Online Proceedings

Poster Session 1

Commons East, Easel 66

11:00 AM to 1:00 PM

Assessing Methods for Sampling the Blue Point Pyrg: A Springsnail Endemic to Southern Nevada

Cody Cris, Senior, Biological Sciences, University of Nevada Las Vegas

Louis Stokes Alliance for Minority Participation, McNair Scholar

Mentor: Jef Jaeger, Departmenf of Life Sciences, University of Nevada, Las Vegas

Mentor: Chenoa Wilcox, Department of Life Sciences, University of Nevada, Las Vegas

Springsnails are a broad group of highly diverse North American gastropods, with the most speciose genus being *Pyrgulopsis* (139 species). These tiny aquatic gastropods are often found in thermally-influenced freshwater springs, where endemism is common. Restricted occurrence and narrow habitat requirements of many species has generated conservation concern because of anthropogenic threats to spring systems and associated aquafers. The Blue Point Pyrg is endemic to Blue Point Spring in Lake Mead National Recreation Area within the eastern Mojave Desert. Two other springsnail species also occupy this unique spring. Little is known, however, about the distribution, abundance, and habitat associations of these springsnails. As a first step in a larger study investigating these parameters, we addressed methodological questions associated with the use of artificial tile samplers for long-term monitoring of springsnail abundance. We determined that the area of our tile samplers was adequate for sampling these springsnails and that our multiple, replicate tiles did not produce significantly different measures of abundance within our one meter sampling areas. These results indicate that monitoring could be accomplished with fewer tile replicates. We identified all three springsnail species within our samples and determined that identification of adult springsnails by shell morphology under magnification appears possible; we are currently developing genetic markers to confirm the morphological assessment. The relative abundance of springsnails peaked at approximately four meters below the springhead, and then dropped precipitously further downstream. Limited numbers of springsnails downstream may be related to the presence of nonnative, predatory fishes. We are currently investigating this hypothesis, and the findings of our methodological assessment have allowed us to use half of our sampler replicates in a fish exclusion experiment to assess the impact of predation.

Poster Session 2

Balcony, Easel 108

1:00 PM to 2:30 PM

Characterizing Stem Cell Specific-Kinases as Regulators of Self-Renewal in Rhabdomyosarcoma

Shelly Lin, Senior, Biology (General)

Mentor: Eleanor Chen, Pathology

Mentor: Thao Pham, Pathology

Rhabdomyosarcoma (RMS) is the most common pediatric soft tissue sarcoma. The survival outcomes are poor for patients with relapsed and metastatic disease. Embryonal RMS (ERMS), a major subtype of RMS, is driven by genetic mutations in the RAS pathway. Cancer stem cells (CSCs) drive the process of self-renewal to recapitulate the heterogeneity of the cancer and are thought to be responsible for cancer relapse due to their resistance to conventional chemotherapy. In ERMS cells, a population of CSCs has been identified and is believed to play a critical role in tumor relapse and metastasis. A previous screen of all known human kinases identified candidates that played a role in regulating self-renewal of ERMS CSCs. I have prioritized two most promising candidates, ERN1 and MAPK10, for further characterization. I hypothesize that these kinases regulate the self-renewal capacity of CSCs but do not affect growth of non-CSC tumor cells. Using the gene-editing tool, CRISPR (Clustered Regulatory Interspaced Short Palindromic Repeats)/Cas9, I knocked out ERN1 and MAPK10 in ERMS cells and assessed their role in regulating ERMS CSC cell growth and self-renewal. To assess whether the effects of gene disruption are specific to the changes in the CSC population in vitro and in vivo, I used an ERMS CSC reporter cell line that specifically expresses Green Fluorescent Protein (GFP) in CSCs. The ERMS CSC reporter cell lines harboring ERN1 or MAPK10 gene disruption are subjected to self-renewal assays in cultured ERMS cells in vitro and ERMS xenograft model in vivo. My findings will provide new insights into the biological mechanism underlying as well as new potential targets against relapse and metastasis of ERMS.

