by gene ontology (GO) and pathway enrichment analysis that were highly enriched in SLE T cells included mediators of adaptive responses and inflammation, and those regulating co-stimulation. By contrast, negative regulators of cell proliferation and function were found in the healthy control cluster, and diminished in SLE.

**Conclusions** Our data demonstrate altered transcriptional programs of lupus Thfh and Tcm cells, and therapeutic targets in disease. They also represent the first detailed transcriptional profiling, and single cell transcriptional profiling, of Thfh cells, the necessary and critical driver of humoral immunity in SLE.

**Acknowledgements** Supported in part by grants from the NIH/NIAMS (AR40072 and AR053495) and from the Alliance for Lupus Research (to JG).

**AI-23**

**MULTIPLEXED MECHANISTIC ASSAYS FOR CHARACTERISING SLE**

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10.1136/lupus-2016-000179.23

**Background** SLE is a complex disease with very few approved therapeutic options. Unique opportunities exist to characterise blood cells, tissues such as kidney and skin, urine, serum and plasma as part of ongoing longitudinal cohort studies such as Accelerating Medicines Partnership (AMP) and Autoimmunity Centres of Excellence (ACE), and investigator initiated or company-sponsored clinical trials.

**Materials and methods** SLE blood and tissue samples are being studied using a variety of single cell measurements as well as studies of biofluids. Several of these technologies are becoming firmly established in the SLE field, including single cell RNA Seq of blood and dissociated kidney using recently-developed methods in several consortia related to SLE and cancer; low input RNA-Seq of bulk purified cells; Assay of Transposase Accessible Chromatin (ATAC Seq); CyTOF and EpiCyTOF developed through the ACE consortium; transcript profiling using many methodologies; meta analysis of existing transcript profiling datasets; autoantibody profiling using autoantigen microarrays, and arrays composed of secreted factors such as cytokines and chemokines; multiplexed ion beam imaging (MIBI); and unpublished imaging methods such as CODEX.

**Results** An overview of multiplexed methods will be presented and will focus on efforts by the Stanford ACE and collaborating investigators to develop methods specifically for the study of SLE. Historical methods will be compared, and ACE datasets on human SLE, and mouse models of SLE characterised as part of ALR studies, will be described that demonstrate unique roles for interferons and STAT signalling in lupus.

**Conclusions** Big data analyses and multiplexed assays of samples derived from SLE patients, as well as patients with related autoimmune diseases, have tremendous potential and should be included in all clinical trials, with a goal to better understand pathogenesis and to identify novel therapeutic targets.

**Acknowledgements** Alliance for Lupus Research, NIAID, NIAMS, Henry Gustav Floren Trust, Baxter Foundation, and many patients with SLE who have provided samples.