S. Abir, J. Liu, D. Bhattacharya, M. Pauli, M. Rosenblum, P. Nanzani and W. Liao1 University of California Los Angeles, California, United States, 2 University of California San Francisco, San Francisco, California, United States and 3 Immunology, Oncology, Angen Research, South San Francisco, California, United States

Psoriasis is a chronic, inflammatory skin disease characterized by an aberrant immune response, and it affects 125 million people worldwide. However, the underlying pathways contributing to psoriasis pathogenesis have not been fully elucidated. This project utilizes single-cell transcriptomics of T cells from healthy and psoriatic skin in an effort to identify key biomarkers and pathways of psoriasis. T cells were clustered into subtypes and differential gene expression analysis was performed between lesional and healthy skin to identify psoriatic marker genes in each T cell subtype. Regulatory CD4+ T cells in psoriatic lesional skin were found to mediate IL-10 and IL-23 production and be enriched for CD1d tetramer binding, NF-kB signaling, and putrescine catalytic pathways. As a result, psoriatic Tregs may amplify several of the pathways behind psoriasis and drive inflammation via IL-32, a proinflammatory cytokine.


Recently, we have detected a subset of CD1a-restricted CD4+ T cells that specifically responds to acylated membrane lipid present in many gram-positive bacteria, including S. aureus. This lipid antigen, CD1a-LPG, binds to the CD1d molecule and is recognized by T cells in vitro. Furthermore, we have shown that these T cells are abundant in lesional psoriatic skin and that they respond in a dose-dependent manner to LPG. CD1a-LPG reactive T cell lines showed a predominantly Th2 cytokine profile, with abundant IL-4 and IL-13 release. Beyond cell-mediated signaling, NF-kB signaling, and putrescine catalytic pathways. As a result, psoriatic Tregs may amplify several of the pathways behind psoriasis and drive inflammation via IL-32, a proinflammatory cytokine.