UNIVERSITY of WASHINGTON

Undergraduate Research Symposium May 19, 2017 Mary Gates Hall

Online Proceedings

POSTER SESSION 1

MGH 206, Easel 170

11:00 AM to 1:00 PM

Seeing Eye to Eye: Identifying Genetic Modifiers of Alzheimer's Disease Proteins Expressed in *Drosophila* Eyes

Natalie Pearlman, Junior, Biology (Molecular, Cellular & Developmental)

UW Honors Program

Fatima Mohamed El Ghazali, Senior, Neurobiology

Mentor: Daniel Promislow, Department of Pathology, University of Washington School of Medicine

Mentor: Adrienne Wang, Pathology

Alzheimer's Disease (AD), the most common form of dementia, is the sixth leading cause of death in the United States. With a prevalence of over five million cases, the number of AD patients is expected to increase as a new diagnosis of AD is made every sixty-six seconds. Early symptoms include short-term memory loss and gradually progress to devastating deficits in cognition, temperament, and behavior. AD is a neurodegenerative disease linked to the accumulation and aggregation of two proteins, A$\beta_1 - 42$ and hyperphosphorylated Tau, which form the characteristic amyloid plaques and neurofibrillary tangles found in the brains of AD patients. Previous studies support the notion that genetic variation underlies the wide array of phenotypes observed among AD patients, and several genes have been discovered to cause early onset AD. In sporadic (late onset) AD patients, it remains unknown what effects natural variation may have on the expression and severity of the AD phenotype. To explore the influence of genes on AD, our study utilizes a *Drosophila* model of AD that allows us to express both the A$\beta_1 - 42$ and Tau peptides in the fly eye. Expression of AD-related proteins in the eye leads to degradation of the hundreds of ommatidia that make up the fly eye. To quantify the extent of ommatidial degradation, we use a computer program, ImageJ, which measures the size and circularity of each ommatidium. To introduce natural variation, the AD fly is crossed with flies from the *Drosophila* Genetic Reference Panel (DGRP), consisting of 192 fully sequenced isogenic lines. By comparing the A$\beta_1 - 42$ and Tau-induced degradation in each background, we can identify naturally occurring modifiers of the AD phenotype. We have already identified promising hits, which could eventually help in the development of novel targeted therapies.

POSTER SESSION 1

MGH 206, Easel 171

11:00 AM to 1:00 PM

Drosophila as a Model of Rotenone-Induced Parkinson's Disease

Rebekah Zaharia, Senior, Biology (Molecular, Cellular & Developmental)

Justin Chandler Bethel, Senior, Neurobiology, Biochemistry

Mentor: Daniel Promislow, Department of Pathology, University of Washington School of Medicine

Mentor: Adrienne Wang, Pathology

Parkinson's Disease (PD) is an age-related neurodegenerative disorder that leads to progressive decline in motor control. Tens of thousands of new cases are diagnosed each year. Many cases of PD are linked to chronic exposure to rotenone, a commonly used pesticide. As an inhibitor of complex I in the mitochondrial electron transport chain, rotenone causes deterioration of dopamine-producing neurons in the substantia nigra (part of the midbrain). Studies suggest that there is a genetic component to how likely one is to develop symptoms in response to rotenone exposure. To test this hypothesis, we used the *Drosophila* Genetic Reference Panel (DGRP), a set of 192 fully sequenced inbred lines of the fruit fly, *Drosophila melanogaster*. Exposure of *Drosophila* to rotenone has been shown to cause dopaminergic-specific degeneration, similar to the pathology observed in PD patients. When fed highly concentrated rotenone-infused food, we see significant variation in rates of survival among female flies from different lines of the DGRP, allowing us to capture natural variation in response to rotenone exposure. After most of the DGRP has been tested, we will carry out a genome-wide association study to identify specific genetic variants associated with rotenone resistance. In this way, we hope to uncover genetic factors that could point to specific mechanisms behind PD pathogenesis. With further related research, new genetic pathways could lead to new drug targets.

POSTER SESSION 3

Commons East, Easel 73

2:30 PM to 4:00 PM

VoiceBox Head Unit

Sean Leisle, Senior, Electrical Engineering
Dinggao (Pan) Pan, Senior, Electrical Engineering
Jiaqi (Evelyn) Zhang, Senior, Electrical Engineering, Applied & Computational Mathematical Sciences (Scientific Computing & Numerical Algorithms)
Yang Mi, Senior, Electrical Engineering
Mentor: Jenq-Neng Hwang, Electrical Engineering
Mentor: Barry Roitblat

The purpose of this project is to develop an automotive "head unit" entertainment system which can be installed into existing vehicles. It employs several connectivity technologies such as bluetooth, Wi-Fi, cellular, USB, and radio. It is also integrated with the vehicle's data bus for indoor temperature and fan speed control. It includes features for sending or receiving texts and calls from a bluetooth connected phone, playing music from multiple sources, Point of Interest search and navigation around local areas or to a specified location. In addition, it has over the air update and upgrade capabilities, allowing additional content to be added to the system after deployment. Users can interact with the system through both voice and touch inputs. This system is being built with a Dragonboard 410C at the heart, running a modified version of Android. Connected to this board will be a 7" touchscreen, a CAN bus controller, a software-defined radio, and a cell modem. Included inside the board is a bluetooth module, a WiFi module, and adequate internal storage. The voice recognition technology is supported by VoiceBox's cloud platform - VIBE. This project was developed in collaboration with VoiceBox Technologies.