

Kevin Sokoloski

Alphaviruses are mosquito-borne pathogens that represent a significant threat to public health due to their capacity to cause significant epidemics in immunologically naïve communities. Unfortunately, there are no safe, or clinically approved, vaccines or antiviral therapeutics available for the treatment of alphaviral disease. While disease symptomology varies amongst the members of the genus, the majority of the alphaviruses cause severe arthritis. Notably, Chikungunya virus (CHIKV) is capable of causing incapacitating multi-joint arthritis in otherwise healthy individuals. Approximately half of CHIKV clinical infections resolve following the acute infectious period; however, the remaining individuals experience ongoing persistent arthritis in the absence of infectious virus particles.

Recent studies have largely indicated that severe acute and persistent CHIKV infection is likely the result of a multifactorial disease process with genetic, environmental, and pathogen-based influences. Recent work assessing the role of host microbiota has indicated that pro-inflammatory dysbiosis enhances the pathology associated with auto-immune arthritis. Given the similarities between Rheumatoid arthritis and alphaviral arthritic disease, we postulate that host pro-inflammatory dysbiosis will enhance the severity and duration of CHIKV infection. To this end, we are using a mouse model of CHIKV infection to determine the role of pro-inflammatory dysbiosis on the acute and persistent stages of infection.