stimulating factor (GM-CSF) significantly enhanced IFN-\(\lambda/3\) production by 2–5 fold. In pDC-NK cell co-cultures from SLE patients, IFN-\(\alpha/2b\) and GM-CSF increased the proportion of RNA-IC responding IFN-\(\lambda/1/3\) producing individuals from 9% to 36%. Hydroxychloroquine as well as an interleukin receptor 1 associated kinase 4 inhibitor (IRAK4i) significantly inhibited the RNA-IC-triggered IFN-\(\lambda/1/3\) production by pDCs and pDC-NK cell co-cultures by >90%.

Conclusions Type III IFN production in a small subset of pDCs can be triggered by RNA containing IC, enhanced by NK cells and several pro-inflammatory cytokines, and inhibited by blocking the TLR-MyD88 pathway, resembling the regulation of type I IFN. Thus, our results support a contributing role for both type I and type III IFN in SLE, which needs to be considered when targeting the IFN system in this disease.
with disease activity and nephritis. The population of Colombia has a mixture of European, indigenous American, and African ancestries. It is not known if Colombian patients have the same DN B cell distributions previously described and if they are associated with clinical manifestations.

**Methods**

40 SLE patients who met the 1982 ACR criteria and 17 Healthy Controls matched by age and gender were recruited from Medellin, Colombia. Cryopreserved peripheral lymphocytes were analyzed by multiparametric Flow Cytometry. DN cells were characterized using CD3, CD19, CD27, IgD, CD11c, and CD21 markers.

**Results**

SLE patients showed similar DN and DN2 distributions comparable to those described in African American patients. DN and DN2 cells were higher in patients with active disease, especially with severe activity. Patients with active nephritis and a history of nephritis had the same increase in DN and DN2. The evaluation of DN and DN2 in patients receiving treatment with mycophenolate and/or cyclophosphamide also showed this increase.

**Conclusions**

The alterations previously described in the frequency of DN and DN2 B cells are also found in Colombian patients. DN2 are generated through an extrafollicular differentiation pathway, which has an essential role in the autoantibodies production on SLE. These findings suggest a relevant contribution of an extrafollicular DN2 production on SLE pathophysiology in patients with mixed ancestry, as described before, for African American patients.

**P100 PROPORTIONS OF B CELL SUBSETS ARE ALTERED IN INCOMPLETE LUPUS ERYTHEMATOSUS PATIENTS AND CORRELATE WITH INTERFERON SCORE AND IGG LEVELS**

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**Background**

Incomplete systemic lupus erythematosus (iSLE) patients display symptoms typical for SLE but have insufficient criteria to fulfill the diagnosis. Biomarkers are needed to identify iSLE patients that will progress to SLE. Interferon (IFN) type I activation, B-cell activating factor (BAFF) and B-cell subset distortions play an important role in the pathogenesis of SLE. The aim of this cross-sectional study was to investigate whether B-cell subsets are altered in iSLE patients, and whether these alterations correlate with IFN scores and BAFF levels.

**Methods**

iSLE patients (n =34), SLE patients (n =41) with quiescent disease (SLEDAI ≤ 4) and healthy controls (HCs; n =22) were included. Proportions of B-cell subsets were measured with flow cytometry, IFN scores with RT-PCR and BAFF levels with ELISA.

**Results**

Proportions of age-associated B-cells were elevated in iSLE patients compared to HCs and correlated with IgG levels. Switched memory B-cells were elevated in SLE patients compared to healthy controls. Age-associated B-cells were elevated in iSLE and SLE patients compared to HCs. The dotted line in the left lower image represents the median IFN score of the iSLE group. The dotted line in the right lower image represents +2SD above the mean of the HC group, which was used as cut-off for BAFF positivity. *p< .05, **p< .01. HC = healthy controls, iSLE = incomplete SLE, ABC = age-associated B-cell, IFN = Interferon, BAFF = B-cell activating factor.