediatrics
Poster Title: **Analysis of Behavior During the Barnes Maze in Adult Mice Previously Subjected to Neonatal Phlebotomy-Induced Anemia**

Abstract: For preterm infants admitted to the neonatal intensive care unit (NICU), anemia is a common problem due to daily blood draws. At the same time, their brains are still developing, in particular the hippocampus, which is vital to proper learning and memory. The loss of iron-rich red blood cells, which carry oxygen to tissues, decreases the amount of oxygen and iron available to the brain. This deficit in early life could negatively affect long-term behaviors even though they recover from the anemia. In this model, mice were phlebotomized from postnatal day (P)3 to P14 to reach a level of anemia similar to NICU preterm infants. These mice were then allowed to recover from anemia until P65, which is neurodevelopmentally equivalent to a human young adult. Adult mice were tested on the Barnes maze. This test uses spatial navigation as a measure of learning and memory, which will help explain the effects of anemia on the hippocampus. Analysis of their behaviors during the tasks revealed differences between phlebotomized mice and non-bled control mice. These differences demonstrated that phlebotomized mice may have more flexibility in learning yet have more anxious phenotypes compared to non-bled control mice. Clarity on the effects of anemia in preterm infants will help neonatologists improve treatment strategies in the NICU. Understanding this impact of early-life anemia on brain development will also demonstrate how early life events can have long-term neurobehavioral consequences.

Rachel Thorson
Heart, Lung, & Blood

Rachel Thorson is a senior at University of Wisconsin - Stevens Point double majoring in biology and biochemistry. Her future plans include biomedical research at the molecular and cellular levels. Rachel is a varsity athlete in swimming and has a passion for being outdoors. Next year she hopes, pending application approval, to join the Peace Corps before heading to

graduate school. Rachel enjoys helping people and is always on an adventure to try and do new things.

Institution: University of Wisconsin - Stevens Point
Faculty Mentor/Department: Dr. Margaret Titus/Genetics, Cell Biol, Dev MEDXX
Poster Title: **Exploring the Biochemistry of Myosin VII Functional Domains and Filopodia Formation In Dicty**

Abstract: The ability for cells to move is critical for our immune response and is necessary for cancer metastasis. Both cancer and immune cells move with amoeboid movement similar to the model organism *Dictyostelium discoideum* (Dicty). Myosin VII is a MyTH4-FERM motor protein necessary for filopodia production. Filopodia are small, hair like extensions of the cytoskeleton and plasma membrane that help the cell to adhere to its substrate and to sense signals during directional migration. We have been focusing on a biochemical and phenotypic analysis of the myosin VII protein. To study the biochemistry we have been developing a protocol for the isolation of the GFP tagged motor domain of myosin VII, called HMM. This will enable us to further understand the kinetics and structure of the myosin VII motor. The phenotype of myosin VII null cells and cells that have the Dicty MyTH4-FERM tail domain replaced with the human myosin X MyTH4-FERM domain were analyzed to determine the cellular role of myosin VII. We found that the Dicty and human MyTH4-FERM domains are interchangeable, suggesting an important conservation in structure that allows for filopodia production. Finally, we observed that loss of filopodia decreases the efficiency of development, in a 3-dimensional environment.

Anibal Tornes-Blanco
Neuroscience

Anibal Tornes-Blanco is a freshman at the University of Puerto Rico - Cayey with a special interest in Neuroscience, specifically on Brain Plasticity behavior. He is an active member at the Chemistry circle and Research Initiative for Scientific Enhancement (RISE). Being the first member of his family to pursue PhD studies is nerve wracking, but he likes pressure. His hobbies include running and reading anything he gets his hands on. In his free time, he volunteers at Centro Medico Hospital, working on various tasks for the oncology department. He aims to obtain a PhD in Rehabilitation Science with a minor in Neuroscience.

