vs 9, IQR 5.5-14.5), median prednisone daily dosage (7.1mg, IQR 5-11.9 versus 11mg, IQR 7.6-17.5) and median SLEDAI-2K (8, IQR 6-9.75 vs 8, IQR 5-10). Differences in concomitant medications or clinical manifestations are reported in Figure 1A.

Among the group that received the standard dose, 7 patients discontinued the treatment (20.6%), specifically 2 renal flares and one adverse event occurred. Whereas, in the reduced dose group, 2 discontinuations occurred (14.3%) due to a pregnancy and a renal flare. Noteworthy, the patient with the renal flare resumed subcutaneous belimumab 12 months after the flare. Finally, the disease activity was analysed in the 12 months after the first drug reduction (Figure 1B-E), with no sign of worsening of evaluated parameters (Figure 1B-E), but with a significant reduction of steroids and SLEDAI-2K, 6 months after tapering.

Conclusion: In our single retrospective cohort, we have demonstrated that belimumab tapering seems to be safe and not associated with a worsening disease activity over the following months. However, further studies are required to identify eligibility criteria and to determine the optimal dose-tapering strategy both for the intravenous and the subcutaneous formulations.

Figure 1.

Disclosure of Interests: None declared
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POS0108 PLASMA MITOCHONDRIAL DNA AS A BIOMARKER IN DIAGNOSIS AND FOLLOW-UP OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: A fundamental role of mitochondria in systemic lupus erythematosus (SLE) was recently demonstrated (1). In brief, mitochondrial ROS participate in the formation of neutrophil extracellular traps (NETs) (2), while extrusion of cell-free mitochondria and highly oxidized interferogenic mtDNA causes disease in an animal model of SLE (3-5).

Objectives: The diagnostic and prognostic value of cell-free DNA in SLE is still unknown. The aim of the present study was therefore to examine the clinical utility of cell-free DNA quantification as a non-invasive biomarker in SLE.

Methods: Total DNA was isolated from platelet-free plasma samples of healthy individuals (HC) and consecutive SLE patients. Plasma and clinical data were collected at baseline and follow-up. Copy numbers were quantified by qPCR for mitochondrial (mt) DNA (ATP-6 gene) and nuclear (n) DNA (GAPDH gene).

Results: Fifty-six HC (median age 48.3 ± 13.5, 64% female) and 103 SLE patients (median age 46.8 ± 15.8, 99% female, mean SLEDAI: 3 ± 4) were available for analysis.

mtDNA levels were significantly elevated in SLE plasma (1.3x10⁸ copies/ml plasma, 95% CI: 7.3x10⁷ to 1.7 x10⁸), compared to HC plasma (8.6x10⁶ copies/ml plasma, 95% CI: 6.9x10⁶ to 1.0x10⁷, p<0.0001). nDNA levels in contrast did not differ between SLE (8.3x10⁶ copies/ml plasma, 95%CI: 5.9x10⁶ to 1.4 x10⁷) and HC (1.0x10⁷ copies/ml plasma, 95%CI: 2.0x10⁶ to 1.5 x10⁷, p=0.61). Receiver operating characteristic curve analysis showed that a cut-off value of 1.9x10⁷ mtDNA copy numbers differentiated between SLE and HC with 87.4% sensitivity, 94.6% specificity and an AUC of 0.95 (Figure 1a).

mtDNA levels correlated with the SLE Disease Activity Index 2000 (SLEDAI-2K) (r=0.29, p=0.0026), less so also nDNA copy numbers (r=0.24, p=0.014). There was no association of mtDNA elevation with any particular type of SLE organ involvement and no correlation between mtDNA copy numbers in SLE plasma and dsDNA antibody levels.

Follow-up data were available for 32 SLE patients (median follow-up 4.0 months, IQR: 4.0). delta mtDNA-levels robustly correlated with changes in SLEDAI-2K (r=0.51, p=0.0012, Figure 1b).

Conclusion: The quantification of cell free mtDNA, but not nDNA copy numbers allows a sensitive and specific distinction between healthy individuals and patients with SLE. mtDNA levels correlate cross sectionally with disease activity in SLE patients and within individual SLE patients longitudinally with the SLEDAI. Plasma mtDNA quantification may therefore aid in the diagnosis of SLE and in monitoring SLE activity.

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Declaration of conflict of interest: UW is coinventor of patents owned by Freiburg University; NV is coinventor of patents owned by Freiburg University.

Figure 1. (a) Receiver operating characteristic curve for mtDNA plasma concentrations to discriminate between HC and SLE patients. AUC: area under the curve. (b) Changes in plasma mtDNA levels in SLE patients correlate with the evolution of disease activity at follow-up.

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POS0109 IDENTIFICATION OF PRIMARY SJOGREN'S SYNDROME SUBTYPES BY MACHINE LEARNING

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Background: Sjogren's syndrome (pSS) is a chronic, progressive, and systematic autoimmune disease characterized by lymphocytic infiltration of exocrine glands ^{1,2}. Sicca symptoms and abnormal fatigue are the main clinical presentation, but those symptoms are non-specific to patients, which lead to delayed diagnosis ^{1,3}. The heterogeneous of clinical manifestation raise challenges regarding diagnosis and therapy in pSS, thus it's necessary for us to sub-classify pSS.

Objectives: To explore new biomarkers for diagnosis and subtypes of pSS based on Machine Learning Primary.

Methods: All microarray raw data (CEL files) were screened and downloaded from Gene Expression Omnibus (GEO). Meta-analysis to identify the consistent DEGs by MetaOmics. Weighted gene co-expression network analysis (WGCNA) was used to the modules related to SS for further analysis. Subclasses were computed using a consensus Non-negative Matrix Factorization (NMF) clustering method. Immune cell infiltration was used to evaluate the expression of immune cells and obtain various immune cell proportions from samples. P value < 0.05 were considered statistically significant. All the analyses were conducted under R environment (version 4.03).

Results: A total of 3715 consistent DEGs were identified from the four datasets, including 1748 up-regulated and 1967 down-regulated genes. Four