1138-92-353 **Cameron J Browne***, cambrowne@louisiana.edu. *Modeling immune escape in intra-host HIV and CTL networks.*

During HIV infection, an array of CTL immune response populations recognize specific epitopes (viral proteins) presented on the surface of infected cells to effectively mediate their killing. However HIV can rapidly evolve resistance to CTL attack at different epitopes, inducing a dynamic network of viral and immune response variants. We consider models for the network of virus and immune response populations. Our analysis provides insights on viral immune escape from multiple epitopes. In the "binary allele" setting, we prove that if the viral fitness costs for gaining resistance to each of n epitopes are equal, then the system of 2^n virus strains converges to a "perfectly nested network" with less than or equal to $n+1$ persistent virus strains. Overall, our results suggest that *immunodominance*, i.e. relative strength of immune response to an epitope, is the most important factor determining viral escape pathway of HIV against multiple CTL populations. To conclude, I briefly discuss ongoing collaborative work to connect the models with intra-host SIV/immune response data. (Received February 13, 2018)