stimulating factor (GM-CSF) significantly enhanced IFN-α/β production by 2–5 fold. In pDC-NK cell co-cultures from SLE patients, IFN-α/β and GM-CSF increased the proportion of RNA-IC responding IFN-α/β 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-α/β 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.

P97 DEFICIENCY OF MARGINAL-ZONE B CELLS IN PERIPHERAL BLOOD OF SLE PATIENTS IN CLINICAL REMISSION OR LOW DISEASE ACTIVITY STATE IN A LONG-TERM STUDY

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Purpose Deficiency of marginal-zone B cells was observed in peripheral blood (PB) of SLE pts in clinical remission or low disease activity (LDA). Goal of the prospective, comparative, long-term study is follow-up of this phenomenon.

Methods Forty five adult SLE (ACR/1982, updated 1997) pts in complete remission or LDA and 10 age- and sex-matched healthy controls (HC) were enrolled in „month 0“, and SLE also after twelve-months („month 12“) and 36-months („month 36“) period; overlap syndromes, infection, renal failure and monoclonal gammopathy in SLE were excluded. The DuraClone IM panel (Beckman Coulter) was used to identify CD19+CD27+IgM+ B cell subpopulation in PB samples by flow cytometry navios (Beckman Coulter) with software analysis using Kaluza version 1.2.; data obtained were expressed in relative% of PB lymphocytes and absolute values x10⁶/L, and processed using Medcalc-Statistical Software programme.

Results Significant differences (p =0.002 - <0.001) were obtained between absolute values of CD19+CD27+IgM+ B cells in HC (median 31.36, 95% CI 21.49 – 36.33) and SLE „month 0“ (median 13.17, 95% CI 7.87 – 17.09), SLE „month 12“ (median 10.36, 95% CI 7.24 – 16.04), and SLE „month 36“ (median 9.66, 95% CI 7.22 – 13.21), but not between values obtained in SLE „month 0“, „month 12“ and „month 36“ (p>0.05); not significant differences were found using analysis according to relative% of B cells under study (p>0.05).

Conclusion Data obtained demonstrated a long-term deficiency of marginal-zone B cells in PB of SLE pts in complete remis-sion or LDA; susceptibility to infection should be supposed, but further studies are necessary.

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