SESSION 2S

THE POWER OF MEDIA REPRESENTATIONS AND DIGITAL ARCHIVES

Session Moderator: Carmen Gonzalez, Communication
JHN 175
3:30 PM to 5:15 PM
* Note: Titles in order of presentation.

Presenting Digitized Historical Manuscripts: A User-Centered Approach
Yogasai Gazula, Sophomore, Linguistics, International Studies: Asia
UW Honors Program
Cheryl Wu, Freshman, Pre-Sciences
Simon Talusan, Freshman, Pre-Sciences
Darren Huang, Junior, Pre-Sciences
UW Honors Program
Daniel Kim, Sophomore, Pre-Major (Arts & Sciences)
Jennifer Wang, Sophomore, Pre-Major
Chuangzuo Liu, Junior, Pre-Major (Arts & Sciences)
Corina Geier, Senior, Mathematics
Nicholas Alexander Verghese, Sophomore, Pre Engineering
Mentor: Annie Chen, Biomedical Informatics and Medical Education, University of Washington School of Medicine
Mentor: Walter Andrews, Near Eastern Languages and Civilization

The Svoboda Diaries Project works with personal diaries written at the turn of the 19th century, capturing over 40 years of the life, politics, and landscape of Ottoman Iraq. Written through the unique lens of a British steamship purser with a rich family history and connections in the area, these texts provide a unique insight into a locale on which there exists minimal literature for this time period. Undergraduate interns transcribe these diaries and develop open-source tools to make the texts available in a variety of formats. We are currently redesigning our website to better serve the needs of its various users, and our main question is: How do we realize the needs of prospective users when creating a digital platform for viewing historical manuscripts? Our current website is not sufficient in meeting the needs of the project's diverse users: historians/researchers, contributors, and the general public. Therefore we intend to create an engaging and interactive user interface. At its core, the Svoboda Diaries comprise a personal narrative. We also intend to infuse a storytelling approach to present these unique documents in a larger political and historical context, and allow the user to explore them in different ways. We utilize a variety of user-centered research and design methods, such as conducting user interviews with domain experts and other interested individuals, creating prototypes, and conducting pilot usability sessions to refine the website.We anticipate that our website redesign will allow users greater access to explore the diaries. In addition, the redesign will draw attention to the most important aspect of the website: the diaries themselves, and the fascinating and valuable accounts within them.

POSTER SESSION 3

MGH 258, Easel 180
2:30 PM to 4:00 PM

Variants in STUB1 Causes Autosomal-Dominant Late-Onset Spinocerebellar Ataxia
Elyana Lux Heigham, Senior, Neurobiology
UW Honors Program
Mentor: Dong-Hui Chen, Neurology

Heritable spinocerebellar ataxias (SCAs) are rare genetic neurological disorders that affect the cerebellum and sometimes the spinal cord. As a result, those with SCA often have problems with movement and coordination. In my experiment, I analyzed a pedigree where several family members had a dominant, late-onset spinocerebellar ataxia. The family tested negative on the tests for all known variants causing dominant spinocerebellar ataxias. Thus, my goal was to find what genetic variant was responsible for the SCA in this pedigree. I began by obtaining exome sequences for two of the affected family members. The exome sequences listed all of the variants present in each individual's exome. My approach to variant filtering steps were selecting shared heterozygous variant, an assessment of population frequency, functional significance, and evolutionary conservation, and then prioritizing the remaining variants based on candidate gene function and expression, animal models and relevance to neurologic disease. The process resulted in a list of candidate variants. I then created primers for my candidate variants and ran PCRs with DNA samples of both affected and unaffected family member. The DNA fragments generated from the PCR were then sequenced. The only candidate that cosegregated was a variant c.158T>C, p.Ile53Thr in STUB1 a gene known to be associated to a recessive SCA. The pathology study to review the abnormality in the patient autopsy brain was performed by our collaborators. Our finding confirmed that STUB1 can cause autosomal dominant hereditary cerebellar ataxia in addition to recessive form of this disease. It is increasingly apparent that variants once associated only with one form of inheritance are in fact capable of causing both recessive and dominant forms.

