UCDAVIS SCHOOL OF MEDICINE

MEDICAL MICROBIOLOGY & IMMUNOLOGY

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Medical Microbiology and Immunology

MMI 291 Seminar Series

Emerging Challenges in Microbiology and Immunology
Current Theme: Interdisciplinary Research

Sarah Stanley, Ph.D.

Assistant Professor
School of Public Health
University of California, Berkeley

"Metabolic regulation of M. tuberculosis infection"

Friday, April 27, 2018
Genome and Biomedical Sciences Facility, Auditorium Room 1005
12:10 PM – 1:00 PM

Research work: Dr. Stanley's research focuses on understanding the mechanistic basis for effective immune responses to M. tuberculosis, and on how M. tuberculosis is able to resist elimination by the immune system. Her recent findings shed light on how IFN-γ activates macrophages to control M. tuberculosis, integrating host metabolic shifts with specific effector responses. Furthermore, her findings have implications for the role of nitric oxide (NO) during infection, supporting the idea that NO functions as a signaling molecule during macrophage activation. She is also interested in macrophage/T cell interactions and vaccine development, and on developing host-targeted therapies that augment the immune response to infection.

Publication references:

Knight M, Braverman J, Asfaha K, Gronert K, Stanley SA. Lipid droplet formation in Mycobacterium tuberculosis infected macrophages requires IFN-γ/HIF-1α signaling and supports host defense. PLoS Pathogens, 2018. PMID: 29370315

Braverman J, Stanley SA. Nitric oxide modulates macrophage responses to M. tuberculosis infection through activation of HIF-1α and repression of NF-kB. J. Immunol, PMID 28754681

Braverman J, Sogi KM, Benjamin D, Nomura DK, Stanley SA. HIF-1α is an essential mediator of IFN-γ dependent immunity to Mycobacterium tuberculosis. J. Immunol 2016. PMID 27430178