2018 Kathleen L. Miller Fellow

Perri Callaway

UC Berkeley PhD Candidate in Infectious Disease and Immunity Program, Feeney Lab at UCSF

Perri Callaway is a graduate student in UC Berkeley's Infectious Disease and Immunity Program. Before coming to UC Berkeley. Perri graduated from Barnard College with a degree in Biology and a minor in Spanish and Latin American Cultures. She is currently conducting her dissertation research in the lab of Dr. Margaret Feeney at UC San Francisco where she studies how a subset of gamma delta T cells contribute to the development of natural immunity to malaria.

Fellowship Proposal

Each year malaria infects ~200 million people and leads to approximately half of a million deaths worldwide. The vast majority of this morbidity and mortality is caused by Plasmodium falciparum in Sub-Saharan Africa in children under the age of five. While potent treatments exist, drug resistance is a constant concern. An effective vaccine is necessary to prevent disease and death caused by malaria. The development of a vaccine against malaria has been difficult in large part because development of sterilizing immunity is rare. Instead, individuals in endemic countries slowly develop "clinical immunity" after multiple episodes of malaria. When an individual is clinically immune they can still be parasitemic but no longer develop symptoms and eventually clear the parasites from their blood.

Perri's research will be part of a collaboration with the Infectious Diseases Research Collaboration (IDRC) in Uganda where she can access to nearly 10,000 peripheral blood mononuclear cell (PBMC) samples from a cohort of Ugandan children, which includes samples from a highly endemic region and a region with little malaria exposure. The goal of Perri's research is to understand how clinical immunity develops at the cellular level which will help determine immune correlates of protection necessary to the development of a malaria vaccine.

Her research specifically focuses on a subset of human gamma delta T cells that are intrinsically reactive to malaria called Vγ9Vδ2 T cells (Vδ2s). These cells have a T cell receptor (TCR) that recognizes phosphoantigens (pAgs), including the pAG HMBPP that in P. falciparum is produced as a byproduct of the non-mevalonate isoprenoid synthesis pathway and strongly activates Vδ2s2. Vδ2s make up the majority of gamma delta T cells circulating in adult peripheral blood and have been shown to be important in responding to malaria infection. She seeks to determine how Vδ2 cells integrate signaling through the TCR, CD16 and KIR receptors to activate effector functions in response to malaria infection and to determine how these responses change with malaria exposure.