UNIVERSITY OF MINNESOTA

2018 SUMMER UNDERGRADUATE RESEARCH SYMPOSIUM

Life Sciences Summer Undergraduate Research Program (LSSURP)

Faculty Director: Dr. Colin Campbell

Administrative Director: Dr. Jon Gottesman

Program Coordinator: Evelyn Juliussen

Presenter: Damilola Ademola-Green
Poster Number: 1
Home Institution: Hamline University
Program: LSSURP
Faculty Mentor: Dr. Kaylee Schwertfeger
Research Advisor: Chelsea Lassiter, Emily Irey
Poster Title: **Tumor-Stromal Interactions in Breast Cancer**
Abstract:

Breast cancer is one of the leading causes of cancer death among women. While there is not a cure to breast cancer there have been many treatments developed to fight the disease. However, some breast cancer lack treatment options such as triple negative cancer cell lines. These triple negative cell lines lack hormone receptors that most cancer cell lines have that allow them to be treated. This poses a threat to many victims with diseases involving these cells and similar cells. However, JAK/STAT signaling has shown to be activated in 70% of breast tumor, including triple negative cell lines. In a recent study, macrophages were treated with conditioned media from many different cancer cell lines, in hopes of finding a correlation between cancer cell lines and JAK/STAT signaling in macrophages. Of the many cell lines that were tested, it appeared that many triple negative cell lines activated JAK/STAT signaling in macrophages. This project analyzed 12 cell lines from this study in order to identify the factors that triple negative cancer cell lines secrete to activate JAK/STAT signaling in macrophages. Soluble factors were collected from the 12 cell lines then used to treat and test JAK/STAT macrophages. Results have confirmed that triple negative cell lines do secrete factors that activate JAK/STAT signaling in macrophage and that the factor is a protein, indicating there may be a mechanism that allows triple negative cell lines to activate JAK/STAT within macrophages.

Presenter: Joseph Alisch
Poster Number: 2
Home Institution: Providence College
Program: LSSURP
Faculty Mentor: Dr. Matthew Johnson
Research Advisor: Annie Brinda, Alex Doyle, Kelton Wilmerding, Jordan Krieg
Poster Title: **Understanding Changes in Local Field Potentials Using Electrode-Tissue Interface Modeling Following Deep Brain Stimulation Lead Implantation**
Abstract:

One consequence of Deep Brain Stimulation (DBS) lead implantation for the treatment of Parkinson's Disease (PD) is the immune response at the electrode-tissue interface (ETI). The presence of edema and tissue fibrosis can play a crucial role in regulating local field potential (LFP) recordings, which are currently the most promising biomarker for determining therapeutic stimulation parameters. This study seeks to model ETI dynamics in order to explain alterations seen in LFPs. A nonhuman primate was implanted with a DBS lead in the subthalamic nucleus, then LFPs and electrochemical impedance spectroscopy (EIS) were recorded over the following two weeks as the acute immune response progressed. Power spectral density analysis was used to analyze LFPs within the beta band (10 – 30 Hz). EIS was analyzed using equivalent circuit modeling (ECM) in order to characterize ETI dynamics. Abrupt changes in LFPs lined up temporally with those in ECM parameters. Many of the ECM parameters are significantly linearly-correlated with beta band LFP recordings, depending upon the bipolar electrode configuration. These results show that there is a potential relationship between tissue impedance and LFPs. With further studies, this model would help to characterize the ETI and the LFP signals needed for closed-loop DBS.

Presenter: Cierra Alleyne
Poster Number: 3
Home Institution: Hampton University
Program: LSSURP
Faculty Mentor: Dr. Margaret Titus
Research Advisor: Ashley Author
Poster Title: **The Role of Myosin 7 in a True Wild Type Amoeba *Dictyostelium Discoideum* (Ddb)**
Abstract:

When dealing with infectious pathogens it is crucial for leukocytes to quickly sense the wounded area and engulf destructive bacteria in a process called phagocytosis. Much previous research investigating phagocytosis in the model organism *Dictyostelium* was using an axenic lab strain that has a problematic mutation that alters their cytoskeleton. In the Titus lab, we are conducting research on true wild type *Dictyostelium* strains that show the proper behavioral conditions to produce dependable results. We were able to examine the different eating habits of the lab axenic and non-axenic or wild type strains of *Dictyostelium* by taking a time course of particle and liquid uptake using fluorescent microscopy. Based on the phagocytosis and pinocytosis assay results, the wild type Ddb was victorious in eating particles while the axenic strain devoured the sugar medium by pinocytosis. Additionally, the *Dictyostelium* cells appeared to show more filopodia during phagocytosis. The molecular motor, myosin 7 protein is a vital component in the devolvement of filopodia in non-axenic cells. Future work will focus on the function of myosin 7 in wild type cells by knocking out the myosin 7 gene and developing a GFP tagged myosin to look at myosin 7 localization during phagocytosis assay.

Presenter: Aliyah Allick
Poster Number: 4
Home Institution: Johns Hopkins University
Program: LSSURP
Faculty Mentor: Dr. Kelvin Lim
Research Advisor: Dr. Abhrajeet Roy
Poster Title: **Resting State Functional Connectivity of the Visual System in Mild Traumatic Brain Injury**
Abstract:

Blast-related traumatic brain injury (TBI), often causes visual system disruptions that persist years after the injury (Gilmore et al. 2016). Our goal is to perform an analysis of the resting-state visual system functional connectivity in mTBI patients. We hypothesized that the LGN's activity would be correlated with V1, fusiform gyrus, and the medial prefrontal cortex (Gilmore et al. 2016). Our methodology included the creation of functional connectivity maps for the bilateral fusiform gyri, lateral occipital cortex (LOC), lateral geniculate nucleus (LGN), V1, and medial prefrontal cortex. Additionally, pairwise correlations between the LGN and the other ROIs were calculated (Gilmore et al. 2016). There was a significant correlation between LGN and V1 in four out of five subjects, with Pearson's r between .13 and .28. Two subjects displayed a significant correlation between the LGN and the bilateral fusiform gyri. The decrease in the number of significant correlations can be attributed to the fact that the LGN is directly connected to V1 whereas areas like the fusiform gyrus are not directly connected to the LGN (Gilmore et al. 2016). However, a larger sample size and the addition of covariates (i.e., age, and mTBI severity) are needed to make definitive conclusions.

Presenter: Maria Alvarado Torres
Poster Number: 5
Home Institution: University of Puerto Rico - Mayagüez
Program: LSSURP
Faculty Mentor: Dr. Julie Olson
Research Advisor: Nhungoc (Ti) Luong
Poster Title: **Virus Distribution in Chronic Progressive Virus-Induced Mouse Model of Human Multiple Sclerosis**

Abstract: Epidemiological studies have suggested that infectious agents such as viruses can contribute to the development and progression of neurodegenerative diseases like human multiple sclerosis (MS). Thus, Theiler's murine encephalitis virus-induced demyelinating disease (TMEV-IDD) mouse model has been served as a useful experimental model to study MS. TMEV infection of susceptible mouse strains can lead to a life-long infection of the CNS and chronic progressive demyelinating disease. TMEV is known to establish persistence infection in the CNS-resident microglia. However, location and abundance of virus throughout the TMEV-IDD is yet identified. Thus, our current study attempted to utilize a novel in situ hybridization technique call RNA Scope to detect viral RNA at various days post infection, primarily the acute infection stage. Preliminary results suggest a high number of viral copies during the first few days of the infection, followed by a declined in viral numbers during the middle of the acute phase.

Presenter: Hermes Aponte Rivera
Poster Number: 6
Home Institution: University of Puerto Rico - Rio Piedras
Program: LSSURP
Faculty Mentor: Dr. Natalia Tretyakova
Poster Title: **Does Ubiquitination Play a Role in DNA-Protein Cross-Link Repair?**

Abstract: DNA-protein cross-links (DPCs) are bulky DNA lesions formed when proteins become covalently trapped on DNA strands following exposure to endogenous, environmental, or chemotherapeutic agents. Owing to their substantial size and helix-distorting nature, DPCs can interfere with the progression of replication and transcription, potentially contributing to mutagenesis, carcinogenesis, neurodegenerative and cardiovascular diseases. Unfortunately, due to their structural complexity, the mechanisms involved in DPC repair remain largely elusive. Literature suggests that DPCs are degraded by proteasomes into smaller peptide lesions, which can be subsequently removed through Nucleotide Excision Repair. We hypothesized that ubiquitination of DPCs serves as a signal for their proteasomal degradation and repair. To test this, we treated HT1080 cells with cisplatin, a DPC inducing agent, and MG132, a proteasome inhibitor, to evaluate ubiquitination of these lesions by dot blot analysis with anti-Ub antibody. Qualitative data shows that ubiquitin is involved in tagging DPCs for degradation, since cells treated with cisplatin displayed a higher signal. However, minimal differences in intensity between cells treated with only cisplatin compared to those with both cisplatin and MG132 suggest that while ubiquitin does tag these lesions, they are not subject to proteasomal degradation. Further experimentation is needed to elucidate the role of DPC ubiquitination in repair of these bulky lesions.

Presenter: Marcos Armendariz
Poster Number: 7
Home Institution: University of Texas - El Paso
Program: LSSURP
Faculty Mentor: Dr. Martina Bazzaro
Research Advisor: Edith Emmings, Mihir Shetty, Juri Habicht, Ashley Mooneyham
Poster Title: **UNC-45A Promotes Cancer Cells' Metastasis via Inhibition of the RhoA/ROCK Mechanotransduction Pathway**

Abstract: This project is designed to understand how the Non-Muscle Myosin II (NMII) co-chaperone UNC-45A promotes cell migration with the overall goal of defining UNC-45A as a novel molecular target for metastatic cancers. The rationale for this investigation is that: a) UNC-45A is a negative prognosticator of survival in ovarian, cervical, breast and melanoma cancers, b) its loss in cancer cells results in decreased cell motility, and c) its loss results in increased acto-myosin contractility. Because the RhoA/ROCK mechanotransduction pathway is the main regulator of acto-myosin contractility and its increased activity suggests it is a negative regulator of cell motility, our working hypothesis is that UNC-45A promotes cancer cells' metastasis via inhibition of the RhoA/ROCK mechanotransduction pathway. Here we show that the reduced cancer cells' migration following genetic silencing of UNC-45A is accompanied by increased activation of the ROCK downstream effectors including NMII, MYPT1, and cofilin. Furthermore, loss of UNC-45A in cancer cells is accompanied by an increase in stress fibers, which is a phenotype consistent with increased acto-myosin contractility and cell adhesion. Taken together, our preliminary data suggest that UNC-45A is a potential molecular target for disrupting cancer cell migration that is driven by cell mechanics.

Presenter: Cecilia Barajas
Poster Number: 8
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. George Wilcox
Research Advisor: Cristina D. Peterson, Kelsey R. Pflepsen, Jennifer Cook, Kelley F. Kitto, Lucy Vulchanova, Carolyn A. Fairbanks
Poster Title: **Efficacy and Side Effect Monitoring of AAV Gene Therapy for Chronic Pain**

Abstract: Since 2013, the number of synthetic opioid overdose deaths has been rising exponentially, thus increasing the demand for alternative treatment options for patients living with chronic pain. It has been previously shown that agmatine, or decarboxylated L-arginine, is effective at reducing hyperalgesia in neuropathic pain models. Here we describe a technique in which the amount of endogenous agmatine is synthetically augmented by genetically overexpressing the enzyme responsible for its production, arginine decarboxylase (ADC). Considering previous adverse effects seen clinically when using adeno-associated viruses (AAV), we also investigate the degree of immune response of animals treated with the vector against their matched controls.

Presenter: Elizabeth Baum
Poster Number: 9
Home Institution: University of Toledo
Program: LSSURP
Faculty Mentor: Dr. Zhi Yang
Research Advisor: Dr. Teris Tam, Tong Wu
Poster Title: **Mechanical Design for a 3D Printed Neuroprosthetic Hand with Partial Hand Amputee Compatibility**

Abstract: Prosthetic hands have been used to assist upper limb amputees for decades. However, the existing prosthetic hands are expensive and the control schemes are limited in functionality and intuitiveness. In addition, there is a lack of prosthetic hands compatible with partial hand amputees. When designing a prosthetic hand, one must consider ease of reproduction and modification, low expense, degrees of freedom (DOF), lightweight components, and many other key characteristics. We present the design of a prosthetic hand with 6 DOF compatible for partial hand amputees that could be 3D printed with basic solid filament such as ABS. Finger flexion is achieved through a linkage system that is driven by linear actuators instead of the commonly used DC motor. The thumb incorporates 2 DOF using both a linear actuator and a DC motor. With the completion of this project, we were able to model, print, and construct a prosthetic hand with the modification, lightweight, and low cost characteristics we were striving for.

