UNIVERSITY of WASHINGTON

Undergraduate Research Symposium May 19, 2017 Mary Gates Hall

Online Proceedings

Poster Session 1

Commons East, Easel 64

11:00 AM to 1:00 PM

A Survey, Model Design, & Benchmark Test Implementation of Agent-Based Applications

Caleb Jonah Ezra (Caleb) Yang, Senior, Computer Science & Software Engineering, Mathematics (Bothell Campus)
Mentor: Munehiro Fukuda, Computing and Software Systems, University of Washington Bothell

Our research at the Distributed Systems Laboratory of the University of Washington Bothell seeks to develop and release an agent-based simulator (Multi-Agent Spatial Simulation C++) for researchers in various fields such as in biology, business/industry, and economics/social sciences. Researchers already use agent-based modeling techniques; where the chosen micro-behavior for agents produce the simulation's macro-behavior. For example, the life-cycle of a mosquito agent is a chosen micro-behavior a researcher would use to model the spread of disease in a city. Which contrasts with the assumed macro-behavior in differential equation based simulations. However, researchers do not use a universal application that is easily programmable, but instead have the overhead of learning how to use other dedicated applications. My objective is to utilize the identified core logic of new agent-based models by coding them as a benchmark test program, further improving the in-house simulator through the benchmark analysis. The benchmark analysis will compare speed, performance efficiency, and scalability with RepastHPC and FLAME. RepastHPC and FLAME will be considered the alternatives because of their known popularity and usability in the research of agent-based modeling. We expect and have already found that not all applications will be capable of being directly ported because of factors such as an agent's micro-behavior, how agents communicate, even how the topology is constructed. Where a topology is how the system arranges the environmental elements such as in slices of a 2D plane, or a network of connected nodes. This information can be used to improve the in-house simulator's library for future releases. Additionally, with the benchmark analysis we can determine empirically whether or not the in-house simulator is a better alternative for researchers.

Session 1I

McNair Session - Exploring Science from Cells to Exoplanets

Session Moderator: Janneke Hille Ris Lambers, Biology

MGH 254

12:30 PM to 2:15 PM

* Note: Titles in order of presentation.

Pollen Preference of *Osmia lignaria* (Hymenoptera: Megachilidae) in a Commercial Blueberry Field

Margaret Elizabeth (Maggie) Hartman, Senior, Fisheries and Wildlife Management, Northrn Michigan Unv
McNair Scholar
Mentor: Rufus Isaacs, Entomology, Michigan State University
Mentor: Mario Pinilla-Gallego, Entomology, Michigan State University

Alternative pollinators such as the mason bee (*Osmia spp.*) are becoming increasingly important in agriculture as European honey bees (*Apis mellifera* L.) continue to decline. *Osmia lignaria* are especially important pollinators because they make contact with the anther and stigma during nearly every flower visit and are less likely to rob the plants of nectar without pollination. Previous studies indicate *O. lignaria* preferentially visit plants in the Rosaceae family, such as apple, cherry, pear and plum. The objectives of this study were (1) to analyze pollen loads in relation to proximity of natural areas surrounding the blueberry field and (2) to determine their pollen preference in a blueberry monoculture ecosystem. This study was conducted in a commercial blueberry field in southwestern Michigan. Pollen samples from flowering plants and *O. lignaria* nests were collected. Acetolysis was performed on all pollen samples, and pollen from *O. lignaria* nests was qualitatively and quantitatively analyzed. At this study site, *O. lignaria* collected pollen from white clover (Trifolium repens) and black cherry (Prunus serotine) most often. In spite of its availability, pollen collected from blueberries (Vaccinium corymbosum) accounted for significantly less of the total pollen. These findings are useful in determining the viability of *Osmia* species as crop pollinators. Based on the results, *Osmia lignaria* would not be a good option for

blueberry pollination.

POSTER SESSION 3

MGH 241, Easel 162

2:30 PM to 4:00 PM

Examining Mutations in Presenilin 2 Associated with Alzheimer's Disease

Leah Ariel Osnis, Senior, Biochemistry
UW Honors Program
Mentor: Suman Jayadev, Neurology
Mentor: Susan Fung, Neurology

Mutations in the gene Presenilin 2 (PSEN2) cause familial Alzheimer's disease (fAD). FAD shares clinical and pathological features of sporadic, late onset AD thus fAD cell models can be useful to study mechanisms relevant to all forms of AD which is critical to developing effective AD therapeutics. Presenilin 2 protein (PS2) forms the catalytic subunit of the γ-secretase complex, which cleaves amyloid precursor protein and releases Aβ1-42, considered a pathogenic contributor to Alzheimer's disease (AD). Our laboratory is interested in a fAD associated PSEN2 mutation, a frameshift two base-pair deletion (PSEN2 K115Fx) . The PSEN2 K115Fx is predicted to either lead to a truncated protein suggesting that the mutation may create a shortened peptide that interferes with normal cellular function (dominant negative or toxic gain of function) or result in degradation of the RNA transcript and subsequent loss of normal amount of PS2 protein (loss of function). To better understand how PSEN2 mutations cause disease, we have two objectives. The first aim of this project is to determine if the PSEN2 K115Fx does indeed result in a truncated protein or influence levels of wildtype PS2. I will be collecting human cultured fibroblasts isolated from AD patients with the PSEN2 mutations or controls and prepare cell lysate for analysis by Western blot. My colleague will also be analyzing mRNA levels from those same samples to determine the stability of the PSEN2 mutant and wildtype transcripts in all cases. The second aim is to determine the impact of the mutation on PS2 enzymatic activity. I will culture the cells described above, then infect with a luciferase based enzyme reporter assay to compare the impact of PSEN2 mutations on γ-secretase mediated cleavage of APP. My work will help identify the candidate mechanisms by which the PSEN2 K115Fx mutation causes AD.