that NKT cell deficiency in sarcoidosis results in abnormal monocyte activity.

**Methods** Twenty-five steroid-naïve non-smoking patients with histological confirmation of sarcoidosis were recruited from the Sarcoidosis-ILD service. Circulating monocyte numbers and phenotype were first characterised using multi-colour flow cytometry. We then isolated monocytes from blood using magnetic microbeads, examined cytokine production after LPS stimulation with intracellular cytokine FACs staining and ELISA, and using monocyte-NKT cell co-culture assays, questioned whether NKT cells affected these monocyte functions.

**Results** We found an increase in circulating CD14CD16 inflammatory monocytes in patients with sarcoidosis, and identified a population of interleukin 10 producing monocytes in patients and controls after LPS stimulation. Monocytes from sarcoidosis patients have reduced capacity to produce IL-10 after LPS stimulation compared to control (6.57% vs 11.71% of total monocytes, p<0.001, Abstract S109 figure IA); but addition of NKT cells improved this capacity (6.57% to 9.15%, p<0.001, Abstract S109 figure IB). We then questioned the role of IL10-producing monocytes and show (with mixed lymphocyte reaction and CFSE assays) that these cells suppress T cell proliferation (p<0.001, Abstract S109 figure IC).

**Conclusions** Our data show that sarcoidosis patients have increased inflammatory monocytes but a reduced IL-10-producing, T cell suppressing subset. NKT cells were able to interact with monocytes in vitro and increased IL-10 production by monocytes. These previously unrecognised findings, both in monocyte-NKT cross talk and in sarcoidosis immunobiology, suggest that one consequence of NKT deficiency in sarcoidosis is abnormal monocyte function with resultant loss in control of T cell proliferation. This reveals a potential new pathway of pathogenesis in sarcoidosis.

**Abstract S109 Figure 1**

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**S110**

**TARGETED DELETION OF Gq/11 IN SURFACTANT PROTEIN C-POSITIVE EPITHELIAL CELLS REDUCES TGFß ACTIVATION AND RESULTS IN INFLAMMATION AND ALVEOLAR AIRSPACE ENLARGEMENT**

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**Introduction** The concept of driving cellular apoptosis as a potential therapy for diseases characterised by inappropriate cellular persistence or proliferation is of widespread interest. We previously showed a death receptor ligand, TRAIL, accelerates neutrophil apoptosis without associated cell activation (J Immunol 170:1027–33) and other work revealed TRAIL-induced apoptosis of human lung fibroblasts. The aims of this project were to study the role of TRAIL in a bleomycin lung injury model in wild-type and TRAIL–/– mice and in patients with idiopathic pulmonary fibrosis (IPF).

**Methods** Mice received intratracheal bleomycin or saline control. Bronchoalveolar lavage (BAL) at 5, 7, 16 and 25 days was analysed by cytospin morphology and haemocytometer count for % neutrophils, % neutrophil apoptosis, total number of neutrophils and total number of apoptotic cells. Flow cytometry was also used to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed. Serum and lung tissue was analysed to analyse apoptosis. Collagen deposition in whole lung samples was analysed using a hydroxyproline assay. TRIAD expression and TUNEL positive events were also analysed.