Presenter: Greta Becker
Poster Number: 10
Home Institution: Loras College
Program: LSSURP
Faculty Mentor: Dr. Gregory Vercellotti
Poster Title: **Endothelial Cell-Associated Sialic Acid and Sickle Cell Disease**

Abstract: Sickle hemoglobin (HbS) polymerizes under decreased oxygen conditions leading to alterations in erythrocyte physiology in patients with sickle cell disease (SCD). One of the many complications resulting from SCD is vascular inflammation caused by the release of free heme from sickle erythrocytes and a constant cycle of ischemia/reperfusion. Released heme activates toll-like receptor 4 (TLR4) leading to the activation of the pro-inflammatory NF-kB transcription factor and the expression of adhesion molecules, such as P-selectin and von Willebrand factor (vWF), which promote vaso-occlusion. Previous research indicates that the formation of the TLR4 complex requires the removal of α-2,3 sialyl residues from TLR4 by Neuraminidase-1. We hypothesize that treatment of human umbilical vein endothelial cells with a bacterial neuraminidase, prior to treatment with heme or LPS, will enhance TLR4 activation of NF-kB and cell-surface expression of P-selectin and von Willebrand factor. We measured vWF and P-selectin on endothelial cells stimulated with heme or LPS with and without neuraminidase. We found that treatment of endothelial cells with a neuraminidase does not lead to significant increases in vWF and P-selectin expression.

Presenter: Sarah Becknell
Poster Number: 11
Home Institution: Berea College
Program: LSSURP
Faculty Mentor: Dr. Matthew Clark
Research Advisor: Dr. Julie Grossman
Poster Title: **Quantifying the Effects of Summer Cover Crops on Soil Quality through Measurement of Particulate Organic Matter (POM)**

Abstract: Soil organic matter (SOM) can provide ecosystem services by contributing to soil fertility and preventing nutrient leaching. Cover crops are non-harvested plants grown for their ecosystem benefits. Among the most important is cover crops capacity to decompose in soil to become SOM, contributing to soil quality. One way to quantify the effects of cover crops on soil quality is to measure particulate organic matter (POM), which is actively decaying organic matter in the soil. In this project we measured the concentration of coarse fraction (CF) POM (POM+ sand) at three dates within a growing season as affected by four cover crop species treatments grown for two lengths of time, compared to bare control plots. CF-POM concentration did not differ across treatments or timing, despite significant differences in cover crop biomass. Preliminary soil texture analysis indicates that differences in sand concentration may mask difference in CF-POM concentrations. Further research will explore whether high microbial activity limited the amount of freshly decaying plant matter in soil. The results of this study suggest that changes in POM over one season of cover crops may be too small to measure as CF-POM, and more research is needed to determine whether summer cover crops are an effective tool to improve soil quality by increasing POM.

Presenter: Jovany Betancourt
Poster Number: 12
Home Institution: Florida International University
Program: LSSURP
Faculty Mentor: Dr. Alexander Khoruts
Poster Title: **Detection and Classification of Gut Bacteria in a Mouse Sepsis Model**

Abstract: Sepsis, or septicemia, is characterized by systemic inflammatory response that can lead to damage of multiple organ systems. It is responsible for about 10% of fatalities in patients treated in intensive care units. Standard care of these patients typically involves administration of broad-spectrum antibiotics and interruption of enteral nutrient flow. While these interventions are important to combat infections, they also suppress and disrupt the indigenous microbiota. Our central hypothesis is that the damage to the host gut microbiota results in a weakened gut barrier, increased translocation of pathosymbionts into the host, and altered host immune response that in totality lead to poor outcomes. Here we develop a technique in the laboratory to measure the amount of circulating bacterial DNA in blood using quantitative PCR and characterize its composition using 16S rRNA gene profiling. Specifically, we use the Cecal Ligation Puncture (CLP) mouse model of sepsis, where the cecum is ligated and perforated. Control animals receive sham surgery. The circulating microbiome in the animals will be compared to the microbiome in the cecum to determine which organisms are more able to penetrate into the bloodstream.

Presenter: Gauri Binoy
Poster Number: 13
Home Institution: Cornell University
Program: LSSURP
Faculty Mentor: Dr. Laurie Parker
Poster Title: **Determination of Direct Substrates of PDK1 Kinase Through Novel KALIP-KINATEST-ID Workflow**

Abstract: Kinase proteins are critical to the function of cell signaling pathways and regulate various components ranging from metabolism to the cell cycle. Mutations in kinases cause a wide range of disorders, most notably cancer. Despite having developed inhibitors for kinases, the unique substrate preferences for each are unknown, which is critical for effective cancer targeting methods. Substrates allow for kinases to phosphorylate proteins and help assay the enzymatic activity in kinases. PDK1 is a serine-threonine kinase involved in P13K and RAS pathway activity, and when dysregulated, can lead to many types of cancer. The KALIP (kinase assay linked to phosphoproteomics) process coupled to the KINATEST-ID workflow was used to determine the direct substrates of PDK1. This coupled method uses a recombinant kinase reaction with a trypsin digest, and then uses mass spectrometry to identify phosphopeptides. This is then used as input into the KINATEST-ID algorithm to determine the kinase substrate motif preferences. These preferences will be critical to ultimately design a peptide biosensor for PDK1 for use in vivo and in vitro. Developing biosensors for this kinase protein will help monitor its enzymatic activity and to screen for novel inhibitors in future assays.

Presenter: Autumn Brunson
Poster Number: 14
Home Institution: Tennessee State University
Program: LSSURP
Faculty Mentor: Dr. Wensheng Lin
Research Advisor: Dr. Sarrabeth Stone
Poster Title: **The Role of the Unfolded Protein Response on the Cuprizone Model of Remyelination**

Abstract: Oligodendrocytes are glial cells of the central nervous system that myelinate neuronal axons. Myelin wraps around neuronal axons, allowing rapid transmission of electrical impulses between neurons. During active myelination oligodendrocytes produce large quantities of proteins making them extremely susceptible to disturbances in protein homeostasis. Unresolved endoplasmic reticulum (ER) stress, caused by the accumulation of unfolded/misfolded proteins, activates the unfolded protein response (UPR). The UPR consists of three signaling pathways, pancreatic endoplasmic reticulum kinase (PERK), inositol-requiring enzyme (IRE1), activating transcription factor 6a (ATF6a). We employed the cuprizone remyelination model to investigate the role of the UPR in remyelination using a mouse model lacking PERK and ATF6a in oligodendrocytes. loxPERK+/+; PLP/CreERT; ATF6α-/- mice are ATF6α deficient and utilize tamoxifen induced Cre-Lox recombination to knockout the PERK gene specifically in oligodendrocytes. 8 week old male loxPERK+/+; PLP/CreERT; ATF6α-/- mice and controls were treated with cuprizone for 6 weeks, and were treated with tamoxifen for 10 days following cuprizone removal. Mice were perfused 9 weeks after starting cuprizone, and myelin integrity and oligodendrocyte number in the corpus callosum was assessed by histology. Remyelination failure is a common problem in myelin disorders. This study will enable us to determine the role of the UPR in oligodendrocytes during remyelination.

Presenter: Naomy Candelaria Morales
Poster Number: 15
Home Institution: University of Puerto Rico - Utuado
Program: LSSURP
Faculty Mentor: Dr. Mary Rogers
Research Advisor: Nathan Hecht, Kat LaBine, Jennifer Nicklay
Poster Title: **Evaluating Key Pest and Beneficial Arthropods in Urban Community Garden Sites in the Twin Cities Metro Area**

Abstract: In recent decades, more than half of the global population has integrated into cities, creating dense urban environments. However, not all urban neighborhoods are well developed, and many are experiencing economic, social, and ecological decline. For example, because of dramatic changes in the economy of some areas, there has been a decrease in purchase and sale of properties, creating vacant or abandoned lots. If a vacant lot loses all of it's real estate value it's considered a permanent vacant lot, contributing to the fractioned development of cities, in which relative population loss, extended economic decline, or both can be experienced in certain urban areas. Such vacant lots can be an opportunity to revitalize urban spaces into enriching areas like urban community gardens, which can provide nutritional, health, social, and ecological benefits, especially in low-income neighborhoods. While directly managing the flora in community based gardens has a large influence on their growth, other organisms play a key role in their development. Arthropods are an important part of the interactions that take place. Though some are pests, many contribute to the vitality of our productive green areas. These arthropods are considered beneficial, and provide ecosystem services such as pollination, pest management, recycling of nutrients, decomposition of plants and animal waste, and soil aeration. Arthropods also serve as food for fish, birds and other living organisms. Though it is known that aboveground plant diversity contributes to the diversity and abundance of arthropods, this has not been extensively studied in urban, highly-managed areas. In this study, we investigated arthropod diversity across four urban community garden sites in the Twin Cities metro area using visual plant scouting, pitfall traps, and sticky cards over a four week period during summer, 2018. Our results highlight key pests and beneficial insects common to urban community garden sites, reveal trends based on surrounding plant diversity, and can be used for follow up studies with the goal of improving urban ecosystem functioning by conserving beneficial arthropods.

Presenter: Nathan Castro Llanos
Poster Number: 16
Home Institution: University of Puerto Rico - Arecibo
Program: LSSURP
Faculty Mentor: Dr. Maxim Cheeran
Research Advisor: Venkatramana Krishna, Andrew Crane, Wei-Cheng Lu, Feng Xiao, Walter Low, Andrew Grande
Poster Title: **Neuroinflammation Ensuing Traumatic Brain Injury by Controlled Cortical Impact in a Rodent Model**

Abstract: Treatment options for Traumatic Brain Injury (TBI) are scarce and those available are mainly palliative. Previous studies have shown that inflammatory responses persist following TBI (Lagraoui et. al. Frontiers in Neurology. 2012; 3:155); and are linked to neurodegenerative diseases later in life. Human derived, non-hematopoietic umbilical cord blood stem cells (nh-UCBSCs) have shown a beneficiary role in treating ischemic stroke by modulating immune responses. Thus, we hypothesize that treatment of TBI with nh-UCBSCs would decrease inflammatory responses and increase beneficial outcomes. TBI was inflicted on rats using a Controlled Cortical Impactor (CCI) and 48 hours post injury, the animals were treated intravenously with nh-UCBSC. Brain tissue samples from different groups (Control, Treated, and Vehicle-treated) were collected at 7 days after the injury and analyzed for immune cell phenotypes by Flow Cytometry. This study may provide future insight into understanding the immune responses after TBI and develop newer treatments.

Presenter: Crystal Cheng
Poster Number: 17
Home Institution: Cornell University
Program: LSSURP
Faculty Mentor: Dr. Scott Dehm
Research Advisor: Alex Ling, Jacob Hildebrand, Mark Daniel, R. Stephanie Huang Ph.D.
Poster Title: **Examining Drug Sensitivity Predictions in Prostate Cancer Cells**

Abstract: The androgen receptor (AR) plays an important role in prostate cancer (PCa) progression. Inhibition of AR function is beneficial for men with locally advanced or metastatic disease. However, emergence of castration resistant prostate cancer (CRPC) is responsible for almost all death. Contributing to this state of resistance are constitutively active AR variants (AR-Vs), which are known drivers in CRPC. Substantial experimental evidence exists to support the concept that AR-Vs mediate resistance to endocrine-based therapies; however, the field is unaware how expression of AR-Vs may influence sensitivities to non-endocrine therapies. To address this, we used microarray expression data from a pair of isogenic prostate cancer cell lines that were engineered to express either full-length AR or an AR-V. Using a drug sensitivity prediction algorithm, we were able to nominate three drugs with differential drug sensitivities, Navitoclax, Tozasertib, and Nutlin-3. These drugs were evaluated for their effects on cell viability using crystal violet assays, and the half maximal inhibitory concentration (IC50) values were determined. Our results show that AR-V-expressing cells were more sensitive to Navitoclax, a BCL-2 inhibitor, compared to full-length AR-expressing cells, however the opposite was true in cells treated with Tozasertib, an Aurora Kinase inhibitor. The high sensitivity of PCa cells to these drugs indicates they should be evaluated further as potential PCa therapies.

