

1.8 pg/mL) levels than patients with rheumatoid arthritis (RA) (1.8 ± 1.01 pg/mL and 3.2 ± 1.6 pg/mL, respectively, $p < 0.001$ for both) or HCs (2.2 ± 1.64 pg/mL and 2.2 ± 1.7 pg/mL, respectively, $p < 0.001$ for both). The levels of salivary IGHG3 correlated with erythrocyte sedimentation rate ($r = 0.26$, $p = 0.01$), anti-dsDNA antibody levels ($r = 0.25$, $p = 0.01$), and nephritis ($r = 0.28$, $p = 0.01$).

Conclusions The levels of salivary IGHG3 and lactoferrin were higher in patients with SLE. In addition, salivary IGHG3 levels correlated with SLE disease activity markers.

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P41 **PREDICTING AUTOIMMUNE CONNECTIVE TISSUE DISEASES: THREE YEAR FOLLOW UP OF AN AT RISK COHORT IDENTIFIES LATE PROGRESSION AND BIOMARKERS TO PREDICT NEED FOR THERAPY**

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Background Autoimmune connective tissue diseases (AI-CTDs: SLE, pSS, IIM, Scleroderma, MCTD) are preceded by asymptomatic ANA positivity. We previously recruited a cohort of new ANA-positive referrals without AI-CTD. 17% met criteria by AI-CTD at 12 months, and this was predicted an IFN Score. This study includes 3 year follow up of that cohort with more detailed analysis of the 'non-progressor' group and predictors.

Methods Patients were recruited if they had: (i) ANA; (ii) did not meet criteria for AI-CTD; (iii) symptoms less than 12 months. Diagnostic criteria for AI-CTDs and therapies were assessed at baseline then 12-monthly for 3 years. We categorised progression as:

1. Absolute-non-progressors (no clinical criteria at all time points: 0–36 months)
2. Undifferentiated-CTD (≥ 1 clinical criterion at baseline and/or at follow up but not meeting criteria)
3. Year-1-progressor (meeting criteria for AI-CTD within 12 months)
4. Late-progressor (meeting criteria for AI-CTD later than 12 months)

Interferon Score A and Interferon Score B were measured at baseline using taqman as previously described.¹

Results 3-year follow up was available in 146/150. Proportions in the above categories were: Absolute-non-progressors: 33/146 (23%); Undifferentiated-CTD: 86/146 (59%); Year-1-progressors: 21/146 (14%); Late-progressors: 5/146 (3%). No patient progressed or required immunosuppression after 2 years. 6/86 patients with Undifferentiated-CTD received an immunosuppressant. The present work therefore defines a larger group of 32/146 (22%) with clinically significant disease including 21 Year-1-progressors, 5 late-progressors, and 6 undifferentiated-CTD who needed an immunosuppressant.

Clinical features had limited utility in predicting these outcomes. Of 31 patients with no clinical criteria at baseline, 1 progressed to meet criteria within 1 year, 2 progressed at 1–2 years, 3 were prescribed hydroxychloroquine and 1 was prescribed an immunosuppressant. The 108 patients with at least 1 criterion at baseline had the highest risk: 20 progressed to meet criteria within 1 year, 2 progressed at 1–2 years, the others all had U-CTD. 35 were prescribed hydroxychloroquine and 13 were prescribed an immunosuppressant. There was also no association between ENA, C3 or C4 and clinical outcome.

The association between interferon scores and progression was stronger when comparing Year 1 progressors with absolute non-progressors ($p = 0.007$). Late progression was not predicted by baseline IFN Scores. However, within U-CTD, patients who required an immunosuppressant had higher expression of IFN Score A ($p = 0.011$) and IFN Score B ($p < 0.001$) than those who did not.

Conclusions Among ANA-positive referrals, no clinical feature or routine laboratory test could rule out development of clinically significant disease, but IFN Scores had a unique value in predicting these outcomes. At-Risk individuals who ultimately developed clinically significant disease are therefore immunologically but not clinically distinctive. Future work will incorporate biomarkers into clinically applicable risk models to allow earlier exclusion of AI-CTD or trials of preventative treatment.

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P42 **DISEASE ACTIVITY, IMPAIRED IRON TRANSPORT AND FAILED SEQUESTRATION: A NOVEL MECHANISM FOR ANAEMIA IN SYSTEMIC LUPUS ERYTHEMATOSUS**