POSTER SESSION 3

MGH 258, Easel 179
2:30 PM to 4:00 PM

Identification of a Causal Gene for a Novel Form of Hereditary Spastic Paraplegia

Olga Sarby Cherepakhin, Senior, Biology (Molecular, Cellular & Developmental), Anthropology: Medical Anth & Global Hlth

Mary Gates Scholar, UW Honors Program

Mentor: Dong-Hui Chen, Neurology

Hereditary Spastic Paraplegia (HSP) is a classification for a group of neurogenetic diseases that cause affected individuals to have severe contractions and stiffness in the lower limb muscles. This group includes a genetically diverse range of disorders that vary in age of onset, rate of progression, and severity. The purpose of my project is to identify the causal gene involved in this novel form of HSP. My lab acquired DNA samples from affected and unaffected members of a family with an unassigned autosomal dominant HSP. At first, we obtained whole exome sequencing on DNA from three affected family members. In a file containing all the variants (differences from a reference DNA exome) detected in any of these three subjects, we looked for previously identified causal variants to ensure that the family did not have a known HSP subtype. Then we identified potential variants by filtering for those that were present in one copy (heterozygous) in all three family members, since this form is autosomal dominant. We further filtered them for low prevalence in the Genome Aggregation Database, since HSP is not a common disorder. Candidate variants for testing were selected based on their predicted change in the protein, relevance of gene function, and predicted impacts from CADD and GERP models. We are currently in the process of analyzing the candidates. We are amplifying and sequencing them using DNA from all the family members to determine whether these variants are present in all those who are affected and absent from those who are not affected, since that is how the causal variant would present. My project will contribute to our understanding of the pathogenesis of and improve clinical diagnostics for HSP. The implications of my research could also extend to other genetic disorders if a novel genetic mechanism is elucidated.

POSTER SESSION 3

Balcony, Easel 112

2:30 PM to 4:00 PM

Loss of Vacuolar Acidity and Iron-Sulfur Cluster Production Affect Yeast during Aging

Dexter Euwen Chen, Senior, Biochemistry, Philosophy

Rachael Kate Tran, Senior, Spanish

Justin Drake Dillard Telm, Sophomore, Engineering Undeclared

Mentor: Kenneth Chen, Genome Sciences

Mentor: Matt Kaeberlein, Pathology

The lysosome is a critical organelle affected by the aging process. Changes to lysosomal physiology during aging and their effects on cellular function are poorly understood. Here, we study the lysosome-like vacuole of the budding yeast during replicative aging. Using a microfluidic device, we trap hundreds of mother cells and visualize them over the course of their replicative lifespans. Using strains with fluorescent protein tags we investigate protein abundance, intracellular physiological conditions, and organelle morphology. We find that during aging, the vacuole becomes progressively less acidic. Using a vma mutant as genetic model of vacuolar pH dysfunction, we find that loss of vacuolar acidity triggers a loss of respiratory capacity, loss of iron-sulfur cluster protein activity, and iron starvation response. We find that iron supplementation rescues both the loss of respiratory capacity and shortened replicative lifespan of vma mutant cells. We see that during aging, a similar iron starvation response occurs, correlated on a single-cell level with the loss of vacuolar acidity. We propose that the loss of iron-sulfur cluster synthesis capacity due to loss of vacuolar acidity is a conserved process underlying multiple aging phenotypes.