Presenter: Keislamarí Cintrón Berrios
Poster Number: 18
Home Institution: Interamerican University of Puerto Rico
Program: LSSURP
Faculty Mentor: Dr. Neil Anderson
Poster Title: **Effects of Organic and Inorganic Fertilizers on Lettuce and Kale Grown in Non-Circulating Hydroponic Systems**

Abstract: Hydroponics is a method of growing plants in a water-based, nutrient-rich solution. Hydroponics is important because it helps to economize water and fertilizers, providing an inexpensive method of crop production. Non-circulating hydroponic systems refers to the hydroponic systems where the water does not circulate among plants whereas circulating systems have the solutions circulated throughout all plants in the cropping system. The use of organic sources of nutrients may help to increase sustainability of vegetable production through the promotion of nutrient recycling. We compare different organic and inorganic fertilizers in non-circulating hydroponic systems grown with lettuce (*Lactuca sativa*) and kale (*Brassica oleracea*). We hypothesized that even though all fertilizers were used at the same nitrogen level, the fertilizers source would have a significant effect on plant growth, with the inorganic fertilizers having the greatest effect. Once a week for six weeks, we measured the water quality (pH, electrical conductivity and nitrate level) and plant quality (growth, health, chlorophyll, diameter and number of leaves); fresh and dry weights were measured on week six. In conclusion, plants with inorganic fertilizers have higher yields and is possible to grow leafy vegetables in non-circulating hydroponic systems.

Presenter: Hinsoukpo Dagan
Poster Number: 19
Home Institution: North Hennepin Community College
Program: LSSURP
Faculty Mentor: Dr. Carrie Wilmot
Poster Title: **Expression, Purification and Crystallization Of OleC: A Beta-lactone Synthetase**
Abstract: OleC is an enzyme involved in synthesis of β-lactones, a class of molecules that includes compounds with anti-bacterial, anti-tumor, and anti-obesity properties. β-lactone synthetase, is a stable monomer that along with more than sixty other confirmed β-LS enzymes, a large, new family within the ANL superfamily. In 2010, our lab was able to crystallize *Stenotrophomonas maltophilia* (Sm) OleC and, were able to collect X-ray diffraction data, but we were unable to phase that data set. However, to get our data set phased, we turned to selenomethionine labeled preps. The primary difficulty in crystallizing OleC is the protein propensity to aggregation. The expressed protein was purified using Ni-NTA agarose, an affinity chromatography matrix for purifying recombinant proteins carrying a His tag. Followed by size exclusion chromatography optimization of buffer additives to evaluate the effect of different detergents. The most effective additive was CHAPS, a zwitterionic detergent, with the least aggregation of our purified protein. Our protein has been sent for broad crystallization screening and we identified over 20 crystallization conditions and are now attempting to reproduce them.

Presenter: Olivia Drake
Poster Number: 20
Home Institution: Juniata College
Program: LSSURP
Faculty Mentor: Dr. Sarah Heilbronner
Poster Title: **Tract-Tracing in Rhesus Macaques Reveals Anatomical Connectivity of the Posterior Cingulate Cortex and Medial Temporal Lobe**
Abstract: Brain connectivity often correlates to function, and the understanding of connectivity may also lend itself to bettering the diagnoses and treatments of psychological disorders. One brain network that is often disrupted in psychological disorders such as Alzheimer's and depression is the default mode network (DMN). The exact function of the DMN is disputed. It is well known that areas within the DMN are functionally correlated, but whether they have anatomical connections remains less clear. Using tract-tracing in rhesus macaques, we investigated the connectivity between the posterior cingulate cortex (PCC), a primary node in the DMN, and the medial temporal lobe (MTL). The activity in these areas has previously been found to be abnormal in those with disorders, such as Alzheimer's, schizophrenia, depression, and autism. We found that these areas do have axonal connections to each other, primarily between the PCC and the entorhinal cortex of the MTL. Our findings offer information about the exact function of the DMN and could lead to a target for treatment of some disorders. These results can also be used to compare macaque to rat brain connectivity to determine whether rats are an appropriate DMN model for humans.

Presenter: Danielle Dudley
Poster Number: 21
Home Institution: Normandale Community College
Program: LSSURP
Faculty Mentor: Dr. David Zarkower
Research Advisor: Kellie Agrimson, Anna Minkina, Robin Lindeman, Vivian Bardwell
Poster Title: **Liver Receptor Homolog 1 (LRH1) is Required in Spermatogonial Stem Cells for Normal Spermatogenesis in the Mouse Testis**

Abstract: Spermatogenesis is the continuous process by which spermatogonial stem cells (SSC) differentiate into mature spermatids in the testis via a series of mitotic and meiotic cellular divisions. This process is dependent upon SSC pool maintenance throughout the entire male reproductive lifetime. Misregulation of the SSC population can result in germ cell depletion and eventually male infertility. Liver Receptor Homolog 1 (Lrh1) is an orphan nuclear receptor transcription factor known to be important for metabolic, steroidogenic, and developmental processes throughout the body. Lrh1 is expressed in several cell types in the testis, including germ cells. To evaluate its function, we used a Cre-lox mouse breeding scheme to specifically delete Lrh1 in germ cells. We performed histological analyses on testes from mice aged from 9 days post-partum to six months post-partum. At seven weeks most Lrh1 germ cell-specific knockout testicular tubular cross sections appeared relatively normal but a few tubules were completely depleted of germ cells. By six months, Lrh1 knockout testes were considerably smaller in size than controls and most tubule cross sections lacked germ cells. Based on this progressive loss of germ cells we conclude that Lrh1 likely plays a role in SSC self-renewal. Future studies will investigate the signaling pathways controlled by LRH1 expression and how these affect SSC maintenance.

Presenter: Elise Dunshee
Poster Number: 22
Home Institution: State University of New York - Binghamton University
Program: LSSURP
Faculty Mentor: Dr. Aaron Goldstrohm
Poster Title: **Functional Mapping of *Drosophila* Pumilio to Identify Regions Involved in mRNA Regulation**

Abstract: The RNA-binding protein Pumilio (Pum) represses gene expression by stimulating decay of specific messenger RNAs (mRNAs). Pum is required for embryogenesis, germline stem cell maintenance, and neurological function. Furthermore, changes in expression of mammalian Pum orthologs are linked to cancer and neurological disease. *Drosophila* Pum consists of a conserved RNA binding domain and three N-terminal Repression Domains (RDs), possessing independent activity. While Pum utilizes deadenylation and decapping factors to mediate repression, the role of each Repression Domain (RD) in this mechanism is largely unknown. To identify motifs within RD1 that facilitate key functional interactions, we deleted conserved regions in RD1 and quantified repressive function using cell-based reporter assays. Although our data suggest that none of the conserved regions are individually necessary for function, we identified a short divergent motif that is partially important for activity. We conclude that RD1 may contain multiple functional motifs, and these may not be conserved. Ongoing work includes refining each functional motif. Once minimal motifs are identified, we will use these peptides in biochemical assays to study interactions with cofactors. Overall, a better understanding of RD structure-function relationships will further elucidate the mechanism of Pum-mediated repression and inform future studies on its role in disease.

Presenter: Samantha Ealy
Poster Number: 23
Home Institution: Hamline University
Program: LSSURP
Faculty Mentor: Dr. Lawrence Wackett
Research Advisor: Serina L. Robinson
Poster Title: **Purification and Characterization of an Enzyme Producing the Antibiotic Ebelactone**
Abstract:

β-Lactones resemble β-lactams, the latter are the major class of antibiotics, including penicillin, that have saved millions of lives over the last eighty years. There is rising concern over resistance to β-lactam antibiotics, sparking a call to discover new natural products for antibiotic and other medicinal uses. Recently, β-lactones have been investigated as potential drugs with antiobesity, anticancer, and antimicrobial properties. The discovery of the first β-lactone synthesizing enzyme was OleC, part of an olefin synthesis pathway in the bacterium *Xanthomonas campestris*. Other proteins with high percent identity to OleC have been identified and tested for catalysis of β-lactone ring formation. This has led to the discovery of OleC homologs that can process a variety of β-hydroxy acid substrates. Orf1 from the bacterium *Streptomyces aburaviensis* and NltC from the bacterium *Nocardia brasiliensis* are two such enzymes. The NltC pathway synthesizes trans β-lactones whereas the OleC pathway synthesizes cis β -lactones. This research focuses on Orf1 which has been found to process terminal β-hydroxy acids, whereas OleC processes β-hydroxy acid moieties that are substituted on the carbons bearing both the hydroxyl group and the carboxylate. Orf1 is not part of an olefin synthesis pathway, but a polyketide synthesis pathway that synthesizes ebelactone A, an antimicrobial natural product. In this project, Orf1 was expressed successfully in *E. coli* BL21(DE3) cells with a histidine tag and purified via fast protein liquid chromatography. The enzyme showed activity on a terminal β-hydroxy acid, as predicted. In the presence of ATP, Orf1 catalyzed ring closure of 3-hydroxymyristic acid and produced a β-lactone. Further research could include testing a wider substrate range, analyzing products via NMR or FTIR, and testing with its native substrate ebelactone A. This research contributes to the widening the substrate range of β-lactone synthetases to enzymatically produce important products and pharmaceuticals.

Presenter: Olumide Fagboyegun
Poster Number: 24
Home Institution: Ann Arundel Community College
Program: LSSURP
Faculty Mentor: Dr. Michael Lee
Research Advisor: Philipp Portz, Joyce Meints
Poster Title: **Effect of Casein Kinase 2 α' On HSF1 Degradation in Parkinson'S Disease**
Abstract:

Abnormal α-synuclein (αS) aggregation and neurodegeneration are hallmark features of Parkinson's disease (PD). Heat shock transcription factor 1 (HSF1) is a protein responsible for regulating protein folding via heat shock proteins (Hsps) and is transcriptionally activated in response to cell stress and certain disease states. Reports suggest that HSF1 may be degraded in PD via phosphorylation by casein kinase 2a' (CK2α') and we hypothesized impaired HSF1 function may contribute to PD pathophysiology. We sought to determine whether HSF1 phosphorylation and degradation are increased in PD due to upregulated CK2a'. To test this, brains from A53T mutant αS-expressing PD transgenic (Tg) and non-transgenic (nTg) mice were analyzed via immunoblot analysis and immunohistochemistry. We found, compared to nTg and pre-symptomatic Tg mice, end-stage transgenic mice showed a significant reduction in expression of HSF1 and two Hsps (90, 70), an increase in expression of Hsp25, and no change in expression of CK2α' and Phosphorylated HSF1 in the brainstem/spinal cord regions. These results indicate that the αS-A53T mutation in our mouse model has no effect on the expression of CK2α' or phosphorylated HSF1. However, the significant HSF1 reduction likely facilitates PD symptom progression. Further research is needed to clarify the mechanism of HSF1 degradation.

Presenter: Joseph Floeder
Poster Number: 25
Home Institution: Fordham University
Program: LSSURP
Faculty Mentor: Dr. Paul Mermelstein
Poster Title: **Behavioral Differences in Optogenetic Self-Stimulation in Male and Female Mice**
Abstract: A substantial amount of scientific literature is devoted to studying sex differences in addiction. While the causes of these sex differences are not fully understood, it is clear that females have a heightened vulnerability to drugs of abuse and progress from being casual drug users to addicts more quickly than males. In this project, we set out to test for sex differences in the glutamatergic pathway between the infralimbic cortex and the nucleus accumbens shell, a pathway known to be involved in motivated behavior, by using optogenetic self-stimulation in mice. Mice were tested in a two-sided real time place preference chamber and received optogenetic stimulation on one side of the chamber at three different frequencies: 10, 20, and 30 Hz. We hypothesize that at higher intensities of stimulation, both the male and female mice will develop a preference for the side of stimulation while only the females would develop a preference for the side of stimulation during lower intensities. We propose that associations between glutamate receptors and estrogen receptors in this specific neural pathway are responsible for the heightened response to drugs of abuse that females exhibit. My project can provide a stronger understanding of the role this pathway plays in sex differences and potentially lead to techniques that address the issue of female susceptibility to drugs of abuse.

Presenter: Dylan Forenzo
Poster Number: 26
Home Institution: Rutgers University
Program: LSSURP
Faculty Mentor: Dr. Stephen Engel
Research Advisor: Dr. Kendrick Kay
Poster Title: **Measuring Cortical Magnification in 181 Human Subjects**
Abstract: Visual cortex contains multiple maps of the visual field, and past work has revealed that a large portion of cortex is devoted to the relatively small portion of the visual field at the center of the gaze. However, the exact amount of this cortical magnification, and how it varies between individuals, is still unknown. To address this problem, we built a tool to measure cortical magnification in 181 subjects, using fMRI data from the Human Connectome Project (HCP). Data contained the coordinates of the visual field location most closely associated with each location in visual cortex; the eccentricity coordinate specified distance from center of gaze. The tool allowed users to visualize these data and manually trace iso-eccentricity lines that connected cortical points that responded best to stimuli at 0.5, 1, 2, 4, and 7 degrees eccentricity. The relationship between surface areas enclosed by different regions bounded by these contours will determine the cortical magnification (e.g. how much cortex per degree falls between the 0.5 and 1 deg lines vs between 4 and 7 deg) . Comparing multiple subjects will determine individual variation. Preliminary results on 100 brains already reveal substantial variability in cortical magnification.