Institution: University of Puerto Rico - Cayey
Faculty Mentor/Department: Dr. Anna Lee
Poster Title: **Influence of Estrous Cycle on Alcohol & Nicotine Consumption in C57BL/6 X 129 Female Mice**

Abstract: The prevalence of substance use disorders (SUD) is generally higher in males; however, studies suggest that 18-24 year old females exhibit more

frequent drug consumption. Moreover, preclinical studies have shown higher drug consumption rates in female rodents, although there are conflicting results regarding to the amount female rodents consume. The influence of sex hormones on neurotransmitter modulation and their effects in drug consumption have been understudied. Our objective in this study was to probe for correlations between drug consumption and hormone presence during estrous cycle in female C57BL/6X129 mice. Mice were presented bottles of ethanol (10% v/v) and water during the alcohol consumption test, and bottles of nicotine (20 μg/mL) and water during the nicotine consumption test. All of the bottles were weighed daily and no rodent training was required. Using a visual observation technique of the genitalia we tracked the reproductive versus non-reproductive phases of the cycle. This technique allowed us to audit many mice in a short amount of time, however, it is not as precise as vaginal cytology. We also investigated effects of daily handling on drug consumption. Our results showed that daily handling did not affect alcohol or nicotine consumption and preference. We also found that estrous cycle phases did not significantly affect alcohol or nicotine consumption preference. Future drinking studies in female C57BL/6x129 mice could be performed without tracking the estrous cycle. Differences between male and female drug consumption are not likely due to the effect of the estrous cycle hormones.

Tiauna Travers
Heart, Lung, & Blood

Tiauna Travers is currently a sophomore at Tuskegee University majoring in animal science. She plays volleyball at her university and enjoys swimming, exercising and reading books in her spare time. Tiauna's favorite genre is fantasy because she likes keeping her mind open to all possibilities whether strange or completely outrageous. She plans to become a veterinarian and then go back to school to obtain a PhD in pathology and epidemiology. Tiauna is the last child of her immediate family pursuing a college degree. She is preparing herself for a challenging career in disease control.

Institution: Tuskegee University
Faculty Mentor/Department: Dr. Srinand Sreevatsan/Vet Population Med
Poster Title: **Regions of Difference for *Mycobacterium Tuberculosis* Complex Organisms**
Abstract: *Mycobacterium bovis* is predominantly found in livestock animals but can also infect humans. There is an estimation that from the human tuberculosis cases from 1995 to 2005 only 1.4% were caused by *M. bovis* but it is likely that the number of cases have been underreported. It is known that *M. bovis* is the causative agent of tuberculosis in livestock animals with a few cases in humans but little is known about its

epidemiology. Differentiating *M. bovis* at the subspecies level will provide information on its epidemiology, which will assist in proper treatment for the disease. As an identification tool, a collection of 58 samples of both *M. bovis* and *M. tuberculosis* isolates were ran by polymerase chain reaction (PCR) using the programs IS6110 and RD9 to confirm the samples were part of the MTC complex and what species it belonged to. We discovered that out of the 58 samples 41 samples were confirmed as *M. bovis* isolates. We will perform a PCR based analysis of the two mycobacterium species to determine the presence or absence of specific regions.

Frank Valdes
Neuroscience

Frank Valdes is currently a senior at the University of Arizona double majoring in Neuroscience and Molecular/Cellular Biology. He intends to pursue a PhD in Neuroscience so that he can research the genetic basis of neurodevelopmental and neurodegenerative diseases. When not attending classes, or conducting research, he volunteers as a biology tutor at a community college. Frank was born in Miami to Cuban immigrant parents, and has also lived in Tallahassee, FL, and Tucson, AZ. He is a former musician, and enjoys cooking, reading and traveling.

Institution: University of Arizona
Faculty Mentor/Department: Dr. Esther Krook-Magnuson/Neuroscience
Poster Title: **Pre-Ictal Brain State's Effect on Seizure Length and Optogenetic Intervention**
Abstract: Abstract withheld at request of mentor for intellectual property protection.

Charlene Valdez
Heart, Lung, & Blood

Charlene Valdez is a junior at Trinity Washington University majoring in biological sciences with an emphasis in public health. She is learning more about epidemiology and bioethical issues within modern day society. Her future plans include a career combining global and national public health research. She is looking to ultimately pursue her career goal of becoming a health ambassador for the United States. Her specific interests include type II diabetes mellitus and its prevalence among children and adults in the US or developing countries. Charlene's hobbies include playing guitar, writing in her journal, reading, and exercising.

