Gabrielle Law: Chemistry & Biochemistry
Mentor: Matthew Taylor -- Microbiology & Immunology
Assessing evolutionary pressure on Herpes Simplex Virus Type 1 genomes during infection and spread

This project will quantify evolutionary pressure, through mutation and selection, of herpes simplex virus type 1 (HSV-1) during replication and spread. HSV-1 infection involves replication and spread in neurons, in part contributing to HSV-1 as the leading cause of viral encephalitis in developed countries. HSV-1 is a dsDNA virus with an accordingly low point mutation rate. Yet a high level of viral genome diversity has been observed across populations. Viral diversity is produced through the combined effects of evolutionary pressure and selection on HSV-1 genomes. We are establishing methods to quantify viral population diversity and the effects of evolutionary pressure on HSV-1 genomes. We quantified inter-viral genomic recombination using a marker transfer assay to understand the impact of selection pressures, such as neuron replication, on viral diversity. The marker transfer assay utilized two unique, comparably fit viral isolates with genetically distant fluorescent protein expressing cassettes to co-infect cells. We scored viral progeny as either parent or recombinant based on marker expression as a percentile of the population. We adapted the marker transfer assay for both in vitro and in vivo HSV-1 infections. We utilized the HSV murine eye model to quantify the effect of spread and replication in neurons on viral populations. Understanding how HSV responds to antiviral selections during replication and spread will enhance current treatment for herpes infection and provide insight for vaccine developments.

Acknowledgements: Alix Herr (MSU Graduate Student) - Microbiology & Immunology, Irena Kochetkova (MSU Faculty Member) - Microbiology & Immunology