Presenter: Johnathan Germick
Poster Number: 27
Home Institution: Iowa State University
Program: LSSURP
Faculty Mentor: Dr. Andrew Oxenham
Poster Title: **Modified Least Squares Fitting Algorithm for use in a pRF Model for The Auditory Cortex**
Abstract: The population receptive field (pRF) model has been shown to be an effective means of determining response properties of voxels in the visual cortex and, to a lesser extent, the auditory cortex. Up to now, the stimuli used for pRF mapping in the auditory cortex have been limited to pure tones. To begin to study the cortical representations of more complex sounds, such as harmonic complex tones, we propose using pitch and "brightness" (i.e., timbre) as two distinct perceptual dimensions in the auditory pRF model. As a starting point for this larger project, we developed our pitch-timbre symmetric Gaussian pRF model and applied it to simulated fMRI data with additive white Gaussian noise to assess its viability. The model performance was measured using several optimization strategies, including nonlinear least squares fitting (LSQ), particle swarm optimization (PSO), and variants of these methods. The results have revealed that successful analysis depends on careful design of the optimization parameters. For example, to accurately recover the receptive bandwidth of a given voxel, the primary benefit of the pRF model, the initial seed of the LSQ should be algorithmically selected to avoid local minima without the necessity for other global optimization schemes.

Presenter: Kristen Gregory
Poster Number: 28
Home Institution: University of Arkansas - Little Rock
Program: LSSURP
Faculty Mentor: Dr. Marco Pravetoni
Poster Title: **Determining the Role of IgG Effector Functions in Mediating Oxycodone Vaccine Efficacy**
Abstract: The opioid abuse and overdose epidemic has been declared a national crisis in the United States. Current treatments for opioid abuse show sub-optimal efficacy in preventing relapse and fatal overdoses. Vaccines against opioids could be a promising novel therapeutic strategy. Vaccines elicit opioid-specific antibodies that reduce opioid effects in the brain. Our lab has previously shown that depletion or ablation of interleukin 4 (IL-4) increases vaccine efficacy against oxycodone by altering the quality of the antibody response in mice. This study investigates the role of IgG antibody subclasses in mice treated with an oxycodone vaccine combined with an anti-IL-4 monoclonal antibody (mAb). Experiments focused on in vivo and in vitro assessments of IgG-mediated phagocytosis of oxycodone-antibody immune complexes. First, we tested the role of FcγRCIIIA receptors, which are expressed on macrophages and monocytes. Wild-type and FcγRCIIIA knock-out (-/-) mice were immunized with an oxycodone vaccine in combination with an anti-IL-4 mAb. Immunized FcγRCIIIA-/- mice showed higher oxycodone-specific IgG titers than wild-type mice, but did not show altered vaccine efficacy. Additionally, we did not observe Fcγ receptor-mediated phagocytosis of oxycodone-specific antibodies *in vitro*. Future studies will focus on the role of Fcγ receptors in removal of antibody bound opioids

Presenter: Abigail Gress
Poster Number: 29
Home Institution: Purdue University
Program: LSSURP
Faculty Mentor: Dr. Yoji Shimizu
Research Advisor: Brandon Burbach, Stephen O'Flanagan, Meredith Song, Meagan Rollins
Poster Title: **The Effects of Cytokine Type and Inflammation vs. Infection on Tissue Populations of Memory CD8+ T cells**

Abstract: Cytotoxic T cells (CTL) destroy damaged cells during the adaptive immune response. Upon exposure to a cognate antigen, naive CD8+ T cells are activated and expand to form effector CTL. A subset of CTL are retained as memory cells which aid in protection from future reinfection or antigen encounter. Cytokines such as interleukin 12 (IL12) and 2 (IL2) are thought to affect the effector and memory functions of CTL during activation and contraction. However, the impact of these cytokines on tissue distribution of CTL is unclear. Cells are damaged via different mechanisms, including infection, tumors, and trauma. The goal of this project is to determine whether cytokine type and systemic infection vs local inflammation affect the number of memory CTL localized in tissue. We hypothesize that higher concentrations of IL12-activated CTL will preferentially populate the site of inflammation and evenly populate tissues during systemic infection. These experiments have potential to optimize cancer immunotherapy treatments that utilize CTL. This project was conducted through treating SPF mice with a localized dose of 2,4-dinitrofluorobenezene irritant on the skin or systemically infecting with vesicular stomatitis virus. Four days post-treatment, the mice were injected with CTL primed with a combination of IL12 and IL2. Tissue harvests were conducted both four and 30 days post-CTL transfer; various tissues were analyzed for CTL populations via flow cytometry.

Presenter: Taylor Hammonds
Poster Number: 30
Home Institution: University of Arkansas - Pine Bluff
Program: LSSURP
Faculty Mentor: Dr. Dorothy Hatsukami
Research Advisor: Dr. Dana Carroll
Poster Title: **Racial Differences in Biomarkers of Tobacco-Related Exposure among Smokers**

Abstract: Blacks have higher rates of smoking-related diseases than Whites. Exposure to tobacco toxicants is the first step to developing smoking-related disease. We hypothesized that Black smokers would have higher levels of tobacco-related exposure than Whites. Baseline urine from a RCT of adult smokers were analyzed for the following urinary biomarkers of exposure: total nicotine equivalents(TNE), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol and its glucuronides(total NNAL), 3-hydroxypropylmercapturic acid(3-HPMA), 2-hydroxypropylmercapturic acid(2-HPMA), 3-hydroxy-1-methylpropylmercapturic acid(HMPMA), S-phenylmercapturic acid(SPMA), 2-cyanoethylmercapturic acid(CEMA), and phenanthrene tetraol(PheT). Levels of overall and per cigarette were summarized by race using geometric means adjusted for age, gender, BMI, menthol, and duration of smoking. Statistical significance was considered at a $p<0.05$. Compared to Whites, Blacks smoked fewer cigarettes per day (20 vs 15) and had lower TNE levels (64.5 vs 47.7 nmol/mg/creatine). Whites and Blacks did not differ in TNE per cigarette (3.7 vs 3.5 nmol/mg/creatine). Whites had higher levels than Blacks of total NNAL, 3-HPMA, 2-HPMA, HMPMA, SPMA, CEMA, and PheT. Per cigarette, Whites and Blacks did not differ in the majority of the biomarkers. The higher exposure among Whites was driven by Whites consuming more cigarettes per day. Higher rates of tobacco-related disease observed among Blacks is likely not due to exposure. Additional factors along the pathway to disease that occur after exposure, such as detoxification or DNA repair, should be examined.

Presenter: Annellie Harlos
Poster Number: 31
Home Institution: St. Louis University
Program: LSSURP
Faculty Mentor: Dr. Theoden Netoff
Poster Title: **Optimal Stimulation Parameters for Deep Brain Stimulation Treatment of Epilepsy with Bayesian Optimization**

Abstract: Epilepsy affects about 1% of the population worldwide. While some cases of epilepsy are well-controlled with medication, about 30% of patients do not achieve satisfactory control of their seizures [1]. Deep Brain Stimulation (DBS) is a recently implemented therapy for this population. However, finding the optimal stimulation parameters, such as frequency and amplitude, can be difficult. This is due to the extraordinary amount of possible parameter combinations and that optimal settings can vary from patient to patient. Bayesian optimization is an algorithm that uses a Gaussian process model to efficiently explore parameter spaces for variables within a system. In this experiment, Bayesian Optimization was applied to a computational model of recurrent seizures and DBS to find the optimal frequency, pulse width, and amplitude for seizure suppression. The optimization was successful at finding combinations of parameters that left the system with little to no bursting activity. The next step for DBS to treat seizures is to optimize stimulation parameters for individual patients. Bayesian Optimization could be implemented during clinic visits to help suggest stimulation parameters. It could also utilize the information from patient seizure frequency records to model the stimulation effect and make new suggestions on subsequent visits.

Presenter: Salma Hassam
Poster Number: 32
Home Institution: Minneapolis Community and Technical College
Program: LSSURP
Faculty Mentor: Dr. David Bernlohr
Poster Title: **FABP4 And FABP5 Secretion By 3T3-L1 Adipocytes.**

Abstract: Fatty Acid Binding Proteins (FABPs) are small cytoplasmic proteins that modulate metabolism and lipid flux. They belong to a multigene family of lipid carriers that bind long chain fatty acids. FABP4 is highly expressed in adipocytes and facilitates lipolysis of free fatty acids during starvation. Recent studies show that FABP4 is also secreted by an unconventional mechanism from adipocytes in response to lipolysis and hypoxia. Secreted FABP4 has been suggested to increase insulin secretion from pancreatic β-cells and contribute to the development of Type 2 diabetes. To evaluate FABP4 secretion, and to parallel that analysis with a second FABP expressed in adipocytes, FABP5, monolayers of differentiated 3T3-L1 adipocytes were treated with forskolin to induce lipolysis and the secretion products assessed immunochemically. Using SDS-PAGE followed by immunoblotting, both FABP4 and FABP5 secretion was increased 5-fold in response to forskolin. During a 4-hour lipolysis experiment of FABP4 and FABP5, intracellular protein levels declined 2-fold. These results suggest that the secretory mechanism does not discriminate between FABP4 or FABP5 and that similar amounts of both proteins are actively released from adipocytes. As with FABP4, these results suggest that FABP5 may also contribute to the development of Type 2 diabetes.

Presenter: Chandler Hellenbrand
Poster Number: 33
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. Michael Freeman
Research Advisor: Marissa Quijano
Poster Title: **A New Method to Observe Post-Translational Modifications of Natural Products in Native Hosts**

Abstract: Ribosomally encoded peptides comprise an important class of natural products due to the multitude of biological functions they can perform, and the biosynthesis of these peptides can be easily manipulated for pharmaceutical or industrial purposes. Thus, it is of great interest to understand the various pathways that produce ribosomally encoded, post-translationally modified natural products (RiPPs). A new RiPP family, termed borosins, have been recently identified and are characterized by unique autocatalytic N-methylations of the peptide backbone. Unfortunately, it is difficult to induce high-level expression of these peptides in their native fungal hosts, often hindering the study and discovery of the final natural products. Thus, we are developing a system to tag and insert the unmodified precursor peptides of RiPPs directly into their native hosts to observe the modifications that occur after translation. This project aims to engineer a three-plasmid system to heterologously express and tag the precursor peptides into an amber codon-less strain of *E. coli* with a noncanonical amino acid. The first plasmid contains the protein of interest, mutated to contain a single amber codon. The second plasmid expresses an RNA polymerase to constitutively transcribe the peptide, and the final plasmid contains an engineered tRNA molecule that recognizes the amber codon and inserts the noncanonical amino acid during translation. Once purified, the precursor peptides can be inserted back into their native hosts, and the amino acid tag will allow for the observation of the final natural product. This technology provides a method to study final metabolites that originate from uncultivated microbial sources, thus expanding natural products research to unique proteins that would otherwise go undiscovered.

Presenter: Ashley Hiebing
Poster Number: 34
Home Institution: Milwaukee School of Engineering
Program: LSSURP
Faculty Mentor: Dr. Daniel Schmidt
Research Advisor: David Nedrud, Dr. Theoden Netoff
Poster Title: **Simulating Cultured Neurons to Understand and Predict Network Characteristics in the Context of Ion Channel Perturbances and Mutations**

Abstract: Ion channels mediate brain function, including signal processing, learning, and behavior. While much is known about ion channel properties individually, how ion channels give rise to emergent and aggregate properties of both neurons and networks remains hard to study. In order to address this need and understand the role of ion channels in neural computations, our lab has developed a genetically encoded tool, called lumitoxin, which is able to perturb individual ion channel types. To assist with the further engineering of lumitoxins, we have developed a simulated mesoscale neural network, which simulates lumitoxin's effects on neuronal and network activity. Through systematic variation of model parameters, we could determine what ion channel perturbations might cause changes in both excitability of individual neurons as well as other network properties. These results will assist in identifying neurons with improved characteristics when screening through libraries of lumitoxin variants expressed in primary hippocampal neuron cultures. Furthermore, our findings have implications for determining what aspects of intrinsic neuron excitability affect neuronal ensembles involved in learning and memory.

