**HOST-PATHOGEN INTERACTION NETWORK AMONG** *SULFOLOBUS ISLANDICUS* **AND ITS VIRUSES.** Jesse A. Black<sup>1</sup>, Maria A. Bautista<sup>2</sup> and Rachel J. Whitaker<sup>3</sup>, University of Illinois at Urbana-Champaign.

The viruses that infect hyperthermophilic archaea of the genus *Sulfolobus* and other organisms from the order Sulfolobales often show distinct, novel morphologies as compared to viruses that infect eukaryotes or bacteria [1]. The co-evolution and pathogenic interactions between these strains and their Archaeal hosts will provide great insight into the diversity and ancient origins of viral defense.

To further our understanding of evolutionary ecology and local adaptation between viruses and their microbial hosts in this system, we have focused our research on two geographically isolated populations of Sulfolobus islandicus from Yellowstone National Park. USA, and the Mutnovsky Volcano region of the Kamchatka peninsula, Russia [2]. Using a total of 31 strains of S. islandicus and viruses isolated from multiple hot springs in both YNP and Kamchatka, we have performed cross-infections of panels of 12 host and 19 virus strains. The patterns of host-virus interactions are determined experimentally by crossing standardized concentrations of both cells and viruses for each host-virus pair using traditional spot-on-lawn assays. The challenged lawns are monitored for plaque formation, size, as well as an increase in plaque turbidity over time that may indicate recovery of an immune host population, or presence of a resistant host minority.

To date, a network of host-pathogen interactions between genetically and geographically distinct S. islandicus hosts has not been described. Our data shows that some virus strains produce different infective patterns and plaque morphologies across different host strains. These patterns of host-virus interactions may be influenced by the varying sequences of CRISPR-Cas repeat-spacer arrays [3] across individual host strains on the panel, and these experimentally derived patterns can be compared to expected patterns of immunity as predicted by CRISPR-Cas sequence data. By piecing together networks of host-pathogen interactions both experimentally and through sequence data, we can resolve a clearer picture of the co-evolutionary process in this extreme environment and uncover novel mechanisms of virus defense.

**References:** [1] Zillig W et al. (1996). FEMS Microbiol Rev. 18(2-3):225-36. [2] Cadillo-Quiroz H et al. (2012) PLoS Biology. 10(2):e1001265. [3] Barrangou R et al. (2007) Science. (5819):1709-12.