and insulin resistance. Adiponectin, on the contrary, was independent of SLE activity, but had a favorable, although weak, effect on blood pressure and lipid profile.

PO.3.66 TLR9 PROTEIN LEVEL IS ASSOCIATED TO PROINFLAMMATORY CYTOKINE LEVEL OF IL10 AND INF1A IN SLE PATIENTS

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10.1136/lupus-2022-elm2022.95

Purpose The aim is to investigate the association among TLR7 and TLR9 serum levels with previous viral infections, disease activity and proinflammatory cytokine levels in SLE patients.
Methods Cross-sectional observational study in SLE patients (SLICC/ACR 2012 criteria) and healthy controls (HC). Previous infection data with RNA (HCV) and DNA virus (CMV, Epstein-Barr, Herpes simplex, Parvovirus B19 or HBV), disease activity and clinical data were collected. Biological samples of SLE patients and HC from the medical visit were supplied to the TLR7, TLR9, IL10 and INF1A determination by enzyme-linked immunoassay.
Results 94 SLE patients (91.5% female) with a mean age of 51 (13) years old and 35 HC (80% female) and 42 (12) years old were recruited. Mean age at diagnosis was 33 (14) years old and mean disease evolution was 19 (10) years. Mean SLEDAI index was 5.35 (4.58).

The 48.94% of patients reported almost one DNA virus infections, the 2.13% reported HCV infection, the 4.25% with HCV and DNA virus, and the 31.92% not reported any infection. HC had no history of acute (3 months) or lasting chronic infections with viruses of bacteria.

TLR7 and TLR9 did not correlate between them. TLR9 levels were significantly higher in SLE patients than HC (P<0,001). Even though TLR7 levels did not show any difference between both groups, an association with the age of individuals was observed (P<0,001).

No association among TLR7 or TLR9 levels with CRP, ESR, anti-dsDNA, ENAs or antiphospholipid antibodies was observed, and nor with disease activity, age at diagnosis and disease evolution time. In contrast, however, we reported low TLR7 levels in SLE patients and antiphospholipid syndrome in comparison to those without antiphospholipid syndrome (P=0,001).

High TLR9 levels were significantly associated to increased levels of IL10 and INF1A in SLE patients (P<0,001). TLR7 levels were not associated with INF1A levels but it is noticeable that there is a tendency to increase TLR7 levels in cases with increase of IL10 levels.
Conclusions TLR9 is increased in SLE patients in comparison to HC. TLR7 increases with age. No evidence of association between previous infections and TLR levels was found. Nor do we observe any difference in TLR level according to autoantibodies presence or disease activity, probably due to the long-term SLE evolution and a good control of the disease. There was, however, an association between high TLR9 levels and increase of IL10 and INF1A.

PO.3.67 MEASURING IFNA2 LEVELS BY A SINGLE-MOLECULE ARRAY IN CLINICAL PRACTICE OF CHILDHOOD-ONSET SLE PATIENTS DOES MATTER; RESULTS FROM A SINGLE CENTER LONGITUDINAL STUDY

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10.1136/lupus-2022-elm2022.96

Introduction Type-I interferon (IFN-I) pathway activation plays a pivotal role in the pathogenesis of SLE and has been proposed as biomarker for disease activity. IFN-I pathway activation can be measured by determining the expression of IFN-I stimulated genes or a so-called IFN signature. Ultrasensitive single-molecule array (Simoa) technology enables measurement of IFN protein concentrations at subfemtomolar concentrations. Parallel use of these measuring methods in longitudinal cohorts of childhood-onset SLE (cSLE) patients in relation to disease activity could help in translating the most relevant technique for use in clinical practice.
Objective To determine the association of serum IFNa2 levels and whole blood IFN-I stimulated gene expression with disease activity and study their potential to mark specific disease activity states in a longitudinal cohort of cSLE patients.
Methods Serum IFNa2 levels were measured in 338 samples from 48 cSLE patients and 67 healthy controls using an IFN-a2 Simoa assay (Quanterix) on an HD-X analyser. A 5 gene IFN-I signature was measured by RT-PCR in paired whole blood samples. Disease activity was assessed by the clinical SELENA-SLEDAI (cSLEDAI) and BILAG-2004. Low disease activity was defined by the Low Lupus Disease Activity State (LLDAS) and flares were characterized by the SELENA-SLEDAI flare index. Analysis was performed using linear mixed effect models.
Results A clear positive correlation was present between serum IFNa2 levels and the IFN-I gene signature (r=0.78, p<0.0001). Serum IFNa2 levels and the IFN-I gene signature showed the same significant negative trend in the first three years after diagnosis. In this timeframe, mean baseline serum IFNa2 levels decreased with 55.1% (delta 172 fg/mL, p<0.001) to a mean value of 164fg/mL, which was below the calculated threshold of 219.4 fg/mL. In the linear mixed model, serum IFNa2 levels were significantly associated with both the cSLEDAI and the BILAG-2004 (p<0.001 and p<0.01), while the IFN-I gene signature did not show this association (p=0.35 and p=0.23). Moreover, 69.7% of the time points in LLDAS had a serum IFNa2 level under the calculated threshold, while only 31.9% of the time points in LLDAS reached an IFN-I gene signature below the calculated threshold. Both techniques were equally capable of marking disease flares (79.2% above threshold vs 87.5% above threshold).

Lupus Sci Med: first published as 10.1136/lupus-2022-elm2022.95 on 27 September 2022. Downloaded from http://lupus.bmj.com/ on February 27, 2024 by guest. Protected by copyright.