Presenter: AkpevweOghene Ikoba
Poster Number: 35
Home Institution: University of Iowa
Program: LSSURP
Faculty Mentor: Dr. Laura Shannon
Research Advisor: Cari Schmitz Carley, Husain Agha
Poster Title: **Quantifying Potato Dihaploid Induction from Pollinations with IVP101**

Abstract: Potato is the third largest crop produced worldwide, but suffer from many pressures, such as disease, that have been minimized in other important crops like maize and soy. Unlike maize and soy, potatoes are tetraploid, making the robust breeding techniques developed for other major cropping systems inapplicable to a potato breeding system. This has made it challenging, if not impossible, to breed for and fix specific traits. By creating a pool of diploid germplasm, breeders would be able to breed for characteristics, such as increased disease resistance, because of the multitude of breeding tools readily available for diploids. Our present work aims to create diploid potatoes by prickle pollinating tetraploid potatoes (Solanum tuberosum L) with IVP101 (S. phureja), a known dihaploid inducer. We pollinated 6 different potential tetraploid parents to look for which tetraploids will make diploids. The number of fruits that were produced from pollinations were counted and seeds were examined to determine the frequency of dihaploid induction. Expected results should reveal that a small number of fruit and diploid potato seed was created. IVP101 crosses with potato will then result in some seed with a small spot of anthocyanin, which will be indicative that our crosses were successful. Diploid potato seed and fruit is more easily produced by certain varieties, indicating that dihaploid induction might be influenced by variety. Future research attempts should be made to investigate the relationship between variety and dihaploid induction, and the genetics underlying this relationship.

Presenter: Allison Johnson
Poster Number: 36
Home Institution: University of Michigan - Ann Arbor
Program: LSSURP
Faculty Mentor: Dr. Michael Georgieff
Poster Title: **Effects of Acute Iron Deficiency (ID) on Expression and Epigenetic Modification of Brain-Derived Neurotrophic Factor (Bdnf) in Neuronal Cell Lines**

Abstract: Micronutrient deficiencies during early life are known to cause impairments in cognitive, social and emotional behavior later in life. Iron deficiency, the most prevalent of these, affects almost two billion people globally, most notably young children and expecting mothers. Recent research has shown irreversible changes of certain gene levels and epigenetic modifications despite intervention. To study this, acute ID was induced in mouse hippocampal (HT-22) and neuroblastoma (N2a) cell lines using an iron chelator, Deferoxamine (DFO). Levels of transferrin receptor protein 1 (Tfrc), which codes for a protein that imports iron, and Bdnf, an important protein in neuronal survival, growth, and differentiation, were quantified by RT-qPCR. Acute ID (24 hours after DFO treatment) upregulated transferrin, indicating cellular ID. Consistent with in vivo finding, ID downregulates Bdnf expression, showcasing specific effects of ID in neuronal-derived cell lines. This study provides a basis for further investigation into the iron-dependent epigenetic mechanisms underlying the long-term gene dysregulation induced by early-life ID.

Presenter: Sakeli Kennedy
Poster Number: 37
Home Institution: Florida International University
Program: LSSURP
Faculty Mentor: Dr. Marija Cvetanovic
Research Advisor: Lisa Duvick, Harry Orr, Ling Li
Poster Title: **Is There a Link Between Polyglutamine (CAG) Length in ATXN1 and Alzheimer'a Disease?**
Abstract:

Abnormal expansion of CAG repeats in Ataxin-1 (ATXN1) gene causes Spinocerebellar Ataxia type 1 (SCA1), a neurodegenerative disease characterized by loss of motor control, cognitive and mood impairments. In SCA1, 40 or more uninterrupted CAG repeats is thought to lead to toxic gain of ATXN1 function. However, whether the length of the polyglutamine tract alters ATXN1 function to increase risk for other diseases is unknown. GWAS identified ATXN1 as a risk factor for Alzheimer's Disease (AD). To discern a relationship between the number of CAG repeats in ATXN1 and AD, we sequenced CAG regions of two cohorts. Cohort 1 contained unrelated individuals classified in three groups: Normal (N), Mild Cognitive Impairments (MC) and AD, while Cohort 2 consisted of AD patients and unaffected siblings. We used PCR to amplify CAG regions of ATXN1, gel electrophoresis to separate alleles with variable polyglutamine tracts, then sequenced them using UMN genomic facility. No significant relationship between CAG length was found in Cohort 1 possibly due to a small sample size (n=3). In Cohort 2, we found increased numbers of CAG repeats in AD patients compared to healthy relatives. This result indicates that inheriting ATXN1 with longer polyglutamine tracts may increase risks for AD.

Presenter: Clayton Kettlewell
Poster Number: 38
Home Institution: University of Missouri
Program: LSSURP
Faculty Mentor: Dr. Matthew Johnson
Research Advisor: Julia Slopsema, Lauren R. Madden, David Darrow, Theoden I. Netoff
Poster Title: **Computational Model-Based Analysis of Spinal Cord Stimulation Paradigms for Regaining Lower-Body Motor Control**
Abstract:

BACKGROUND: Epidural thoracic-level spinal cord stimulation (SCS) has been shown to activate lower body muscle groups in individuals with paraplegia (Grahn, et al., Mayo Clinic Proceedings, 2017). While it is possible to activate different muscle groups by adjusting electrode configurations, identifying these stimulation settings is a tedious process that can be challenging to perform during clinical visits. The purpose of this project was to build a patient-specific computational model of SCS and investigate in the models an orientation-selective stimulation paradigm to enhance the selectivity of axonal tract activation within and adjacent to the spinal cord. METHODS: A computational finite element model of SCS was developed in COMSOL using segmented geometries of the lower thoracic spinal cord, subdural and epidural spaces, and vertebrae from patient-specific magnetic resonance imaging. Simulations were conducted for monopolar stimulation through individual electrodes and the superposition principle was used to calculate the resulting electric field for bipolar and multipolar stimulation configurations. These and second spatial derivative for various electrode configurations tested in the clinic. RESULTS: This study resulted in the development of a graphical user interface for visualizing model-based predictions of how SCS electrode configurations affect spinal cord pathways. Retrospective comparisons between EMG recordings and electrode configurations in patients supported the efficacy of several configurations to provide selective activation of muscle groups to aid in ambulatory motion. Additionally, by averaging effective electric fields to find significant areas of focus, several unique electrode configurations were identified. Prospectively, the graphical user interface will provide clinicians with a more efficient means to steer the electric field to align with target axons and provide further control of muscle groups.

Presenter: Anna Khoroshilov
Poster Number: 39
Home Institution: Massachussetts Institute of Technology
Program: LSSURP
Faculty Mentor: Dr. Zohar Sachs
Poster Title: **Repurposing Mebendazole: c-Myb Degradation and its Pathways in Acute Myelogenous Leukemia**

Abstract: Acute myeloid leukemia (AML), with a five-year survival rate of just over 27 %, contributes to over ten thousand deaths a year in the United States. Particularly concerning is AML relapse brought on by leukemia stem cells (LSC) capable of self-renewal. In 2011, Zuber et al. (Genes & Dev) found that self-renewing MLL-AF9 mutant leukemia cells show a c-Myb-dependent gene expression pattern. Furthermore, Myb knockdown induced complete remission in vivo murine models. Sachs et al. (Blood 2015) then showed that NRASG12V is required for self-renewal in MLL-AF9 mutant AML, and, consistently with Zuber et al., that the NRAS expressing cells showed a similar Myb-positive gene expression pattern. So, we hypothesize that Myb inhibition can be effective in targeting NRAS-mediated self-renewal. Recently, Walf-Vorderwülbecke et al. (Leukemia 2018) found Mebendazole (MBZ), a drug initially developed and approved as an anthelmintic, to target c-Myb for degradation by affecting the chaperone activity of heat shock protein 70 (HSP70) required for c-Myb folding. To study the effects of this inhibitor in mouse and human AML we (1) determine an IC50 concentration for both mouse (leukemia bank) and human (THP1) MLL-AF9 mutant cell lines; (2) use high parameter single cell protein profiling (mass cytometry, CyTOF) to probe changes in RAS associated signaling pathways; (3) look for cell cycle arrest (MBZ disrupts microtubules) and check apoptotic markers. Results demonstrate that treatment with MBZ lowers levels of many oncogenes, which suggests that it could be effective in treating AML.

Presenter: Kevin-Phu Le
Poster Number: 40
Home Institution: The University of Texas - Austin
Program: LSSURP
Faculty Mentor: Dr. Bryce Binstadt
Research Advisor: Nathan J. Schuldt
Poster Title: **Engineering a Mouse Model to Assess Dual TCR T Cells and Their Implications in Autoimmunity**

Abstract: T cells play a crucial role in the immune system. The T cell receptor (TCR), most commonly formed from the pairing of an α and β chain, recognizes antigen displayed by the major histocompatibility complex (MHC) and helps orchestrate the adaptive immune response. During maturation, the robust diversity of the TCR repertoire is achieved through recombination of many variable and joining regions to a constant region. Most T cells express a single TCRα chain recombined from one allele. However, about 10% of human and murine T cells recombined TCRα chains from both alleles. Many have hypothesized that dual TCR expression contributes to several immune pathologies. However, due to the lack of available reagents to detect dual TCR T cells, their role in immunity remains unclear. To overcome this hurdle, we have developed an in vivo model that allows detection of all dual TCRα T cells. We attached a unique epitope tag to the TCRα constant region of each allele, allowing us to determine which allele a TCRα chain is recombined from as well as detect cells that express TCRα chains from both alleles. Currently, we are undertaking strategies to optimize tag expression and detection.

Presenter: Christopher LeWarne
Poster Number: 41
Home Institution: Creighton University
Program: LSSURP
Faculty Mentor: Dr. David Potter
Research Advisor: Dr. Zhijun Guo
Poster Title: **Profiling Differential Expression of Cytochrome P450 Enzymes in Mouse Splenocytes and Breast Cancer Cell Lines**

Abstract: Regulatory T cells (Tregs) suppress conventional effector T cells in immune responses. In tumor microenvironments, however, it is known that infiltrating Tregs share some physiological traits with cancer cells. Infiltrating Tregs suppress T cell killing of tumor cells, allowing for tumor progression. In light of this, many immunotherapy studies of cancer have been focused on depleting Treg levels in the tumor microenvironment. Recently, N1-hexyl-N5-benzyl-biguanide (HBB) has been proven an effective inhibitor of various cytochrome P450 (CYP) enzymes in breast cancer cell lines. Inhibition of CYP enzymes in cancer cell lines results in tumor suppression and lowered oxidative phosphorylation (OXPHOS). Preliminary data shows that HBB-treatment also results in suppression of Treg populations. This study aims to survey extant CYP enzymes in Treg-containing splenocyte populations and purified Treg cell populations in mouse models in order to identify potential targets for HBB-treatment. If there is differential expression of CYP enzyme expression between Tregs compared to effector T cells, then it may be possible that these differences can be exploited by HBB-treatment. Expression of members of the CYP2C, 2J, 3A, and 2S families has been investigated using PCR techniques. Further studies will be conducted to continue searching enzyme expression differences that may be exploited for therapeutic strategies.

Presenter: Nile Liu
Poster Number: 42
Home Institution: Johns Hopkins University
Program: LSSURP
Faculty Mentor: Dr. Subree Subramanian
Research Advisor: Xianda Zhao, Lisa Basso, Beminet Kassaye
Poster Title: **Hypoxia Modulates miR-424 Expression in Colorectal Cancer and Increases miR-424 Delivery through Exosomes**

Abstract: Colorectal cancer is one of the deadliest cancers in the USA with upwards of 50,000 deaths per year. microRNAs are short noncoding RNAs that regulate gene expression. It has been shown that in colorectal tumors, miR-424 is implicated in the escape of antitumor immunity. However, transcriptional control of miR-424 in colorectal cancer and the delivery of miR-424 are not known. Utilizing qPCR and nanotracking, the relative miR-424 expression in cells and exosomes were quantified as well as exosome production. In this study we demonstrate that hypoxia positively induces miR-424 expression in colorectal tumor cell lines, exosome production, and miR-424 expression in exosomes. We observed that that the inner parts of the tumor exhibit higher miR-424 expression compared to the outer parts of the same tumor, and miR-424 expression is upregulated in tumor cells compared to normal cells. We found in colorectal cancer cell lines that miR-424 expression positively correlated with hypoxia. Our data additionally showed hypoxic conditions increased exosome production, and exosomes derived from hypoxia conditions exhibited higher miR-424 expression compared to normoxic conditions. This study implicates hypoxia in increasing miR-424 expression in cells in addition to increasing miR-424 delivery in exosomes, suggesting the role of oxygen levels in antitumor immunity in colorectal cancer. Further studies may investigate the mechanism in which hypoxia upregulates miR-424 expression.