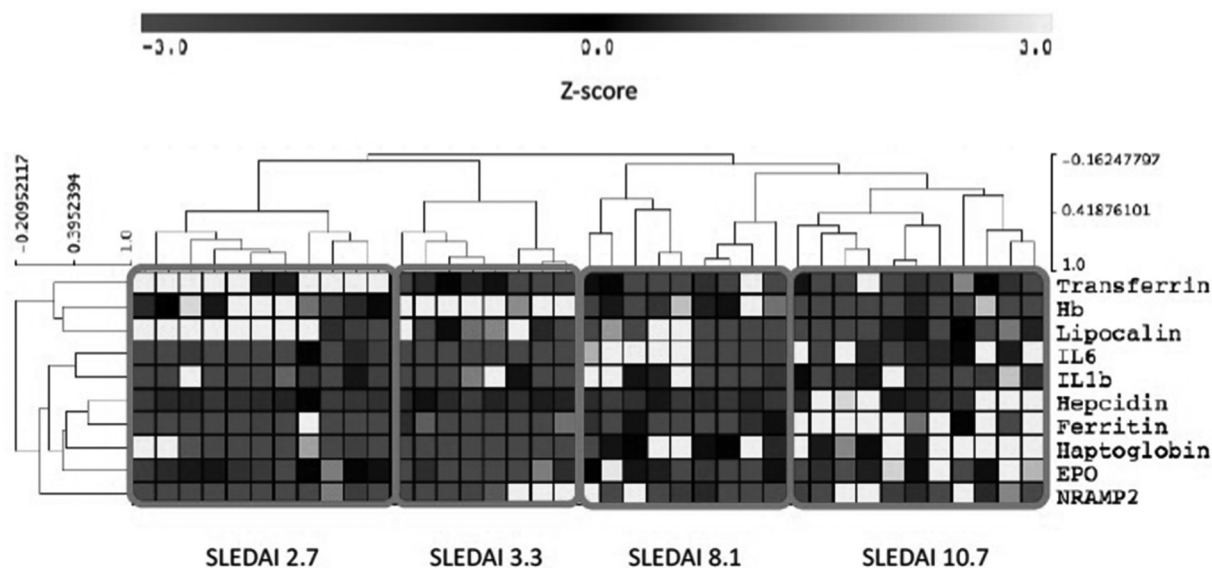
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Background Haematological manifestations are a frequent feature of systemic lupus erythematosus (SLE) yet the role of abnormal iron metabolism is poorly understood. In this study, we investigated the role of key regulators of iron metabolism including ferritin (an iron carrier protein), transferrin (which facilitate iron transport to cell surface receptors), hepcidin (which prevents iron release from stores under the influence of IL6 and IL1 β) and lipocalin-2 (LCN2, which is released by innate immune system activation and induces iron sequestration).

Methods Serum samples were collected from SLE patients without a history of haemolytic anaemia attending University College London Hospital, UK ($n = 39$). Clinical parameters including Haemoglobin (Hb), dsDNA, complement C3 and SLEDAI-2K were recorded. Levels of IL1 β , IL6, hepcidin, ferritin, LCN2 and transferrin in addition to other iron regulators including erythropoietin (EPO), soluble transferrin receptor (sTfR), haptoglobin (Hp) and NRAMP2, were measured by ELISA. Following normalisation of data, hierarchical correlate cluster was performed to produce a heatmap using MeV Software.

Results The results demonstrate a surprising negative correlation between LCN2 and SLEDAI-2K ($P < 0.001$, $r = -0.40$), which suggests iron is not being appropriately sequestered in



Abstract P42 Figure 1 Heatmap representation of cluster analysis of key iron mediators in SLE. Normalised data is presented as Z-score with -3.0

spite of immune activation. Hierarchical clustering is presented in figure 1. Cluster analysis demonstrates that the group with the highest mean SLEDAI-2K (10.7) had lower Hb, transferrin and LCN2 in addition to elevated IL1 β , IL6 and hepcidin compared with those with lower SLEDAI-2K. Elevated haptoglobin levels were seen in those in the higher disease activity groups, suggesting that haemolysis was an unlikely cause for anaemia.

Conclusions The findings of this study suggest increased lupus disease activity results in abnormal iron homeostasis through impaired cellular iron import (via reduced transferrin), a lack of stored iron release (under the actions of elevated hepcidin) and reduced iron sequestration by LCN2, which may represent a novel cause of non-haemolytic anaemia.

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P43 SERUM S100A8/A9 CONCENTRATIONS ARE ASSOCIATED WITH NEUROPSYCHIATRIC INVOLVEMENT AND FATIGUE IN SLE

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Background/Purpose Neuropsychiatric (NP) involvement and fatigue are both major problems in SLE. S100A8/A9 is a marker of inflammation, which responds to therapy in SLE patients. S100A8/A9 is expressed in the CNS. We investigated S100A8/A9 in relation to NPSLE and fatigue.

Methods In this cross-sectional study we used ELISA (Bhulmann MRP8/14 ELISA kit, Switzerland) to measure the concentration of S100A8/A9 in serum in 72 SLE patients and 26 healthy controls and in cerebrospinal fluid (CSF) in 33 SLE patients. NP involvement was determined according to ACR

case definitions for NPSLE. An MRI was performed in SLE patients and controls assessing white matter abnormalities and cerebral atrophy. Measurements of fatigue were performed using the Fatigue Severity Scale (FSS) and the Visual Analogue Scale (100 mm) (VAS). Statistical calculations were performed using non-parametric methods.

Results In all, 72 female SLE patients (median age 38, range 18–52) and 26 female healthy controls (median age 40, range 23–52) were included in this study. Forty-four (61%) patients had NP involvement. NPSLE patients had higher serum S100A8/A9 concentrations (median 1.40 μ g/ml) than the non-NPSLE patients (median 0.92 μ g/ml; $p=0.011$) and the control group (median 0.79 μ g/ml; $p=0.004$). Serum S100A8/A9 correlated with increased VAS fatigue in SLE patients ($r=0.311$; $p=0.008$), but not with FSS ($r=0.184$; $p=0.124$). Serum S100A8/A9 did not correlate with the extent of white matter lesions, atrophy of brain segments, or disease activity (SLEDAI-2K). S100A8/A9 was not detected in the CSF.

Conclusions Higher serum S100A8/A9 concentrations in NPSLE patients and patients with fatigue may indicate that S100A8/A9 is involved in the pathogenesis of these manifestations, although further investigation is needed.

P44 URTICARIAL VASCULITIS IN CHILDHOOD ONSET SYSTEMIC LUPUS ERYTHEMATOSUS

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Purpose Urticarial vasculitis (UV) is a rare type of vasculitis in children frequently, idiopathic, however, can be associated childhood onset SLE. The aim of the study is to describe the frequency of UV in cSLE and to compare the epidemiological, clinical and laboratory characteristics of cSLE stratified by the presence of UV in a cohort of patients of Arab ethnicity from Oman.