BACKGROUND: Systemic Lupus Erythematosus (SLE) is an autoimmune disorder characterised by polyclonal B cell activation, production of dsDNA-autoantibodies and cytokines. Subsets of B cells play a central role in SLE-pathogenesis. The inflammatory milieu is characterised by the accumulation of adenosine, which confers immunosuppressive effects.

OBJECTIVES: In SLE, the role of CD73, an enzyme involved in the extracellular generation of adenosine from ATP, is not well characterised. This study aimed to characterise expression of CD73 B cell subsets of SLE-patients as compared to healthy controls (HC).

METHODS: B cell subsets were characterised from peripheral blood of 23 SLE patients attending the outpatient clinic at the Rheumatology Unit of University Hospital Düsseldorf and of 15 HC by FACS. All patients fulfilled the revised SLE-criteria of ACR and were randomly collected in clinical remission state (SLEDAI ≤11.9).

RESULTS: By comparison of B cell subsets between SLE and HC, CD38 was dominantly expressed by SLE-patients (SLE 74.2%±12.9% vs. HC 64.2%±12.2%; p(MWU)=0.018). Furthermore, SLE-patients showed an increase in CD19 -IgD-CD27-CD38 high plasmablasts (SLE 2.1%±3.4% vs HC 0.4%±0.4%; p(MWU)=0.001). Furthermore, SLE-plasmablasts showed decreased CD73 expression as compared to HC(SLE 2.1%±1.9% vs HC 3.5%±2.2%; p (MWU)=0.096). SLE-B cells revealed a trend towards an augmented CD38-highCD138 plasmablast fraction (SLE 0.40%±0.5% vs HC 0.08%±0.7%; p<0.01), yet without any difference in CD73 expression. The other hand, exhausted-memory B cell fraction (CD19 +IgD-CD27-CD21-CD138-) showed an increased CD73 expression in SLE (SLE 13.7%±9.2% vs HC 6.2%±5.4%; p=0.004).

CONCLUSIONS: Our study confirms CD38-plasmablasts as being increased in peripheral blood from SLE patients as compared to HC. Furthermore, the data reveal a deficiency for CD73 on SLE plasmablasts, which suggests a decreased anti-inflammatory capacity of SLE plasmablasts as compared to HC, supporting the notion of a disturbed adenosine axis in SLE. On the other hand, the enlarged CD73 -exhausted memory pool in SLE could point to an accelerated flow of CD73 +B cells into an exhausted B cell fraction. These findings support the hypothesis of dysregulation of the adenosine axis in SLE even in inactive SLE patients.

REFERENCES:

Disclosure of Interest: None declared