Presenter: Koby Ljunggren
Poster Number: 43
Home Institution: Missouri State University
Program: LSSURP
Faculty Mentor: Dr. Douglas Yee
Research Advisor: Kelly LaPara
Poster Title: **Nonimmunoglobulin Protein Scaffold Purification for Insulin Receptor Targeting in Endocrine-Resistant Breast Cancer**

Abstract: Treatment of endocrine-resistant breast cancer continues to be a pervasive clinical problem. More effective treatments may improve prognoses. Recent research indicates that insulin receptor (InsR) isoform A is highly expressed over isoform B in endocrine-resistant breast cancer cells. Through InsR-A-specific targeting, more effective treatments may be developed. A small nonimmunoglobulin protein scaffold for InsR isoform-specific binding was engineered using a T7 phage gene 2 protein (Gp2). Through methods of directed evolution and yeast-surface display, three variants (Gp2-1, 5, 10) were identified with low nanomolar affinity. All variants inhibited insulin-mediated proliferation in endocrine-resistant cell lines without down-regulation of insulin expression. Gp2-5 demonstrated higher affinity binding to InsR-B compared to InsR-A. While InsR-B is not the preferred target, Gp2-5 may be used to either further investigate isoform-specific binding or undergo directed evolution to achieve greater specificity. Significant quantities of Gp2-5 need to be produced optimally for further experimentation and analysis. Induction times and column purification methods were explored for high yields of protein, followed by a final purification step using FPLC. Data suggests that Gp2-5 may aggregate in solution. However, Gp2-5 is a promising InsR-binding peptide with implications for clinical therapy.

Presenter: Caitland Love
Poster Number: 44
Home Institution: Georgetown University
Program: LSSURP
Faculty Mentor: Dr. Lihsia Chen
Poster Title: **Hydrocephalus and the Adverse Role of L1CAM Inhibition in Fluid Permeability**

Abstract: Congenital hydrocephalus affects one in every 500 children, and is defined by an accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain, in which the normal system for CSF drainage and absorption does not function properly. Although it is as common as Down's Syndrome, this serious condition has to be treated surgically, as there are little therapy options for affected individuals. The most common cause for this disorder involves loss of L1 cell adhesion molecules (L1CAM) function, however genetic modifiers are likely involved due to the variability of symptoms and conditions in L1 patients. Identifying these genetic modifiers is difficult in humans or mammals, but because these transmembrane proteins are conserved in model organisms, we can turn to *C. elegans* as a more accessible genetic tool. A genetic interaction between the *C. elegans* L1 orthologue, sax-7, and the Ras gene, let-60, has been found to result in apparent fluid accumulation within the worm. This study evaluates whether that fluid accumulation is due to osmoregulatory defects in the worm, and how that offers insight into molecular interactions which could cause or eventually treat congenital hydrocephalus.

Presenter: Allen Lynch
Poster Number: 45
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. Nicholas Levinson
Poster Title: **A FRET-Based Biosensor for Measuring Conformational Equilibria in Cdk2**
Abstract: Cyclin dependent kinase 2 (Cdk2), is a regulatory checkpoint of the eukaryotic cell cycle. Cdk2 requires phosphorylation and allosteric binding of protein Cyclin A or Cyclin E for activation, and the subsequent phosphorylation of downstream targets by Cdk2 is proposed to transition the cell from G1 to S phase of the cell cycle. Hyperactive Cdk2 can lead to aberrant cell division and is thus implicated in aggressive forms of breast and ovarian cancer. Because Cdk2 knockouts in mice have demonstrated viability, this enzyme is an attractive target for selective inhibitory therapies. However, the equilibria and dynamics of the conformational states of Cdk2 are poorly understood. To elucidate the conformational equilibria in Cdk2 in different biochemical states, we studied the movement of the dynamic activation loop of the kinase in response to cyclin binding and phosphorylation using time-resolved Forster resonant energy transfer (TR-FRET). We excited covalently-attached fluorophores on Cdk2 and fit the resulting lifetime decays to Gaussian distributions for the distance between the probes. This powerful assay allows us to differentiate multiple conformational populations with angstrom-level precision and can be used in a high-throughput screen for small molecule response in Cdk2.

Presenter: Allyson Marrs
Poster Number: 46
Home Institution: University of Notre Dame
Program: LSSURP
Faculty Mentor: Dr. Craig Henke
Poster Title: **Hypoxia Drives Fibrogenesis in IPF Mesenchymal Progenitor Cells**
Abstract: Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease that affects ~100,000 people in the United States. The average life expectancy after diagnosis is 3-5 years and there are currently no treatments that arrest fibrotic progression. A distinctive feature of IPF is the spread of fibrosis from scarred alveoli to healthy alveoli, resulting in hypoxia. Our lab has discovered fibrogenic mesenchymal progenitor cells (MPCs) in the human lung. These cells exist in a hypoxic niche and are extremely prolific, although the mechanism behind their fibrogenicity is not known. Through cell sorting, MPCs can be segregated into two subgroups: CD44HI and CD44LO expressing cells. The CD44HI cells display more self-renewal in hypoxic conditions. We hypothesize that hypoxia is a driver of IPF MPC proliferation. The proposed mechanism involves upregulation of Myc and hTERT and activation of telomerase. To test this mechanism, CD44HI cells were cultured under normoxic or hypoxic conditions. Using Western blot analysis and a telomerase activity assay, we found that hypoxia upregulates Myc and hTERT and increases telomerase activity in CD44HI MPCs. These results align with our proposed mechanism and allow us to continue exploring this pathway. The data also support the claim that CD44HI MPCs are involved in IPF fibrotic progression.

Presenter: Elisa Martinez
Poster Number: 47
Home Institution: Dickinson College
Program: LSSURP
Faculty Mentor: Dr. Rocio Gomez-Pastor
Poster Title: **The Significance of P53 And HSF1 in Regulating Mitochondrial Membrane Potential in Huntington's Disease**

Abstract: Huntington's Disease (HD) has been associated with increased levels of P53 tumor suppressant as well as decreased levels of HSF1, two major transcription factors responsible for controlling mitochondrial function and cellular survival. Increased levels of P53 has been linked to a decrease in mitochondrial genes, PGC1 and CYCS. It has been seen that mitochondrial defects, caused by low levels of mitochondrial genes, leads to striatal cell degradation and HD pathology. However, it is unknown how p53 mediates mitochondrial dysfunction in HD. Our hypothesis is that this defect is mediated through the interaction between p53 and HSF1. To test this hypothesis, we performed treatments in which immortalized WT or HD derived mouse striatal cells were exposed to silencing HSF1 and/or p53 pharmacological inhibition using pifithrin-a. qRT-PCR was used to measure mitochondrial genes levels and the fluorescent JC-1 dye was used to qualitatively analyze the condition of the mitochondrial membrane potential using fluorescence microscopy. Lastly, an Alamar Blue assay based on the spectrophotometric detection of Resazurin reduction to Resorufin was used to analyze cell viability. Our results showed that, in WT cells there was a synergistic effect in which both silencing HSF1 and P53 inhibition had twice as much increase in mitochondrial genes than when only one treatment was done, indicating that the two genes operate in the same pathway. Interestingly, this effect was not observed in HD cells where the benefits exerted by pifithrin-a were lost when silencing HSF1. These results suggest that in HD, HSF1 is necessary to maintain mitochondrial function. Further research will be necessary to dissect the differential roles of p53 and HSF1 in maintaining mitochondrial function between WT and HD cells.

Presenter: Jessyca Martinez Velez
Poster Number: 48
Home Institution: University of Puerto Rico - Utuado
Program: LSSURP
Faculty Mentor: Dr. Cindy Tong
Poster Title: **"Summer Production of Lettuce Under Shade"**

Abstract: There is a demand in Minnesota for locally-grown lettuce. It is difficult to grow lettuce in the summer because of high temperatures. Our team used a 50% shade treatment, which has previously been showed to can lower the temperature of crops. We hypothesized that shade will increase yield and quality of lettuce compare to controls grown in the open field without shade. Our results proved that the shade was helpful to keep and maintain lower temperatures. We tested for possible geographical variation by growing lettuce at two locations. Lettuce fresh weights differed between the two locations and among treatments.

Presenter: Holly McKee
Poster Number: 49
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. Rita Perlingeiro
Research Advisor: Sridhar Selvaraj, Ricardo Mondragon-Gonzolas, Fabrizio Rinaldi, Joy Aho
Poster Title: **Compound Library Analysis Identifies a Combination of Small Molecules that Increase the Terminal Differentiation of Human Induced Pluripotent Stem Cells into Myotubes and their Maturation.**

Abstract: Primary skeletal myoblasts stand as the current gold standard for in vitro modeling of skeletal muscle diseases. Isolation of patient-specific myoblasts requires a muscle biopsy, which is an invasive and uncomfortable procedure for patients. Patient-specific pluripotent stem cell skeletal muscle derivatives represent an attractive potential alternative for in vitro disease modeling without the requirement of muscle biopsy. The technology of reprogramming somatic cells into induced pluripotent stem (iPS) cells offers tremendous potential for the generation of large amounts of lineage-committed cells for disease modeling, as well as for other applications, including drug screening. Nevertheless, the overall embryonic nature of iPS cell-derivatives (across lineages) stands as a barrier for reliable disease modeling studies. Several signaling pathways, such as the TGF-β, have been reported to affect myoblast fusion and their differentiation into myotubes. Thus, screening for the effect of different signaling pathways and epigenetic modifiers is a promising approach to further promote the differentiation of pluripotent stem cells into myotubes, and their subsequent maturation. Following an initial screening using a small molecule compound library, six compounds were selected based on initial improvement of differentiation efficiency of iPS cell-derived myogenic progenitors into myotubes, as identified through the quantification of all myosin heavy chain (MHC) isoforms by immunofluorescent staining. Of note, our results revealed that the combined exposure of the selected compounds during differentiation promotes a significant increase in fusion index (2-fold). More importantly, there was a 100-fold increase in the expression of the neonatal isoform of MHC(MHC-neo) in compound-treated myotubes in comparison to untreated controls, indicating this compound combination induced maturation of pluripotent stem cell-derived myotubes.

Presenter: Sophia Pantano
Poster Number: 50
Home Institution: University of Notre Dame
Program: LSSURP
Faculty Mentor: Dr. Kalpna Gupta
Poster Title: **a1-Antitrypsin (Prolastin-C) Reduces Pain in a Sickle Cell Mouse Model by Decreasing Neutrophil Elastase Activity**

Abstract: Sickle cell disease (SCD) is an inherited recessive disorder with a point mutation in hemoglobin leading to sickle shaped red blood cells (RBC) under low oxygen. The vasoocclusion resulting from the accumulation of these sickle RBCs in the blood vessels leads to acute pain which may be superimposed on chronic pain and inflammation in SCD. SCD is associated with increased neutrophil elastase activity. a1- Antitrypsin is a serine proteinase inhibitor, or Serpin, that has an inhibitory effect on elastase. Our laboratory found that a1- Antitrypsin (A1AT) reduces pain in sickle mice, and we hypothesize that the underlying mechanism involves decreasing neutrophil elastase activity. We treated sickle (HbSS-BERK) and non-sickle (HbAA-BERK) mice with 80 mg/kg A1AT (Prolastin-C, Grifols Therapeutics Inc. NC, USA) an FDA approved drug, through intraperitoneal injection daily for 3 days and examined the neutrophil elastase activity and central nociceptive mechanisms. We found elastase was upregulated in vehicle-treated HbSS mice compared to HbAA in plasma ($p < 0.002$), and lung ($p < 0.05$). Prolastin-C treatment of HbSS significantly reduced elastase activity in the DRG ($p < 0.03$), plasma ($p < 0.002$), and lung ($p < 0.001$). Our results demonstrate a novel role for A1AT as a therapeutic target for treating pain in SCD.

Presenter: Jesenia Perez
Poster Number: 51
Home Institution: Florida International University
Program: LSSURP
Faculty Mentor: Dr. Timothy Starr
Research Advisor: Christopher Clark
Poster Title: **Assessing the Role of WAC as a Tumor Suppressor in Colorectal Cancer**

Abstract: WAC is a scaffold protein that forms a complex with RNA polymerase and functions in elongation during transcription. Studies have supported WAC's role as a functional partner of RNA Pol II during transcription and WAC is also necessary for the monoubiquitination of histone H2B. Using an insertional mutagenesis screen for candidate cancer genes (CCG), we found that the WAC gene was a CCG for colorectal cancer. Despite this finding, the relationship between WAC and tumorigenesis is not fully known. In this study, the role of WAC in colorectal cancer as a tumor suppressor was supported by the results of our soft agar colony formation assay which assesses the ability for anchorage-independent growth. We conducted this assay using two colorectal cancer cell lines: low expression of WAC HCT116, and high expression of WAC HT29. The assay revealed that in HCT116 virally modified to overexpress WAC, there was a significant decrease in the number of colonies compared to EGFP control. In addition, WAC overexpressed HCT116 had larger sized colonies which may imply a relationship between overexpression of WAC and growth-associated genes. HT29 cells modified via CRISPR/Cas9 to knock out WAC, exhibited more colonies than HT29 parental colonies. These results indicate that WAC may play a role in tumor suppression by regulating genes involved in anchorage-independent growth. Future studies will test for other cancer characteristics in WAC deficient and WAC overexpressed cells and further elucidate WAC's role as a tumor suppressor.

