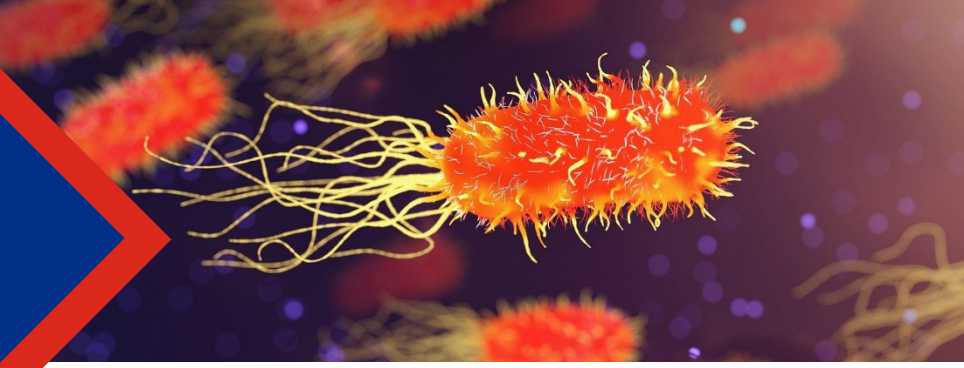
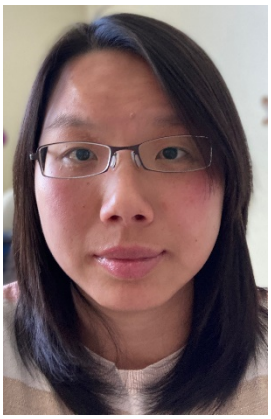
Infectious Diseases Labs

ID LABS



ID LABS SEMINAR SERIES



Dr Chen Hsiuyi

Integrative Biology Laboratory

The Salk Institute

Wednesday 29th September 2021

9am to 10am (SGT)

Join zoom meeting **here**

Meeting ID: 968 2308 8297

Passcode: 791724

Webinar is open to all

No registration required

Interrogating the variation in disease susceptibility to infectious diseases using genomic approaches

Upon infection, CD4+ T cells differentiate into various effector T cell subsets to help clear pathogens. Efficient pathogen clearance relies on properly differentiating into correct effector T cell subsets. Variation in the heterogeneity of pathogen-reactive effector T cell subsets has been shown to correlate with disease susceptibility. I hypothesize that variation in the heterogeneity of pathogen-reactive effector T cells could be resulted from variation in the genetics of the host factors. Genetic variants that are associated with disease susceptibility to flu or tuberculosis are identified close to GATA3 gene, which encodes an important transcription factor for T cell differentiation. I performed CRISPR/Cas9 tiling deletion screen to identify DNA elements that regulate GATA3 expression in naive CD4+ T cells. Interestingly, I found different combinations of DNA elements are used to allow GATA3 to be expressed in different levels to orchestrate the differentiation into different effector T cell subsets. My results showed the genetic variant, rs17432979, which is associated with influenza virus infection intersects with a strong GATA3 regulatory element in Th2 cells, indicating the risk allele of rs17432979 might skew the differentiation toward more Th2 cells and fewer Th1 subsets, making the host more susceptible to virus infection.

Dr Chen Hsiuyi am interested in understanding the variation in disease susceptibility. Starting with my PhD training, I took a systematic approach to dissect epigenetic regulation of dynamic gene expression in the laboratory of Oliver Rando at University of Massachusetts Medical School. After my PhD, I become interested in host response, so I aimed to investigate how the differentiation of naïve CD4+ T cells into various effector T cell subsets is controlled by a specific gene using genomic tools in the laboratory of Graham McVicker at Salk Institute for Biological Studies. The results help to understand the functions of the genetic variants that are associated with diseases susceptibility.