San Miguel Lab overview

Our lab is dedicated to addressing elusive biological questions from a quantitative, systems-level perspective. We combine microfluidics with custom-built experimental platforms, quantitative image processing, statistical and mathematical data analysis, to study the well-known model organism C. elegans.

Developing novel tools to study aging in C. elegans - Sahand Saberi

- The PVD neuron is responsible for harsh-touch sensation and thermal-sensation, and it degenerates due to aging and exposure to acute cold-shock.
- I performed sophisticated segmentation of neurodegenerative bubble-like protrusions that formed along the PVD neuron with exposure to acute cold-shock.
- The morphological variations of the PVD neuron due to degeneration were quantified and various neurodegenerative patterns were identified by extracting 46 metrics describing the structure of the neuron.

Forward Genetic Screens for Aging Pathways - Daniel Midkiff

- Forward genetic screens can identify genetic mutations that cause a specific phenotype.
- We are using microfluidics and automated image analysis to perform a high-throughput forward genetic screen for increased aggregation levels of PAB-1 protein.
- We have identified at least two lifespan mutants so far, showing that protein aggregation can be used to identify lifespan mutants.

In vivo longitudinal tracking of C. elegans aging genes - Javier Huayta

- Several environmental factors affect the longevity of C. elegans, many of which function through the DAF-16/FOXO transcription factor. Our goal is to elucidate how daf-16, when driven by environmental stressors, affects lifespan.
- We are using a GFP-tagged strain, generated with the CRISPR/Cas9 system, to characterize DAF-16 activity.
- Using fluorescent imaging, we can monitor gene expression under different stressors, allowing us to relate stressors, gene expression, and lifespan.

In vivo monitoring of head injury cellular damage in Alzheimer’s disease in C. elegans - Rita Tejada

- Traumatic brain injury (TBI) has been widely associated with an increased risk of developing neurodegenerative diseases, such as Alzheimer’s disease (AD).
- The nematode C. elegans provides a unique experimental platform to perform quantitative analysis of injury, neurodegeneration, and neuronal function in AD models.
- We developed a microfluidic platform that enables performing controlled neuronal injury

Microfluidic Ricochet Particle Separation - Andrew Clark

- The Manta Ray uses a ricochet filter feeding mechanism using inertial particle separation.
- Larger particles continually bounce off of filter lobes and into the main stream.
- Smaller particles and fluid exit through the pores.
- We intend to use this filtering mechanism in a microfluidic device to create a high-throughput microparticle filter.

Building Quantitative Statistical Models to stressors in C. elegans - Karthik Suresh

- Oxidative stress is mediated by SKN-1 Pathway in C. elegans. It is homologous to Nrf2 pathway in humans.
- Recent literature indicates differential response to individual oxidative stimulants. Previously it was theorized all compounds elicit similar kind of SKN1 response from the organisms.
- Statistical Design of Experiments can help elucidate differential response of the organisms subjected to simultaneous stressors under different process parameters such as dietary restriction and heat stress.

Alzheimer’s Disease Pathogen Modeling - James Lichty

- Current models of Alzheimer’s Disease in C. elegans lack key aspects that may play a role in disease progression.
- Recent research has suggested that infection with certain pathogens may initiate Alzheimer’s Disease in humans.
- We aim to improve our models by including human pathogens and monitoring neuron health over time.

Group picture at NC State Farmers Market, which is just a short drive from our lab.