Presenter: Taylor Phillips-Jones
Poster Number: 52
Home Institution: Howard University
Program: LSSURP
Faculty Mentor: Dr. Lucy Vulchanova-Hart
Research Advisor: Dr. Alex Skorput
Poster Title: **The Role of Innate Immune Cells within the Subarachnoid Space in the Development of Chronic Neuropathic Pain**

Abstract: Chronic neuropathic pain is very prominent within our society and characterized by somatosensory hypersensitivity. Previous research suggests that immune cells contribute to the development of chronic pain through neuroimmune interactions both within the central nervous system (CNS) and peripherally. The subarachnoid space serves as both a physical and immunologic barrier between the CNS and the periphery. The function of innate immune cells within the subarachnoid compartment is understudied and their function in neuropathic pain is unknown. We utilized transgenic mice (Iba1-GFP), which express GFP in innate immune cells (microglia, macrophages, dendritic cells, monocytes) to visualize Iba1-expressing cells within the subarachnoid space. To better understand neuroimmune interactions in chronic pain, mice were sacrificed at different time points (3, 7, 46 days) following surgical injury of the sciatic nerve. Immunofluorescence was performed on whole mount lumbar spinal cord preparations for Iba1 and CD68 with DAPI counter staining to quantify the activation state of Iba1-expressing cells following nerve injury. Preliminary data showed an increased percentage of blood vessel coverage by immune cells in nerve-injured mice compared to sham controls, suggesting that immune cells in the subarachnoid space change their interaction with the blood vessels. This may affect subarachnoid space barrier function contributing to neuroinflammation and the chronic pain state. An increased number of Iba1-expressing cells seemed to exhibit a hypertrophied morphology 3 days after nerve injury, such hypertrophy may be an indicator of cellular activation. This highlights the potential importance of innate immune responses in the subarachnoid space following peripheral nerve damage.

Presenter: Sonia Rivera
Poster Number: 53
Home Institution: University of Minnesota - Rochester
Program: LSSURP
Faculty Mentor: Dr. Cheryl Olman
Poster Title: **Visual Population Receptive Field Estimates and Auditory and Motor Mapping in People with Schizophrenia**

Abstract: The population receptive field (pRF) model is an estimate of both the preferred RF center of a population of neurons in a given voxel and the RF size. AFNI generates a pRF model by receiving input parameters from computed Gaussian curves and stimulus time series inputs. The question was to determine the differences in visual pRFs and auditory and motor maps between participants with schizophrenia (SZ), schizophrenic patients' relatives, and healthy controls. We analyzed fMRI data that measured the blood-oxygen-level dependent signal in the visual, auditory, and motor cortices while participants responded to drifting bar apertures, tone sweeps, and motor cues. We found that patients with SZ have significantly smaller visual pRFs when compared to their relatives and healthy controls. By mapping the auditory and motor responses, it was determined that there were no significant differences between groups. We concluded that the differences in visual pRFs between groups may explain the range of visual deficits observed in patients with SZ. Additionally, the motor and auditory maps may exhibit no differences due to the limits of the analyses. The ever growing applications of pRFs are likely to provide insight beyond conventional visual field mapping techniques--both to SZ and other brain disorders.

Presenter: Zachary Sanger
Poster Number: 54
Home Institution: North Dakota State University
Program: LSSURP
Faculty Mentor: Dr. Theoden Netoff
Poster Title: **Quantification of Limb Rigidity in a Parkinsonian Non-Human Primate Model using a Wireless Sensing Glove**

Abstract: Parkinson's disease is a neurodegenerative disease that is expected to affect more than 9 million people by 2030 [1]. Cardinal symptoms include tremor, bradykinesia, and rigidity with the severity of each symptom assessed by an examining neurologist through the Unified Parkinson's Disease Rating Scale (UPDRS). Each question on the UPDRS is assigned a qualitative integer value, between one and four, by the examiner. Differing qualitative scores for the same patient can occur caused by different techniques applied during the examination. Previous attempts to quantify the cardinal symptoms have focused on limb rigidity, but the techniques require complicated machinery [2]. Developing a wireless instrumented glove to measure limb rigidity can create a reliable quantitative metric of rigidity that is easy to implement in practice. Arm and leg extension and flexion are performed on a non-human primate for one minute. Force measurements and position tracking of the examiner's arm is collected through an instrumented glove containing force sensitive resistors and an inertial measurement unit equipped with an accelerometer, gyroscope, and magnetometer. The root mean squared value of the force measurement is weighted by the position of the examiner's arm for each cycle of extension and flexion to obtain a quantified rigidity measurement over the examination period.

Presenter: Adam Smiley
Poster Number: 55
Home Institution: North Hennepin Community College
Program: LSSURP
Faculty Mentor: Dr. Carrie Wilmot
Poster Title: **Expression, Purification, Crystallization, and X-Ray Crystallographic Analysis of a β-Lactone Synthetase**

Abstract: OleC is one of three proteins in a complex that produces long-chain olefinic hydrocarbons, this complex is found in thousands of microbial genomes. When in the presence of MgATP the enzyme OleC produces β-lactone, a class of molecule with biomedical applications. This β-lactone synthetase, along with sixty other confirmed β-lactone synthetase enzymes, comprises a large new family with novel chemistry within the ANL superfamily. In order to develop structural knowledge of the β-lactone synthetase we needed to express, purify, and crystallize the protein for X-Ray crystallographic studies. A variety of expression optimizations, additives, crystallization conditions, and organisms were attempted. OleC is extremely prone to aggregation, this makes crystallization difficult and was our primary obstacle. The addition of the zwitterionic detergent CHAPS was found to be extremely effective in preventing aggregation of the protein. We have since successfully completed a Selenium labeled prep, purified using high concentrations of salt and 0.1% CHAPS and successfully crystallized the resulting protein in a wide screening. We are still attempting to find conditions that will produce diffraction quality crystals.

Presenter: Margarita Soltero Gutierrez
Poster Number: 56
Home Institution: University of Denver
Program: LSSURP
Faculty Mentor: Dr. Donald Simone
Poster Title: **Co-Localization of Mu Opioid Receptor (MOR) and Metabotropic Glutamate Receptor-5 (mGluR5) on Somatosensory Neurons**

Abstract: Opioids are associated with many side effects, including respiratory depression and death. Approximately 115 people die from opioid overdose every day; thus, it is important to develop new drugs to treat chronic pain. MMG22 is a bivalent ligand that targets a putative MOR/mGluR5 heteromer. MMG22 produces potent antinociception without tolerance or respiratory depression, and possibly addiction. We determine the expression and co-localization of MOR and mGluR5 in dorsal root ganglion (DRG) neurons 20 days after nerve injury in mice using RNAscope and unbiased stereology. We hypothesize that MMG22 decreases pain in various models of persistent pain, but not in naïve animals, due to the increase in mGluR5 expression after injury. In addition, if MMG22 works by binding to a MOR/mGluR5 heteromer, then neurons must express both MOR and mGluR5. Preliminary findings indicate that MOR and mGluR5 are present in the same neuron about 55% of the time in the ipsilateral side. Moreover, there is a large number of cells with mGluR5 present throughout the 20 days ipsilateral to the nerve injury and an upregulation of MOR in larger diameter cells. These studies will provide important information about the expression of MOR and mGluR5 following nerve injury and about the mechanisms underlying the analgesic effects of MMG22.

Presenter: Cameron Swenson
Poster Number: 57
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. Christine Salomon
Research Advisor: Daniel Wilson
Poster Title: **Tools for Developing a Microbial Drone to Treat *Pseudomonas* Biofilms**
Abstract:

The bacterial pathogen *Pseudomonas aeruginosa* frequently infects immune-deficient persons, burn wounds, and the lungs of cystic fibrosis patients in hospital settings. Biofilm phenotypes are characterized by *P. aeruginosa* cells surrounded by an extracellular polymeric matrix and are particularly difficult to treat due to physical protection by the matrix and inherent resistance to many antibiotics. Consequently, new treatment methods are needed. *Lactobacillus plantarum* is a probiotic organism known to produce enzymes, antibiotics (bacteriosins), immune system modulators, and other metabolites that inhibit growth of other bacteria and improve wound healing. Bacteriotherapy using an engineered *L. plantarum* strain containing an anti-alginate antibody fragment fused to a bacterial surface protein will enable specific binding to alginate in *P. aeruginosa* biofilms and will disrupt and inhibit biofilm growth. To validate this system, the binding strength of the engineered *L. plantarum* and of the antibody fusion protein itself must be evaluated. Towards this end, I modified alginate by coupling tetramethylrhodamine (TMR) cadaverine and biotin hydrazide via water-soluble carbodiimide chemistry. Biotin-streptavidin affinity enabled immobilization of the modified alginate on streptavidin-coated plates and beads while TMR enabled fluorescent quantification of bound alginate. Additionally, I cultured and characterized biofilms of two different *P. aeruginosa* strains to test growth conditions for future binding experiments of engineered *L. plantarum* to *P. aeruginosa* biofilms.

Presenter: Mario-Cyriac Tcheukado
Poster Number: 58
Home Institution: University of Notre Dame
Program: LSSURP
Faculty Mentor: Dr. Alfonso Araque
Research Advisor: Michelle Corkrum
Poster Title: **Astrocyte Plasticity in the Context of Amphetamine Addiction**
Abstract:

Amphetamine is the second most used drug worldwide of people within the age of 15-64. The physical side-effects of amphetamine addiction include: mental and cognitive impairment, emotional disturbances, physical health problems, and social problems. To further elucidate the biological basis of amphetamine addiction, we investigated the effects of amphetamine in the nucleus accumbens, a primary reward center of the brain. Amphetamine is known to cause structural changes in neurons, such as increased spine density; however, little is known about the effects on astrocytes. The present study investigated amphetamine-induced plasticity on astrocytes utilizing 2-photon microscopy and whole-cell patch clamp recordings. To examine astrocyte process motility with relation to dendritic spines in the presence of amphetamine, we utilized D2-GFP mice to visualize dendritic spines and sulforhodamine 101 to visualize astrocyte processes. To investigate the effects of amphetamine on astrocyte networks, biocytin was infused into astrocytes via whole-cell patch clamp current injection. The present study provides insight into the effects of amphetamine on astrocyte structure and plasticity and advances the knowledge on the biological basis of addiction.

Presenter: Renee Thomas
Poster Number: 59
Home Institution: Minneapolis Community and Technical College
Program: LSSURP
Faculty Mentor: Dr. David Thomas
Research Advisor: Dr. Tory Schaaf, Evan Kleinboehl
Poster Title: **Development of FRET Biosensors to Detect Specific Small-Molecule and Enzymatic Regulation of the Cardiac Calcium Pump SERCA**

Abstract: We have developed biosensors for the human cardiac Sarco-Endoplasmic Reticulum Calcium ATPase (SERCA) to measure changes in fluorescence resonance energy transfer (FRET) due to structural changes caused by binding of known inhibitors and small-molecule effectors to the calcium pump. Diminished SERCA expression or function can lead to high cytosolic calcium levels, which can inhibit natural cardiac cycles and lead to heart failure (HF). In order to develop novel therapeutics for HF, it is important to understand the structural/functional mechanisms of SERCA. Novel small-molecule activators were discovered through initial screening of SERCA tagged with green fluorescent protein (GFP) and red fluorescent protein (RFP). Using site-directed mutagenesis, we targeted specific residues to block the binding of these novel molecules as well as the known enzymatic regulator phospholamban (PLB). After transfection into HEK-293 cells, we will measure the FRET changes in wild-type and mutant SERCA. In the mutagenized SERCA, we expect to see no change of FRET in the presence of the effectors, indicating that the interaction was indeed obstructed. Understanding the specific residues of interaction for certain known effectors will be useful in developing and screening future therapeutic compounds.