POSTER SESSION 3

Balcony, Easel 113

2:30 PM to 4:00 PM

Automation of Budget Microscopes for Microfluidic Yeast Replicative Lifespans

Toby Nathan Ven, Senior, Bioengineering

Mentor: Kenneth Chen, Genome Sciences

Mentor: Matt Kaeberlein, Pathology

Recently, microfluidic technologies have been developed to allow higher throughput collection of yeast replicative lifespan data. Adoption of these devices has been limited, in part, due to the high cost of the motorized microscopy instrumentation from mainline manufacturers. Inspired by recent development of open source microscopy hardware and software, we developed minimal-cost hardware attachments to provide long-term focus stabilization for lower-cost microscopes and open-source software to manage concurrent time-lapse image acquisition from multiple microscopes. This functional 3D printed scaffold along with attached electronics has shown to capture accurate yeast lifespans without costing as much as other third party autofocusing system. We hope that this can help spur the wider adoption of microfluidic technologies for the study of yeast replicative aging.

POSTER SESSION 3

MGH 241, Easel 145

2:30 PM to 4:00 PM

Evaluating Natural Threats to the Grays Harbor PUD's Power System

Blake Donald (Blake) Rose, Junior, Electrical Engineering
Mentor: Daniel Kirschen, Electrical Engineering

Natural disasters pose a great threat to power systems. They can cause major damage to power lines and substation equipment, resulting in widespread outages. It can then take days to weeks and millions of dollars to restore power to customers. Power system resilience is concerned with reducing the impact of disasters on power systems. The purpose of this study is to evaluate the resilience of one power utility in southwest Washington, the Grays Harbor PUD (GHPUD), to earthquakes, earthquake liquefaction, and tsunamis. This study will consider the likelihood of these disasters, potential effects on the GHPUD's electrical system and its customers, and possible mitigation techniques. I have collected seismic hazard curves, response spectra, and liquefaction susceptibility data at GHPUD substation sites from US Geological Survey maps and the M9 project. I have also collected data on tsunami inundation depth at substation sites from Washington Geological Survey maps. With this information I have determined which of their substations are most at risk from these disasters. This data will be used to determine possible impacts on substation equipment and to estimate the probability of equipment failure. When completed, the results of this study can allow the Grays Harbor PUD and other power utilities to be more prepared for future disasters and to be more informed of potential resilience improvements.

POSTER SESSION 3

Balcony, Easel 106

2:30 PM to 4:00 PM

Characterizing the Effects of CRISPR/Cas-mediated Disruption of *PTPN11* in Pediatric Rhabdomyosarcoma

Henna Angel Di, Junior, Biology (Physiology)
Mentor: Eleanor Chen, Pathology
Mentor: Terra Vleeshouwer-Neumann, Pathology

Embryonal rhabdomyosarcoma (ERMS) is a devastating pediatric cancer that affects soft tissue such as skeletal muscle and connective tissue. Currently, there is no effective treatment for patients with ERMS. Mutations in the gene *PTPN11* are found to have cancer-promoting roles in leukemia, lung, and breast cancers, but its role in ERMS is virtually unknown. *PTPN11* codes for the SHP-2 protein, which is a component of the RAS/MAPK (mitogen-activated protein kinase) signaling pathway. Abnormalities in this pathway are known to transform normal cells into cancer cells when certain proteins are upregulated. My central hypothesis is that *PTPN11* promotes RMS tumor growth by allowing cells to proliferate and differentiate out of control. To test the hypothesis, I used CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats)/Cas gene editing technology to disrupt *PTPN11* gene function in two models. I designed constructs that express guide RNAs that target the *PTPN11* locus in the zebrafish or human genome. If *PTPN11* has a tumor-promoting role, I expect targeted disruption of zebrafish PTPN11, delivered via microinjection, to result in reduced RMS tumor formation and growth compared to zebrafish tumor with no *PTPN11* gene disruption. In the human ERMS cell lines, I will use virus-mediated transfer to introduce the CRISPR DNA construct *in vitro*. My hypothesis will be supported if the cells harboring targeted disruption of *PTPN11* have reduced growth and less self-renewal capacity compared to the control cells. My findings will elucidate the role of *PTPN11* in RMS, which will allow further research into the potential therapeutic benefit of targeting *PTPN11* in pre-clinical RMS models.