with a pooled normal human serum sample as a reference. Serial samples were analyzed for ficolin-3 deficient patients when available. Sequencing data were analyzed for *FCN3* frame-shift mutation rs532781899 and other potential loss-of-function (LoF) variants.

Results This screening revealed that the ficolin-3 activity varies largely in patients with SLE. The activity levels also show that SLE patients have elevated ficolin-3 activity compared to the control group ($p<0.0001$). Four SLE patients were determined to be ficolin-3 deficient. For two of these patients, the ficolin-3 activity fluctuated over time, where one even had normal levels at the time of diagnosis with a subsequent depletion over time, indicating an acquired deficiency. For deficient patients, no or very low ficolin-3 protein levels and no lectin pathway-dependent complement activation could be detected. Autoantibodies against ficolin-3 were only detected in serum from one of the deficient patients. No patients were homozygous for the frame-shift mutation rs532781899, whereas 10 patients were determined to be heterozygous carriers. These heterozygous patients displayed lower levels of ficolin-3 activity, but did not include the deficient patients. Additional possible LoF variants were analyzed, but none were enriched in either patients or controls.

Conclusions Contrary to the classical pathway of the complement system, we show no evidence for an association between genetic ficolin-3 deficiency and SLE susceptibility. Instead, acquired ficolin-3 deficiency was observed in a subgroup of SLE patients, possibly due to a potent activation of the lectin pathway that depleted ficolin-3 serum levels in these individuals.

P14 HIGH FREQUENCY OF ITP RELATED AUTO-ANTIBODIES IN PATIENTS WITH ACTIVE SLE

[1]Anders Bengtsson, [2,3]John W Semple, [4]Leendert Porcelijn, [5]Rick Kapur, [1]Petrus Linge. *[1]Section of Rheumatology, Dept. of Clinical Sciences Lund, Faculty of Medicine, Lund University, Lund, Skåne, Sweden; [2]Division of Hematology and Transfusion Medicine, Lund University, Lund, Sweden; [3]Clinical Immunology and Transfusion Medicine, Office of Medical Services, Region Skåne, Lund, Sweden; [4]Dept. of Immunohematology Diagnostics, Sanquin Diagnostic Services, Amsterdam, The Netherlands; [5]Sanquin Research, Dept. of Experimental Immunohematology and Landsteiner Laboratory, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands*

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Objective Systemic Lupus Erythematosus (SLE) is characterized by arthritis, rash, glomerulonephritis, and hematologic manifestations, including secondary immune mediated thrombocytopenia (ITP). Presence of ITP type, platelet specific glycoprotein antibodies have been detected in SLE, but their exact role and frequency is not known although thrombocytopenia can be of prognostic importance. The objective of this study was to determine the frequency of platelet specific antibodies in patients with SLE and if the presence of these antibodies is associated with disease activity.

Methods We collected serum samples from 74 patients with SLE (≥ 4 American College of Rheumatology 1982 criteria) during high (H) and lower (L) SLE Disease activity index 2000 (SLEDAI-2K) score (median SLEDAI-2K, H = 8.5 and L = 0.5). Mean age at the first sampling was 43,6 and the second 48,6 years. Antibodies towards platelet glycoproteins (GP) IIb/IIIa, GP V and GP Ib/IX were detected in H and L serum samples, using the direct monoclonal antibody immobilization of platelet antigens assay (MAIPA). We also analyzed the presence of antiphospholipid criteria and non-criteria antibodies: Anti-CL (IgG and IgM) Anti-B2GP1 (IgG and IgM) Anti-PS/PT (IgG and IgM) Anti-AnnexinV (IgG and IgM), using ELISA.

Results During H and L SLEDAI-2K we detected anti-GP IIb/IIIa antibodies in 29.7% vs 28.4%; anti-GP V antibodies in 48.6% vs 27.0% and anti-GP Ib/X antibodies in 58.1% vs 31.1%. The anti-GP V and anti-GP Ib/X levels were significantly higher in SLEDAI-2K H vs L and positively correlated with SLEDAI-2K score ($r=0,322$, $p<0,0001$ and $r=0,265$, $p=0,001$) using spearman correlation. During active disease, 66% had at least one positive GP-antibody, compared to 44% in the less active disease. All antiphospholipid antibodies of IgG type had significantly higher levels during SLEDAI-2K H. In total, 45% of patients had at least one positive antiphospholipid antibody during SLEDAI-2K H and 26% during L.

Conclusion This study shows that platelet autoantibodies are prevalent in SLE and their increased presence may be modulated by treatment and/or mechanisms regulating disease activity. Future studies will assess the functional contribution from these antibodies on platelet activation, thrombocytopenia and other aspects of SLE pathophysiology.

P15 OXIDATIVE STRESS IN INCOMPLETE SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS COMPARED TO SYSTEMIC LUPUS ERYTHEMATOSUS AND HEALTHY CONTROLS

[1]Lu Liu, [1]Svenja Henning, [1]Karina de Leeuw, [1]Berber Doornbos-van der Meer, [2]Harry van Goor, [1]Johanna Westra. *[1]Dept.of Rheumatology and Clinical Immunology, University Medical Centre Groningen, Groningen, The Netherlands; [2]Dept. Pathology and Medical Biology, University Medical Centre Groningen, Groningen, The Netherlands*

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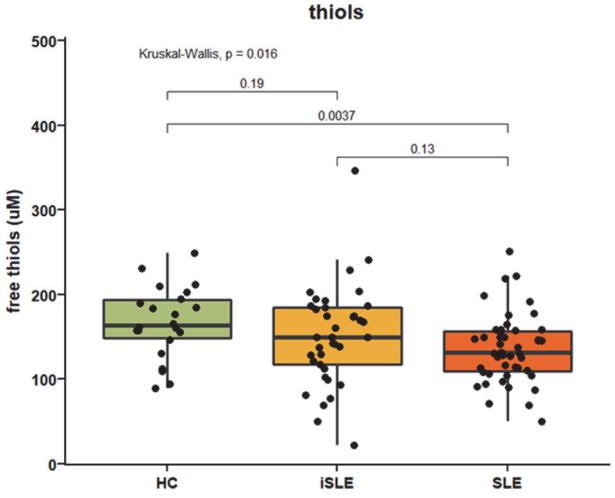
Introduction Systemic lupus erythematosus (SLE) is a complex and heterogeneous autoimmune disease. Patients who present with clinical symptoms and immunologic abnormalities suggestive of SLE but do not meet the classification criteria are diagnosed with incomplete SLE (iSLE).[1] In SLE, chronic inflammation may lead to excessive production of reactive oxygen species (ROS), inducing oxidative stress. Serum free thiols reliably reflect systemic oxidative stress since they are



Abstract P15 Figure 1 Serum free thiol levels in iSLE and SLE patients and healthy controls. (i)SLE, (incomplete) systemic lupus erythematosus, HC, healthy controls