Presenter: Alana Tillery
Poster Number: 60
Home Institution: University of Maryland - College Park
Program: LSSURP
Faculty Mentor: Dr. Suhasa Kodandaramaiah
Poster Title: **Rapid Desktop Inkjet Printing of an Inexpensive and High-Resolution Electrocorticogram**

Abstract: Brain-computer interfaces (BCI) have vast potential to augment and repair cognitive ability. Further, a class of BCI which measures neural activity at the surface of the brain is the electrocorticogram (ECoG). ECoG arrays made from flexible substrates that conform to the complex 3D topology of the brain surface are particularly advantageous for interfacing with large neural regions. To date, the fabrication of flexible ECoG has involved highly specialized methods of photolithography, spin coating, or laboratory grade printers. In this work, we present an implantable ECoG array fabricated entirely using commonly available desktop laboratory tools. ECoG fabrication began with desktop inkjet printing of silver nanoparticles (Ag nano) on photo paper and then the application of an insulator, PMMA, and a conductor, PEDOT:PSS, to the electrodes. The interelectrode distance is 400 μm and the range of electrode impedances is 0.815 - 102 kΩ. This work also describes the implantation of this ECoG in an anesthetized mouse. We expect that this approach will facilitate high-level customizability, accessibility, and rapid prototyping of ECoG that can be of use to the BCI community.

Presenter: Anibal Tornes Blanco
Poster Number: 61
Home Institution: University of Puerto Rico - Rio Piedras
Program: LSSURP
Faculty Mentor: Dr. Mark Schleiss
Poster Title: **Qualitative Characterization of Pentameric Complex (PC) Protein Cell Tropism**
Abstract: Congenital Cytomegalovirus (cCMV) is the most recurring viral infectious disease in pediatrics, causing major public health concerns. The National Academy of Medicine reacted by classifying the necessity for a cCMV vaccine as a Tier 1 priority. cCMV is a β – Herpes Virus, characterized by slow replication cycles and the capacity to lie dormant in its host without eliciting an immune response, making it especially harmful for immunocompromised individuals. cCMV is species-specific due to differential maternal-fetal transmission dynamics and development. The guinea pig cytomegalovirus (GPCMV) genome is homolog to the Human Cytomegalovirus (HCMV) in the Pentameric Complex (PC) between GPCMV as gH/gL/GP129/GP131/GP133 and HCMV as gH/gL/UL128/UL130/UL131. The tropism for endothelial and epithelial cells that line the placenta has been closely associated to the PC. We hypothesize that the ability of specific antibodies generated to target the PC confer the host an ability to block infection of epithelial and endothelial cells. Via western assays we qualitatively characterized GP133 and GP129, confirming that PC vaccination induced antibody response. In summary, cell-mediated immune neutralization response for all components of the PC may provide to be a valuable trait for a cCMV vaccine.

Presenter: Taylor Tuhy
Poster Number: 62
Home Institution: Virginia Polytechnic Institute & State Univ.
Program: LSSURP
Faculty Mentor: Dr. Colin Campbell
Poster Title: **The Hairpin Molecule can be used to Study the Same Repair Mechanism of DNA Protein Crosslinks as the Plasmid Molecule.**
Abstract: DNA protein crosslinks (DPCs) are formed when proteins become covalently bound to DNA; these damaging lesions can be caused by cancer chemotherapeutics, ultraviolet radiation, etc. The bulkiness of these lesions can stall replication and transcription machinery, therefore activating apoptosis within cells. Failure for cells to recognize and repair these drug induced DPCs can alter biological outcomes such as treatment failure which leads to patient death. To gain insight into this repair mechanism we have built a synthetic DNA structure that not only contains a covalently crosslinked human 8-oxoguanine glycosylase (hOGG1) protein; it also contains features which prevent degradation of the DNA. We will then electroporate our DNA substrate into the nuclei of purified human HEK293t cells and recover them for further analysis. From there we can calculate the percent of DPC repair as well as discover DNA repair intermediates throughout the incubation process. Overall, our goal is to discover more about how cancer cells respond to chemotherapeutic induced DPCs; this will lead to improving cancer treatments and prolong the lives of users.

Presenter: Darian Turner
Poster Number: 63
Home Institution: Alabama A&M University
Program: LSSURP
Faculty Mentor: Dr. Mark Schleiss
Research Advisor: Jason Zabeli, Claudia Fernandez-Alacron
Poster Title: **Quantitative Characterization of the Pentameric Complex of Congenital Cytomegalovirus by ELISA**

Abstract: Cytomegalovirus (CMV) is a common herpes virus that spreads through bodily fluids, making immunocompromised individuals at risk of infection. Pregnant women are also at risk of transferring cytomegalovirus to their children. This form of maternal-fetal transmission is known as Congenital CMV(cCMV). Because of the damaging birth defects that occur in infected newborns, vaccination for cCMV is a high priority. Our team has found that the virus forms a complex of five proteins known as the Pentameric Complex (PC) to infect endothelial and epithelial cells, and consequently the placenta. In humans, the proteins are gH/gL/UL128/UL130/UL131. However, CMV is species-specific, and due to the similar reproductive biology and infection in utero, guinea pig models were used for this project. The guinea pig PC proteins are gH/gL/GP129/GP131/GP133. Modified vaccinia Ankara (MVA) vectors were used to vaccinate the guinea pigs with the Pentameric Complex, and serum was collected and tested at different dilutions for antibody signal against these specific proteins via ELISA (Enzyme-Linked Immunosorbent Assay). Results regarding the PC at this time are inconclusive. However, the quantitative characterization of its proteins is a significant step forward in creating an effective vaccine.

Presenter: Lauren Turner
Poster Number: 64
Home Institution: University of Maryland - Baltimore County
Program: LSSURP
Faculty Mentor: Dr. Anna Lee
Poster Title: **Tracking Differences in Saccharin and Ethanol Consumption using a Novel 3D-Printed Lickometer**

Abstract: Current bottle systems used in rodent consumption studies lack a cost-effective and scalable way to track patterns of consumption. The most frequently used systems involve measuring bottle weights, which is a labor-intensive process that risks fluid loss through spilling. Using modern technology, this project developed a novel bottle system which allows for sorted, time-stamped consumption data without the frequent removal of bottles. We modified an existing 3D printable standard tessellation language file for a two-bottle holder so that we were able to include three bottles in our system. After 3D printing,photo-interrupters were inserted into the holder and connected to an Arduino. A single adult female C57BL/6J mouse was presented with water, saccharin, and different ethanol concentrations. The data was downloaded and further sorted and graphed in Excel which showed the consumption relationships between the substances. Future studies will include examining the relationship between alcohol and nicotine to further understand the high abuse comorbidity between these two substances. The development of this novel system will provide a better understanding of the consumption relationships between a multitude of substances.

Presenter: Minh-Tu Van
Poster Number: 65
Home Institution: University of Minnesota - Twin Cities
Program: LSSURP
Faculty Mentor: Dr. Eric Watkins
Research Advisor: Florence Sessoms, Jon Trappe
Poster Title: **Decreased Growth and Yield Response of Red River Crabgrass with L-m-tyrosine Exposure**
Abstract:

Fine fescue grasses are becoming more widely used in lawns due to their low nutrient and water input. Furthermore, research on its potential allelopathy, or release of biochemicals that affect growth of surrounding plants, is becoming an important research topic due to its potential applications in lawns and agricultural systems. It has been observed that certain fine fescue grasses secrete a root allelochemical that affect the growth of lettuce, arabidopsis, sorghum, tomato, and more. Field research part of the national turfgrass evaluation program (NTEP) showed that these species exhibited higher weed control than other species. However, separating true allelopathy from resource competition (e.g. nutrient & light) continues to be difficult. Bertin at el. observed that the main component of the allelochemical released by fine fescue root systems is L-m-tyrosine, a non-protein amino acid that may hinder root growth of surrounding plants. We would like to confirm Bertin at el.'s observations; we will modify their procedure by using agar gels with various concentration of L-m-tyrosine. Secondly, we will observe crabgrass yield response in the presence of different fine fescue grasses accessions and cultivars. Lastly, we will attempt to separate the allelopathic factor from other competitive factors by conducting a hydroponics experiment. We believe that the allelochemical produced by fine fescue grasses, namely L-m-tyrosine, will inhibit crabgrass growth and reduce its yield. There was a inverse significant correlation between levels of m-tyrosine and crabgrass root lengths. In addition, results from the seed head count and hydroponic experiment, albeit not statistically significant, suggest that L-m-tyrosine may reduce crabgrass seed head yield and root growth. These findings suggest that rather than using herbicides, breeding fine fescues with these potential allelochemical tendencies could reduce the need for herbicides.

Presenter: Erik Velez Perez
Poster Number: 66
Home Institution: University of Puerto Rico - Mayagüez
Program: LSSURP
Faculty Mentor: Dr. Kathleen Thomas
Research Advisor: Max Herzberg
Poster Title: **Greater Hippocampal and Prefrontal Activity in Stressed Adolescents**
Abstract:

Allostasis refers to an individual's physiological ability to adapt to stressors. Previous experiences and social support may help individuals to cope with stress more adaptively (McEwen, 2017). In adolescence, however, parent presence during a stressful task has no effect on the cortisol response (Hostinar, Johnson & Gunnar, 2015). In this study, young adolescents (ages 11-14, N=24) were exposed to the Minnesota Imaging Stress Test in Children, a task that includes delivering a speech and completing math problems in front of judges during an fMRI scan. Salivary cortisol samples were taken and used to identify two groups, one that exhibited a physiological response to the task (responders) and one that did not (non-responders). Parent support was measured using the Network of Relationships Inventory (NRI). Our fMRI analysis revealed greater activations in the right hippocampus, right dorsolateral prefrontal cortex, and left ventrolateral prefrontal cortex when contrasting responders and non-responders. Non-responders did not exhibit any regions with greater brain activity. Parent support was not correlated with the cortisol stress response. We suggest that functional allostatic plasticity in the hippocampus and prefrontal cortex might contribute to stress adaptation. Future work is needed to understand the mechanisms of allostatic plasticity and stress adaptation during adolescence.

Presenter: Jessica Weng
Poster Number: 67
Home Institution: Rice University
Program: LSSURP
Faculty Mentor: Dr. Jeffrey Miller
Poster Title: **IL-12-based Trispecific Killer Engager (TriKE) to Enhance Natural Killer Cell Function Against Myeloid Leukemia**

Abstract: Myeloid leukemias have poor prognosis, especially among the elderly population. Natural killer (NK) cell immunotherapy has been for cancer treatment since the 1980s. Despite this, the clinical efficacy of NK-based immunotherapies is minimal, likely a result of lack of activation and cancer cell specific-targeting. A drug was developed at the University of Minnesota called 161233 TriKE. The tripartite molecule contains an anti-CD16 portion which activates CD16, a potent activating receptor expressed by NK cells. Second, the anti-CD33 portion targets leukemic cells by binding CD33, expressed on many myeloid cancers. Third, functionalization of the molecule with IL-12 results in NK cell activation and production of interferon gamma (IFN- ɣ), a proinflammatory cytokine. These three arms of the drug attempt to enhance NK cell function against leukemia cells. Functional assays show that 161233 TriKE significantly increases IFN- ɣ production compared to an older generation 1633 Bispecific Killer Engager (BiKE). Binding assays confirmed enhanced binding of the TriKE to NK cells relative to controls. Further functional and downstream signaling assays will be done to investigate potential synergy between molecules and their effects on cell responses. These data provide evidence that 161233 TriKE may represent a novel and promising immunotherapeutic strategy for cancer treatment.

Presenter: Amelia Windorski
Poster Number: 68
Home Institution: Smith College
Program: LSSURP
Faculty Mentor: Dr. Patrick Rothwell
Poster Title: **Establishing a Model for Oxycodone Exposure and Withdrawal in Mice**

Abstract: The continuous release version of oxycodone (oxy) is prescribed to be taken once every 12 hours. The drug effect often diminishes before the next dose, resulting in intermittent periods of withdrawal and intense drug craving. The oscillation between the peaks and troughs of drug exposure can lead to addiction and warrants further study. We established a mouse model for this cycle by implanting mice with programmable iPrecio pumps that administered drug (63.2mg/kg/day) in an intermittent exposure cycle. Mice were exposed to Oxy for eight hours followed by four hours of no drug exposure, during which we expected the mice to experience spontaneous withdrawal. A litany of tests were used to assess changes in behavior and blood drug levels during withdrawal. These results were compared to continuous oxy and saline controls. Mice in the intermittent group showed greater global withdrawal scores starting two hours after the pumps shut off ($p < 0.006$). Locomotor depression ($p < 0.002$), increased resting time ($p < 0.007$), and decreased blood oxycodone levels ($p < 0.02$) corroborated the presence of withdrawal in mice with intermittent exposure. This research shows that iPrecio pumps can effectively model Oxy exposure and spontaneous withdrawal in mice.