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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10.1136/lupus-2020-eurolupus.144

Background Incomplete systemic lupus erythematosus (iSLE) patients display symptoms typical for SLE but have insufficient criteria to fulfil 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 $\leq$ 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

Abstract P100 Figure 1 Proportions of B-cells subsets, and levels of B-cells activating factor (BAFF) in HCs, iSLE patients and SLE patients. Median and interquartile range are depicted for every group. 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

levels. In iSLE patients, IFN scores and BAFF levels were significantly increased compared to HCs. Also, IFN scores correlated with proportions of switched memory B-cells, plasma cells, IgG levels and correlated negatively with complement levels in iSLE patients.

Conclusions In this cross-sectional study, distortions in B-cell subsets were observed in iSLE patients and were correlated with IFN scores and IgG levels. Since these factors play an important role in the pathogenesis of SLE, iSLE patients with these distortions, high IFN scores, and high levels of IgG and BAFF may be at risk for progression to SLE.

Acknowledgement This study was funded by *ReumaNederland*

P101 HYDROXYCHLOROQUINE SUPPRESSES IFN-INDUCIBLE GENES AND BAFF IN PATIENTS WITH INCOMPLETE AND NEW ONSET SYSTEMIC LUPUS ERYTHEMATOSUS

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10.1136/lupus-2020-eurolupus.145

Background Hydroxychloroquine (HCQ) is the backbone of treatment in Systemic Lupus Erythematosus (SLE). It has been suggested that this drug can also delay the onset of SLE in patients with lupus symptoms, who do not meet the classification criteria yet. Interferon (IFN) type I is an early mediator in the pathogenesis of SLE. IFN-gamma induced protein 10 (IP-10) and B-cell activating factor (BAFF) are increased in SLE, but also prior to SLE diagnosis and correspond with disease activity. The purpose of this study is to analyze the

effects of HCQ on IFN-induced gene expression, serum IP-10 and BAFF levels.

Methods Patients with incomplete SLE (ANA titer $\geq 1:80$, symptoms <5 years, ≥ 1 objectified clinical ACR criterion), or new onset SLE (based on ACR 1997 or SLICC criteria) were included if there was a clinical indication to start HCQ treatment. Blood samples were taken at the start and after 16 weeks. Apart from NSAIDs, no other immunosuppressive drugs were initiated. The IFN-inducible genes MX1, IFI44L and LY6E were measured in whole blood by RT-PCR. An IFN score was determined by the normalized log (relative expression) of these 3 genes. Serum levels of IP-10 and BAFF were measured using ELISA. Differences between the time points were statistically assessed with Wilcoxon test.

Results In total, nine patients were included: six with iSLE and three with new onset SLE. The median SLEDAI was 4. After 16 weeks of treatment with HCQ, the relative expression of all three IFN-inducible genes decreased in 8 of 9 patients, and the IFN score decreased significantly ($p=0.012$). There was a trend towards lower IP-10 levels ($p=0.078$), and a significant decrease in BAFF-levels ($p=0.023$) after treatment with HCQ. The median SLEDAI decreased to 0. The one incomplete SLE patient who had no decrease in IFN score, developed SLE. (See figure 1.)

Conclusion After start of treatment with HCQ in patients with incomplete or new onset SLE, IFN score and BAFF levels decreased significantly, and there was a trend towards lower IP-10 levels. As these are early mediators in SLE, this might indicate that HCQ could delay disease progression in incomplete and new onset SLE. However, larger sample size and longer follow up is needed.

Acknowledgement This study was funded by *ReumaNederland*

Abstract P101 Figure 1 Plasma levels of B-cell activating factor, Interferon-gamma induced protein 10, and calculated IFN score before and 16 weeks after starting hydroxychloroquine. P-values are shown