Institution: Trinity University
Faculty Mentor/Department: Dr. David Bernlohr/Biochemistry
Poster Title: **Purification and Analysis of Human Liver Fatty Acid Binding Protein (FABP1)**

Abstract: Obesity is a chronic disorder, affecting over one third of Americans. It is characterized by excessive accumulation of adipose tissue in the body and increased risk for life threatening conditions. Human Liver Fatty Acid Binding Protein (LFABP) works to metabolize Long Chain Fatty Acids (LCFAs) taken up by the hepatocyte, resulting in altered regulation of metabolic processes, such as storage, oxidation, and gene expression. Studies have shown that LFABP contains a large binding pocket with a high affinity for various LCFAs, which allows LFABP to be a key regulator in lipid homeostasis within the liver. Absence of LFABP decreases hepatosteatosis and lipid storage in the liver, thus improving insulin sensitivity. This suggests that LFABP could be targeted in the prevention of hepatic steatosis. To facilitate this, LFABP was expressed in *Escherichia coli* and purified using nickel affinity column chromatography followed by delipidation through lipidex column chromatography to remove endogenous ligands. Binding affinity was measured through fluorescent 1,8-ANS competition assays. These studies offer the potential for identifying small molecule therapeutic inhibitors of LFABP in the prevention of liver steatosis.

Joseph Vavra
Independent Research-BTI

Joseph (Joe) Vavra is a sophomore at the University of Minnesota. He is majoring in biochemistry, with particular attention given to protein and enzyme function. In the future, he plans to study the relationship between form and function of proteins and enzymes to design more efficient enzymes for the purposes of developing faster, cleaner reactive pathways (i.e. more efficient cellulases). In his spare time, he likes reading science fiction and historical works, spending time outside, jogging, and listening to music.

Institution: University of Minnesota - Twin Cities
Faculty Mentor/Department: Dr. Carrie Wilmot/Biochemistry
Poster Title: **Investigation of Anion Binding to the Diheme Enzyme MauG**
Abstract: MauG is an oxygen-consuming diheme enzyme necessary for the synthesis of the endogenous cofactor tryptophan tryptophylquinone (TTQ) in methylamine dehydrogenase (MADH). The binding of the small anions nitrite, hypochlorite, cyanide, and formate to the oxygen-binding heme of MauG has been investigated. Small anions can act as mimics of gases or small ionic substrates and reveal binding electronics and geometries within heme-containing active sites. In the case of MauG, these studies will give insight into conformational changes induced by binding of oxygen that would otherwise be unobservable. Past experiments have been carried out using CO and NO gasses. By observing changes due to binding anionic ligands, the effects of binding small gasses versus anions as a simulation of oxygen may be compared. Evidence of anion ligation to the

heme iron was assessed through UV-visible absorption spectra of diferric MauG. Use of cyanide as a ligand produced the greatest shift in the wavelength of the dominating MauG heme Soret band, indicating cyanide was bound to the iron. Visualization of the MauG-cyanide complex is being investigated through x-ray diffraction. Obtaining diffraction quality MauG crystals through co-crystallization with MADH for exposure to cyanide has thus far proved elusive and will be the focus of future efforts.

Caleb Vogt
Heart, Lung, & Blood

Caleb Vogt is a senior studying Biomedical Engineering at Michigan Technological University. His focus is in tissue engineering and regenerative medicine. His goal is to obtain a MD/PhD in Biomedical Engineering to develop himself as a scientist who has an intimate knowledge of both research and the nuances of its application to patients. He enjoys camping, road trips, paint balling, and most other sports.

Institution: Michigan Tech University
Faculty Mentor/Department: Dr. Peter Bitterman/Pulmonary, Allergy, Critical Care and Sleep Medicine

Poster Title: **High Resolution Tissue Stiffness Mapping in Idiopathic Pulmonary Fibrosis**

Abstract: Idiopathic pulmonary fibrosis (IPF) is an interstitial lung disease, which causes affected lungs to become stiff as the alveolar spaces are invaded by fibroblasts and dense extracellular matrix (ECM). MicroRNA-29 (miR-29) is a crucial miR responsible for negatively regulating ECM in IPF. Loss of miR-29 contributes to fibroblast activation and subsequent fibrosis. Preliminary data shows that the cytoplasmic miR processing stage of miR-29 is aberrantly mediated through mechanotransduction from diseased extracellular matrix. Argonaute-2 (Ago2) is essential to modifying precursor miR into functional RISC complexes. In this work, human fibroblasts are cultured on decellularized IPF or control ECM to look for a change in Ago2 expression. It is determined that exposure to decellularized IPF ECM decreases Ago2 in human lung fibroblasts and that Ago2 is decreased *in vivo*.

