Grant/research support from: Medimmune, Vanessa L. Bryant Grant/research support from: CSL

**References:**


**Methods:** Peripheral blood samples from 39 patients suffering from SLE (mean±SD; age 43±13 years, 87.2% females, disease duration 11.1±7 years) were drawn over 2 years. All SLE patients were in remission or low disease activity (medianSLE, SLEDAI 2 of 2.0±1.5). B-cells were characterized using CD19, CD20, CD5, CD27 antibodies and were grouped in naïve (IgD<sup>+</sup>CD27<sup>−</sup>), non-switched memory (IgD<sup>−</sup>CD27<sup>−</sup>), memory (IgD<sup>−</sup>CD27<sup>+</sup>), B1 (CD5<sup>+</sup>CD27<sup>−</sup>) and MBL-like (CD5<sup>+</sup>CD27<sup>−</sup>) B-cells. A quantitative flow cytometric bead-based assay (Quantibrite PE kit from Becton Dickinson) was used for the estimation of CD19 antibodies bound per cell. Further, CD38 and CD69 antibodies were used to characterize the B-cell subsets. All cytometric measurements were performed using a standardized BD LSR Fortessa platform. After 3 years of follow-up, patients’ data about disease activity and current medication were obtained.

**Results:** 22 SLE patients were treated with hydroxychloroquine (85.8%) and 19 patients received mycophenolate mofetil (MMF; n=14; 54.6%) or azathioprine (AZA; n=5; 19.5%). 5 patients were treated with other DMARDs. Independently of hydroxychloroquine and/or MMF, no significant differences were seen in naïve, non-switched memory, post-switched memory, plasma blasts, B1- or MBL-like B-cells. Patients treated with AZA had significantly lower naïve B-cells (means±SD; 39.3±6.7 vs. 73.1±19.3 %; p = 0.028), but had significantly higher IgD<sup>+</sup>switched B-cells (31.2±9.1 vs. 12.5 ±9.2 %; p = 0.029, respectively) compared with no AZA-treatment. Interestingly, activated B-cells (5.9±1.6 vs. 18.1±1.1 %; p = 0.009) were significantly higher in AZA-treated. After 3 years of follow-up, almost all patients were in remission (medianSLE, SLEDAI of 2.0±2.0), except of 3 patients with a SLEDAI of ≥ 6. Interestingly, those patients had at baseline, statistically higher naïve B-cells (p = 0.041) and lower B1-like B-cells (p <0.020) compared with patients with low disease activity.

**Conclusion:** Our results suggest that independently of hydroxychloroquine and/or MMF treatment, all patients with low disease activity had similar normal B-cell subsets. Interestingly, in the small group of patients who were treated with AZA, a reduced regeneration of B-cells was shown. Patients with higher disease and high naïve B-cells showed an increased disease activity after three years.

**Acknowledgments:** The research was performed in “CBmed” and funded by the Austrian Federal Government within the COMET K1 Centre Programme, Lano, Steiermark and Land Wien.

**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2020-eular.4412

**Objectives:** To study the effect of immunosuppressive medication on the type-I IFN signature in these patients.

**Methods:** Samples from healthy donors and SLE patients were stimulated with or without CpG-A or Imiquimod (IQ) or transfected with the cGAS agonist G3-YSD to induce IFN-α2b. Aspirin, diclofenac, Hydroxychloroquine and more recently Aspirin on IFN activation and opens avenues for the design of novel therapeutic strategies.

**Results:** A total of 19 DEGs were identified. The enriched functions and pathway enrichment analyses were processed. The protein-protein interaction network (PPI) was constructed, followed by calculation of topological characteristics and sub-module analysis in order to obtain hub DEGs. The expression of some hub genes was verified by Real-Time PCR in 24 SLE patients.

**Results:** A total of 19 DEGs were identified. The enriched functions and pathways of the DEG enriched in toll-like receptor signaling pathway, Salmonella infection, Viral protein interaction with cytokine and cytokine receptor, NF-kappa B signaling pathway and Human cytomegalovirus infection. Seven hub genes (TNF, IL1B, CXCL8, CCL3, CCL4, CCL3L1, CCL4L1) were identified and pathway enrichment analysis revealed that these genes were mainly enriched in toll-like receptor signaling pathway. The relative expression of the CXCL8 mRNA in SLE B-lymphocytes in patients with pSS associated thrombocytopenia was higher than that in the pSS without thrombocytopenia group. No differences were observed in the IL-1β or TNFα expression between these two groups.

**Conclusion:** PSS associated thrombocytopenia might be a subset characterized by a systemic inflammatory state. The identification of upregulated genes involved in thrombocytopenia of pSS provides insight in disease pathogenesis and opens avenues for the design of novel therapeutic strategies.

**References:**