Date: 24 Oct 2017 (Tue)

Time: 1pm to 2pm

Venue: S4A L3 Seminar Rooms (A & B)

Dr. October Sessions received his PhD in Molecular Genetics and Microbiology in 2009 from Duke University, USA where he worked in the laboratory of Prof. Mariano Garcia-Blanco and identified host factors required for DENV propagation. Sessions continued his study of dengue virus as a postdoc in laboratories of Prof. Ooi Eng Eong and Prof. Duane Gubler at Duke-NUS Medical School where he characterized the host transcriptomic response to dengue viral infection and developed tools for pathogen discovery. In 2014, Sessions became a Research Assistant Professor at Duke-NUS where the current focus of his laboratory is deciphering the mechanisms that define the pathogenic potential of flaviviruses. His laboratory frequently utilizes high-throughput sequencing and more traditional molecular genetics and microbiological techniques to elucidate these mechanisms. These tools readily translate to the mechanistic analysis of other pathogens. In collaboration with local and international clinical partners, he is applying these tools to study the emergence of novel pathogens as well as known pathogens with novel clinical manifestations/associations.

SMALL CHANGES HAVE BIG EFFECTS: GENETIC PROFILING TO PREDICT PATHOGENICITY

Dengue virus (DENV) and Zika virus (ZIKV) are mosquito-borne viruses that belong to the Flavivirus genus of the Flaviviridae family. Both DENV and ZIKV have dramatically increased their geographic distribution in recent years with much of the tropical and subtropical world now hyperendemic for these viruses. In light of increasing global population growth, urbanization and international transportation of people and goods, these viruses are likely to continue their rapid expansion for the foreseeable future. Despite aggressive mosquito control and public awareness campaigns, Singapore continues to battle year-round transmission of all four DENV serotypes and more recently suffered its first autochthonous ZIKV outbreak.

While drug and vaccine development efforts are underway for these viruses, there are concerns that resistant strains will rapidly emerge. Indeed, the rapid spread of these viruses has greatly increased overall viral genetic diversity, some of which appears to be associated with increased epidemic potential. The mechanisms underlying viral fitness in epidemiological settings however, remain poorly defined. To address these deficiencies, we are employing a number of novel methodologies to examine both the viral and host determinants of pathogenesis. Our findings provide fresh insights into antiviral targets for these pathogens and could potentially serve as a template for the rapid identification of regions for therapeutic targeting in other pathogens.