Elizabeth Warren
Independent Research-AHSSRP

Elizabeth (Lizzie) Warren is currently a sophomore at Macalester College, majoring in biology and possibly minoring in chemistry and psychology. She plans to attend either medical or graduate school and is interested in pursuing a career in biomedical research related to regenerative medicine or cancer biology. In her free time, she enjoys hiking, reading, cooking,

and traveling when she gets the chance. Lizzie is also a member of the Macalester crew team and is involved in biology club.

Institution: Macalester College
Faculty Mentor/Department: Dr. Anna Lee
Poster Title: **The Role of PKCε in the Regulation of Nicotinic Acetylcholine Receptor Subunits α_6 and β_3**

Abstract: Addiction to nicotine in the form of tobacco is the leading cause of premature death worldwide, and while some drug treatments for nicotine addiction are effective in the short term, long-term cessation rates are low. Nicotinic acetylcholine receptors (nAChRs) containing the α_6 and β_3 subunits are known to play an important role in nicotine consumption and reward by modulating the release of dopamine. Protein kinase C epsilon (PKCε) is believed to regulate the expression of these subunits, but the molecular mechanism remains unclear. To investigate this pathway and identify potential novel drug targets, wild type and PKCε knockout mice were given chronic injections of nicotine (0.5 mg/kg IP for 10 days) or saline. Protein and mRNA samples from the ventral midbrain and striatum were obtained. We quantified α_6 and β_3 subunit mRNA levels by qPCR, and ERK and phospho-ERK protein by Western blotting. We found that wild type mice injected with nicotine expressed lower levels of α_6 and β_3 mRNA and showed decreased ERK activity compared with knockout mice. These data suggest that PKCε downregulates ERK, which in turn increases the expression of α_6 and β_3 subunits. By investigating the molecular mechanisms by which nAChRs are transcriptionally regulated, we can better understand nicotine addiction and identify additional drug targets for its treatment.

Ryan Wolfe
Heart, Lung, & Blood

Ryan Wolfe is currently a senior at Saint Cloud State University majoring in Biomedical Sciences. He is currently working under Dr. Ryan Fink as an undergraduate researcher in molecular microbiology and also has an emphasis on human anatomy and physiology. He is a nontraditional student, 27 years old, married, and a Staff Sergeant for the U.S. Army Reserves. He is planning to apply to the University of Minnesota in a microbiology related PhD program in December for the following academic year and has an interest in working on microbiomes.

Institution: St. Cloud State University
Faculty Mentor/Department: Dr. Michael Sadowsky/Soil, Water and Climate
Poster Title: **Biocontrol of Invasive Species: Eurasian Water Milfiol**

Abstract: Eurasian Water Milfoil (EWM) is an invasive plant species in Minnesota aquatic ecosystems. This species lake ecosystem functioning, aesthetic quality, and human health. EWM is able to grow earlier than native

species and is also able to grow from a fragments, creating a floating mat, displacing native species. EWM is also a potential host for human pathogens, causing the waters in and around the growth area to be unsafe for water recreational use. This issue causes a loss in recreational and tourism revenue for cities, and the state. The current method of dredging EWM causes damage to the lake floor, removes habitat for small fish and minnows, and also releases and disperses human pathogens. In this we are investigating the microbial ecology and microbiota of healthy and decaying EWM. This is done using high throughput sequence analysis of 16S rDNA amplicons obtained from EWM from sequencing analyses of three samples sites at Cedar Lake during the summer months. The samples have been obtained on a monthly basis for the past two years and will continue for another two years. We observed the elevated (~ 100-fold) increases in the number of fecal indicator bacteria (*Escherichia coli*) in EWM compared to that seen in the surrounding water. Sequence data is being analyzed now. Findings from this study will improve our understanding of ecological effects of EWM in Minnesota waterways across time and space and may provide insight for the development of microbe-based biocontrol agents.