ISGs (MX1, IFI44L, LY6E) was analysed with qPCR. ISG expression was summarized as IFN score using the following formula: $\sum$(fold induction($ISG_{subject}$)-mean(fold induction (ISG_{Hc}))/SD(fold induction(ISG_{HC}))).

Results PBMC TLR7 and TLR9 gene expression was similar between HCs, iSLE and SLE patients (figure 1A-B). Upon stimulation, TLR9 mediated ISG expression was significantly lower in iSLE patients compared to HCs ($p<0.001$), whereas no significant difference was observed between HCs and SLE patients (figure 1D). In addition, there was a trend for TLR7 mediated ISG expression to be lower in iSLE and SLE patients compared to HCs ($p=0.059$; figure 1C). TLR9 mediated ISG expression was significantly higher after stimulation in iSLE patients treated with hydroxychloroquine (HCQ) than in patients who were not treated with HCQ ($p=0.014$; figure 1F). We did not find an association between TLR7 and TLR9 mediated ISG expression and disease activity, autoantibodies or complement levels.

Conclusions TLR7 and TLR9 gene expression in PBMCs was similar between HCs, iSLE and SLE patients. TLR7 and TLR9 mediated ISG response was reduced in PBMCs from iSLE and SLE patients, which might reflect desensitisation due to increased overall TLR7 and TLR9 stimulation. Interestingly, HCQ treatment in iSLE was associated with a higher ISG response in PBMCs upon stimulation, indicating that HCQ might restore normal TLR7 and TLR9 response.

P12 SENSITIVITY DIFFERENCES IN ELISA VERSUS ELIA FOR DETECTING ANTI-DSDNA ANTIBODIES: AWARENESS FOR THE CLINICIAN

Anna Kohn[1], Maudi de Jong[1], Merlijn van den Berg[1], Arjan Kwakernaak[2], Ester van Leeuwen[3], Arianne Brandsma[3], Dieneke Schonenberg-Meinema[1]. *[1]Dept. of Pediatric Immunology, Rheumatology and Infectious Diseases. Emma Children's Hospital, Amsterdam UMC, University of Amsterdam, The Netherlands*; *[2]Dept. of Internal Medicine, Dept. of Clinical Immunology, Allergy and Nephrology, Amsterdam UMC, University of Amsterdam, The Netherlands*; *[3]Laboratory of Medical Immunology, Amsterdam UMC, University of Amsterdam, The Netherlands*

10.1136/lupus-2024-el.66

Objective Heterogeneity among clinical presentation can cause difficulties when diagnosing SLE. One of the most specific immunologic criteria for this disease are anti-dsDNA antibodies. However, to detect these autoantibodies, different test methods are offered. This study will compare sensitivity of ELISA and EliA by synchronous testing in SLE patients.

Methods Between March and August 2022 samples from (suspected) SLE patients, undergoing anti-dsDNA testing, were simultaneously measured by dsDNA/nucleosome ELISA (positive IU/ml >100) and dsDNA EliA (positive > 15 IU/ml). SLICC 2012 classification criteria were used to include SLE patients. Clinical data including clinical SLEDAI-2K were retrospectively retrieved from medical records.

Abstract P12 Table 1 Demographic characteristics

		N (%)	Median (IQR)
Sex	Male	15 (10.3%)	
	Female	131 (89.7%)	
Age in years (at time of testing)			38 (25–49)
Disease duration in months			131 (31–205)
Treatment (at time of testing)	No	17 (11.6%)	
	Yes	129 (88.4%)	

Abstract P12 Table 2 EliA vs. ELISA test results and median SLEDAI (IQR) per subgroup

		ELISA +	ELISA -	Total
EliA +	N	78	14	92
	Median SLEDAI (IQR)	7.5 (2.0–10.0)	0.5 (0.0–2.0)	
EliA -	N	51	3	54
	Median SLEDAI (IQR)	7.0 (5.0–12.0)	0 (0–0)	
Total		129	17	146

Results Out of 163 tested patients, 143 SLE patients were eligible for study analysis. Demographic characteristics are summarized in table 1. Sensitivity was 88.4% for ELISA versus 63.0% in EliA (McNemar. $p<0.001$). Of 13/14 patients with EliA+/ELISA- discrepancy (median SLEDAI 0.5), ELISA had been positive in the past. Median SLEDAI was higher for patients with EliA-/ELISA+ discrepancy (see table 2).

Conclusions In our SLE cohort, there was a striking difference in sensitivity between anti-dsDNA test results (mainly EliA-/ELISA+ discrepancy) despite active disease in these patients. We hypothesize that this might be explained by the presence of anti-nucleosome antibodies, which can be detected by dsDNA/nucleosome ELISA or ENA-immunoblot. Clinicians should be aware of lower sensitivity (higher chance of false negatives) in EliA when interpreting anti-dsDNA test results.

P13 ACQUIRED FICOLIN-3 DEFICIENCY IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

[1]Linnea Lindelöf, [2]Solbritt Rantapää-Dahlqvist, [3]Christian Lundtoft, [3]Johanna K Sandling, [3]Dag Leonard, [3]Ahmed Sayadi, [3]Lars Rönnblom, [4]Helena Enocsson, [4]Christopher Sjöwall, [5]Andreas Jönsen, [5]Anders A Bengtsson, [6]Mun-Gwan Hong, [7]Lina-Marcela Diaz-Gallo, [8]Matteo Bianchi, [8]Sergey V Kozyrev, [8,9]Kerstin Lindblad-Toh, the DISSECT Consortium, the ImmunoArray Consortium, [1,10]Kristina Nilsson Ekdahl, [1]Bo Nilsson, [7]Iva Gunnarsson, [7]Elisabet Svenungsson, [1]Oskar Eriksson. *[1]Dept. of Immunology, Genetics and Pathology, Uppsala University, Uppsala, Sweden*; *[2]Dept. of Public Health and Clinical Medicine/Rheumatology, Umeå University, Umeå, Sweden*; *[3]Dept. of Medical Sciences, Rheumatology, Uppsala University, Uppsala, Sweden*; *[4]Dept. of Biomedical and Clinical Sciences, Linköping University, Linköping, Sweden*; *[5]Dept. of Clinical Sciences Lund, Rheumatology, Lund University, Skåne University Hospital, Lund, Sweden*; *[6]National Bioinformatics Infrastructure Sweden, Science for Life Laboratory, Dept. of Biochemistry and Biophysics, Stockholm University, Stockholm, Sweden*; *[7]Division of Rheumatology, Dept. of Medicine, Solna, Karolinska Institutet, Karolinska University Hospital, Stockholm, Sweden*; *[8]Science for Life Laboratory, Dept. of Medical Biochemistry and Microbiology, Uppsala University, Uppsala, Sweden*; *[9]Broad Institute of MIT and Harvard, Cambridge, MA, USA*; *[10]Linnaeus Center for Biomaterials Chemistry, Linnaeus University, Kalmar, Sweden*

10.1136/lupus-2024-el.67

Objective Ficolin-3 is the main initiator of the lectin pathway of complement in humans. Case reports of ficolin-3 deficient patients have suggested that ficolin-3 deficiency may be enriched in patients with Systemic Lupus Erythematosus (SLE), a systemic autoimmune disease where complement plays an important role. Therefore, this study aimed to investigate the activity levels of ficolin-3 and to identify potential ficolin-3 deficient individuals in two Swedish SLE cohorts.

Methods Serum samples from SLE patients (n=786) and matched controls (n=566) were collected from the Karolinska Institute and Umeå University Hospital. The ficolin-3 activity was measured by an in-house developed functional ELISA