Objectives: Determine prevalence of MIN in SLE (LLC).

Compare clinical and echocardiographic (echo) features of patients with and without MIN.

Identify echo predictors of MIN.

Methods: A prospective cross-sectional study was done at Tygerberg Hospital, Western Cape, South Africa. Adult inpatients, fulfilling the 2012 SLICC criteria were screened. Echo analyses included STE and regional function (wall motion score (WMS)). Patients were grouped according to evidence of MIN (absent criteria [AC]; single criterion [SAC]; fulfilling LLC), comparing clinical, laboratory and echo data. Logistic regression and ROC were used to determine predictors of MIN.

Results: 49/106 SLE patients screened were included (Figure 1). 46.9% of patients had MIN (≥ 1 criterion): 12.2% fulfilled LLC for LM and 34.7% had a SAC. SLE disease activity (SLEDAI) ($p=0.022$) was higher in patients fulfilling LLC, but not in the SAC group. A clinical and echo diagnosis of LM was made in all patients fulfilling LLC, in 17.6% of patients in the SAC group and none in the AC group (Table 1). Anti-DsDNA and anti-B2GP1 were more frequently positive in SAC vs the AC group ($p=0.054$ and 0.081). WMS was higher in LLC and SAC groups ($p=0.006$; $p=0.083$) with mid and basal STE more impaired in patients with MIN ($p=0.047$; $p=0.043$). LVID and mid STE score combined was the best predictor of MIN (Table 2; Figure 2).

Conclusion: CMR evidence of MIN is common in SLE, even in the absence of clinical myocardial dysfunction or high lupus activity. Impaired echo regional and global function occurs more frequently in patients with MIN. STE combined with LVID predicts MIN detected by CMR and has potential as a cost effective screening tool. CMR is limited by a high exclusion rate in SLE, mainly due to renal impairment.

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Figure 1

Table 1

	AC n=26	LLC n=6	p	SAC n=17	p
	Med (IQR)				
Age	27 (21-35)	27 (23-28)	1	29 (23-36)	0.542
SLE duration, days	114(6-1366)	35(31-44)	0.724	955(7-2101)	0.486
SLEDAI	13(9-15)	22(16-26)	0.022	12(9-20)	0.813
Antiphospholipid syndrome	2 (8)	0	0.483	4 (24)	0.085
	n (%)				
Female	24 (92)	5 (83)	0.497	14(82)	0.319
Cardiac risk factors	8 (31)	1 (17)	0.489	6 (35)	0.757
Nephritis	9(35)	3(50)	0.483	3(18)	0.225
Clinical LM	0	6(100)	<0.001	3(18)	0.026

Table 2

Logistic regression			
	Univariate		
Echo variable	OR	95% CI	p
LVID	2.6	0.8-8.4	0.109
LV ejection fraction	0.9	0.8-0.9	0.018

Wall motion abnormality	4.2	1.3-13.9	0.018
GLS	1.3	1-1.6	0.049
STE score	Basal	1.6	0.9-2.7
	Mid	1.9	1.2-3.2
	Apical	1	0.9-3.9
		Multivariate	
LVID	3.5	0.9-13.3	0.070
Mid STE score	2.1	1.2-3.5	0.008

Figure 2

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FRI0231 IN PATIENTS WITH UCTD, IFN ACTIVITY IDENTIFIES PATIENTS THAT PROGRESS TO DEFINITIVE CTD CLASSIFICATION CRITERIA

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Background: Undifferentiated connective tissue disease (UCTD) is a term used to describe individuals with positive autoantibodies and symptoms but do not meet criteria for an established autoimmune CTD (AI-CTD). The prognosis of UCTD is unclear, could result in disease stability, remission or progression. As this condition is understudied, there is an unmet need for biomarkers to stratify those who progress to allow for a more aggressive therapy to be employed. IFN plays a major role in the pathogenesis of various AI-CTDs. However there are limited data on its role in AI-CTD.

Objectives: To determine whether IFN assays can predict progression from UCTD to meeting classification criteria of established AI-CTDs.

Methods: A prospective observational study was conducted on 43 consecutive patients with UCTD; as defined by Mosca criteria [1]. Progression was defined by meeting 2012 ACR/SLICC SLE, 2016 ACR/EULAR Primary Sjogren's, Bohan-Peter, 2013 ACR/EULAR Scleroderma, or 2006 Sydney Antiphospholipid syndrome criteria. For the interferon scores, RNA was extracted from PBMCs and a custom Taqman array was used to measure expression of 30 interferon stimulated genes (ISGs) as previously described [2]. A two-score system of ISGs (IFN-Score-A and Score-B), was calculated without the knowledge of participants' clinical status.

Results: Mean (range) age of the cohort was 48 (39-58) years; Female 36 (84%); mean disease duration 5.5 years; concomitant DMARDs including anti-malarials 38 (88%), concomitant corticosteroid 2(5%) and internal organ involvement 9 (21%). At the follow-up visit, 9/43 (21%) met established classification criteria for AI-CTD [SLE=6; IIM=2; and SSc=1]. Comparing the IFN scores between those who progressed (n=9) vs those remained undifferentiated (n=34); the IFN-Score-A was higher in the progressor group; fold difference (FD) 2.76 (95% CI 1.11-6.88); $p=0.030$. While for IFN-Score-B, there was a trend to association for higher expression in the progressor group vs remained undifferentiated; FD 1.74 (0.53-3.21); $p=0.077$.

Conclusion: IFN assays can identify progression to classifiable CTD within cohorts of UCTD. There is a potential role for the use of IFN biomarkers in the prognostication of patients with UCTD.

