**Effects of Individual Orsay Virus Proteins on The Intracellular**

**Pathogen Response of C. Elegans**

**Presenter: Barbara Chen (Biology)**

**Faculty Mentor: Dr. Jessica Sowa (Biology)**

In order to cause infection, pathogens must avoid destruction by the host immune system. However, how pathogens evolved to evade host immunity is not fully understood. Mammals have two systems of immunity, innate and adaptive, which are regulated by complex interactions between the two systems. In order to study innate immunity exclusively, the roundworm C. elegans is a useful invertebrate model because it lacks adaptive immunity and the complexities that arise from interactions between the two types of immunity. The goal of this project is to investigate whether the individual expression of the Orsay virus capsid and delta-fusion proteins can suppress the Intracellular Pathogen Response (IPR) in C. elegans. The IPR is an innate immune response activated by the Orsay virus. To investigate the effects of the viral proteins on the IPR, molecular cloning will be used to construct plasmids that will be microinjected into C. elegans, creating transgenic animals that overexpress each of the two Orsay proteins. We will then test the animals to observe the individual effects of the capsid protein and delta-fusion protein on IPR suppression. From this we will better understand how viruses evolved to evade the host immune system.