"Tuberculous granulomas exhibit microenvironment-specific pro- and anti-inflammatory cytokine expression and signaling"

Nearly one third of the world’s population is estimated to be infected with Mycobacterium tuberculosis, the bacterium that causes tuberculosis (TB). Fortunately, the human immune system can restrain bacterial replication and most individuals never experience more than asymptomatic subclinical (latent) TB. Some individuals fail to control bacterial replication, however, and there are more than nine million new cases of active TB and nearly 1.5 million TB-associated deaths annually. Granulomas are multicellular aggregates that form in response to M. tuberculosis infection, and are the characteristic lesions associated with TB. These structures provide a staging ground for anti-mycobacterial immune responses and when they work properly they can restrict bacterial growth but they also provide an intra-host niche for mycobacterial persistence. Understanding how granulomas function, and why they fail, may lead to important insights into TB and novel therapeutic strategies. My work uses granulomas from cynomolgus macaques, a nonhuman primate with human-like pathology, to examine how a balance of pro- and anti-inflammatory factors contribute to protection at the granuloma level. In my talk, I will discuss how granulomas are organized, and how multiple cell types in different intra-granuloma microenvironments communicate in ways that may promote or inhibit bacterial dissemination.