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FRI0232 PATIENT PERCEPTION OF SLE BURDEN: THE ROLE OF ORGAN DAMAGE

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Background: physician-based assessment of Systemic Lupus Erythematosus (SLE) may not be able to capture the real disease impact on patients' life. In the literature, the impact of disease damage on patients' quality of life (QoL) is controversial.

Objectives: Objective of our study was to investigate the role of organ damage in determining patient perception of SLE burden.

Methods: this is a cross-sectional study that enrolls patients with a diagnosis of SLE (ACR 1997 criteria). For each patient, demographics, comorbidities, treatment, clinical and laboratory data were collected. Disease damage was evaluated with the SLICC-Damage Index (SDI) and a score >2 was defined as "severe damage". The BILD (Brief Index of Lupus Damage) was used for patient self-evaluation of organ damage. Finally, the Lupus Impact Tracker (LIT) questionnaire was used to assess patient perception of SLE burden.

Results: we included 246 adult SLE patients (94.7% Caucasian, 93.1% female, mean age 45.31±13.2 years, mean disease duration 14.3±9.8 years). As for cumulative organ involvement in our cohort, the most prevalent was articular involvement (67.5%), followed by cutaneous (54.1%), hematological (51.2%), renal (43.9%) and serositis (17.9%); 11.8% had a history of NPSLE. Among comorbidities, 10.9% of patients had a concomitant fibromyalgia. 48.8% of patients was presenting at least one organ damage; among those patients, the median SDI was 2 (IQR 1-3); 16.3% of patients had a "severe damage" and among them median SDI was 4 (IQR 3-6). The most frequent items of organ damage in our cohort were: cataract (19.9%), deforming or erosive arthritis (10.2%) and osteoporosis with fracture (8.5%). Moreover, a significant number of patients (18.3%) met the criteria for neuropsychiatric damage, mainly cerebrovascular accidents (7.3%), seizures (5.3%) and cognitive impairment (4.1%). Finally, a not negligible number of patients had premature gonadal failure (4.5%) and malignancy (6.5%).

As far as patients' perception is concerned, the median LIT score in the cohort was 22.5 (IQR 7.5-40) and median BILD was 1 (IQR 0-2).

In a multiple linear regression analysis, we found a direct positive correlation between the SDI score and age at enrollment and disease duration ($p<0.001$). Severe damage resulted associated with a history of serositis ($p<0.01$) and NPSLE ($p<0.001$). We also found a positive linear correlation between the SDI score and the patient's self-reported damage (BILD score; $p<0.001$) and with the patient's perception of disease burden (LIT; $p<0.01$), irrespective of age and disease duration. In particular, in the multivariate analysis, higher SDI scores were significantly associated with a poorer perception of disease burden in terms of: future planning ($p<0.001$), usual activities, family responsibilities, discomfort due to physical appearance ($p<0.01$) and drug side effects ($p<0.05$), irrespective of fibromyalgia and age at enrollment.

Among the different types of organ damage, we found that cognitive impairment, cerebrovascular accidents and premature gonadal failure mainly contribute to determine SLE burden. In fact, they were significantly associated with higher LIT scores ($p<0.05$).

Conversely, SDI score was not related with health-related quality of life and fatigue as measured by SF-36 and FACIT respectively, neither with fibromyalgia.

Conclusion: disease damage seems to have a role in determining patient perception of SLE burden, mainly affecting patients' ability to plan the future and to fulfill daily activities and family responsibilities. In particular, neuropsychiatric damage exerts the greatest influence on patient perception of SLE impact.

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FRI0233 MORTALITY IN SLE PATIENTS COMPARED TO POPULATION CONTROLS IN FINLAND IN YEARS 2000–2015

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Background: It is well established from a variety of studies that systemic lupus erythematosus (SLE) patients have a shortened life expectancy. The literature on SLE has highlighted the impact of cardiovascular diseases (CVD) on increased mortality^{1,2}. However, there is lack of studies comparing results to the background population.

Objectives: Aim of the study was to clarify, whether incident SLE patients have an excess mortality compared to population controls.

Methods: The study included all adult (age ≥ 17 years), incident SLE patients who were entitled to a special reimbursement for SLE medication in years 2000 – 2014 in Finland. For each patient, the Population Register Centre identified 3 population controls matched for age, sex and place of residence. Comorbidities at baseline were obtained from the Care Register for Health Care of the National Institute for Health and Welfare. Data on education at baseline and deaths until the end of 2015 were retrieved from the Statistics Finland.

Results: A total of 1006 incident SLE patients (84% females) and 3005 population controls were found. During the follow-up (mean 8.6 years), 98 patients (mean age at death 70±14 years, 65% females) died. The 5-, 10-, and 15-year survival rates in SLE patients were 95.0% (95%CI 93.3-96.2%), 88.8% (86.2-91.0%) and 82.1% (77.6-85.8%), respectively. The number of deaths among controls was 187. Crude hazard ratio (HR) was 1.61 (95% CI: 1.26 to 2.06), $p<0.001$, adjusted for education- and comorbidities 1.14 (95% CI: 0.88 to 1.48) $p=0.32$. Main causes of deaths in patients were CVDs (33%), malignancies (27%) and neurological diseases (10%). Ten-year cumulative mortality rate due to CVD was in SLE patients 3.3% (95CI 2.2 to 4.9%) and in controls 2.6% (2.0-3.3) and 15-year rate 6.7% (95C% CI 4.2 to 9.9) and 4.9% (3.6 to 12.0), respectively. Crude HR for CVD deaths was 1.28 (95% CI: 0.85 to 1.93), $p=0.24$, adjusted 0.88 (95% CI: 0.56 to 1.39), $p=0.58$.

Conclusion: SLE patients had a slightly increased risk for overall and cardiovascular related mortality compared to population controls. After adjusting for education and comorbidities, the difference was not statistically significant.

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