echocardiogram, performed by a board-certified echocardiographer, to investigate the LALS, the strain of the three phases of the LA cycle and the strain rate. SLE disease activity was defined as a Mexican version of the Systemic Lupus Erythematosus Disease Activity Index (Mex-SLEDAI) ≥ 9 and SLE-related organ damage as SLICC/ACR damage Index (SDI) ≥ 1 . Descriptive statistics with mean and standard deviation or median and Interquartile range were calculated and categoric variables were expressed as proportions. Chi-squared test was done to compare categoric variables; quantitative variables were compared with student's t test. ANNOVA test was used to evaluate differences among multiple groups. A univariate linear regression analysis was performed to determine de independent factors associated with LAGLS less than thirty five percent.

Results: 90% of patients were females and their mean age was 34 (± 12) years old. 16% of patients had positive antiphospholipid antibodies. The mean MEX-SLEDAI was 8.7 (± 5) and the mean SDI was 0.7 (± 0.3). The mean of disease duration was 6 years. The patients with SLE had greater diastolic dysfunction evaluated with conventional echocardiography compared with CG (E/e' Ratio of 7.9 (± 2.6) vs 6.4 (± 1.8), $p=0.001$) (left atrial volume index (LAVI) of 26.1 mL/m² (± 8.8) vs 20.7 mL/m² (± 4.4), $p<0.001$). SLE patients had significant impaired of LALS compared with CG (41.6% vs 50.5%; $p=0.02$). As well in the 3 phases of the LA cycle. Also, the LALS was significantly lesser in patients with active disease than inactive and CS (36.3% (25.3-54.3) vs 43% (35.2-52.6) vs 50.5% (41.7-70.6)). The positivity of antiphospholipid antibodies, lupus nephritis, glucocorticoids use, and the presence of disease activity were significant independent factors for depressed LALS in the univariate analysis.

Conclusion: SLE patients have lower myocardial deformation of the LA, which is expressed as a lower diastolic function correlating with early subclinical myocardial damage.

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SAT0203 THE CORRELATION BETWEEN THE SERUM 1,25(OH)₂D₃ LEVEL AND CLINICAL DATA OF PATIENTS WITH PRIMARY SJOGREN'S SYNDROME

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Background: The role of vitamin D in regulating immune function in autoimmune diseases has been extensively studied. While there is less research on vitamin D in Sjogren's syndrome. This study will explain the relationship between vitamin D and Sjogren's syndrome.

Objectives: To explore the relationship between the serum 1,25(OH)₂D₃ level and the changes of disease activity in patients with Primary Sjogren's Syndrome (PSS) [1], and to explore whether supplementation of VD could be a potential therapy for the treatment of PSS.

Methods: PSS patients (n=60, according to the 2002 International Classification (Diagnostic) Standard for Sjogren's Syndrome) were enrolled and health individuals were normal controls. Laboratory examinations were analyzed including that serum levels of 1,25-dihydroxycholecalciferol [1,25(OH)₂D₃], lymphocytes and CD4+T cell subsets by flow cytometry, urine pH value, ALT, AST, BUN, Cr, IgG, C3, C4, ESR, CRP, serum levels of 1,25(OH)₂D₃. Physical examination data include Salivary flow rate, secretion of tears, breakup time of tear film (BUT), and labial glands biopsy. Patient's disease activity was accord to ESSDAI (EULAR SS disease activity index, 2010) classification.

Results: Compared with that in normal controls, the levels of serum VD in patients with PSS was decrease significantly, $Z = -7.367$, $P < 0.001$ (Figure 1). Spearman correlation analysis and comparison with the collected indexes, showed that VD was significantly correlated with secretion of tears ($r = -0.455$, $p < 0.002$), IgG ($R = -0.581$, $p < 0.000$), B cell absolute count ($R = -0.474$, $p = 0.002$), B cell percent ($R = -0.391$, $p = 0.005$), Th1 absolute count ($R = 0.318$, $p = 0.023$), Th17 absolute count ($r = 0.297$, $p = 0.034$), Th17

Table 2. Correlation between the serum 1,25(OH)₂D₃ level and clinical data

the serum 1,25(OH) ₂ D ₃ level	BMI		disease duration		ESSDAI		base salivary flow rate		stimulant salivary flow rate	
	r	P	r	P	r	P	r	P	r	P
-196	.300		-.113		.416		-.123		.037	
									-.077	
							.621			
									-.049	
									.756	
the serum 1,25(OH) ₂ D ₃ level	WBC		RBC		HB		PLT		LY	
	r	P	r	P	r	P	r	P	r	P
-.558	.684		-.240		.089		-.189		.189	
									-.170	
							.230			
									-.148	
									.301	
the serum 1,25(OH) ₂ D ₃ level	neutrophile granulocyte		urine pH value		ESR		ALT		AST	
	r	P	r	P	r	P	r	P	r	P
-.003	.985		.335		.013		-.111		.432	
							.132		.344	
									.001	
									.996	
the serum 1,25(OH) ₂ D ₃ level	BUN		Cr		C3		C4		CRP	
	r	P	r	P	r	P	r	P	r	P
-.081	.556		.120		.382		-.148		.381	
									.202	
							.224			
									.000	
									.998	
the serum 1,25(OH) ₂ D ₃ level	T cell (CD3 ⁺ CD19 ⁺)		T cells%		B cell (CD3 ⁺ CD19 ⁺)		B cell%		Th cell (CD3 ⁺ CD4 ⁺)	
	r	P	r	P	r	P	r	P	r	P
.034	.810		.242		.088		-.276		.046	
									-.391	
							.005		-.035	
									.809	
									-.092	
									.523	
the serum 1,25(OH) ₂ D ₃ level	Ts cell (CD3 ⁺ CD8 ⁺)		Ts cell%		NK cell (CD3 ⁺ CD16 ⁺ CD56 ⁺)		Th1 cells		Th2 cells	
	r	P	r	P	r	P	r	P	r	P
.239	.091		.372		.007		.121		.388	
									.318	
							.023		.029	
									.842	
									.297	
									.034	
the serum 1,25(OH) ₂ D ₃ level	Treg cells		Th1 cell%		Th2 cell%		Th17 cell%		Treg cell%	
	r	P	r	P	r	P	r	P	r	P
-.274	.052		.282		.045		-.058		.685	
									.263	
							.059			
									-.170	
									.227	
the serum 1,25(OH) ₂ D ₃ level	Th1/Th2		Th17/Treg							
	r	P	r	P	r	P	r	P	r	P
.382	.004						.385		.004	

*P<0.05, **P<0.01, ***P<0.001

percent ($r=0.282$, $p=0.007$), ratio of Th1/Th2($r=0.385$, $p=0.004$) and ratio of Th17/Tregs($r=0.385$, $p=0.004$) (Table 2).

Conclusion: The level of serum VD in PSS patients was significantly lower than that in normal controls, which was negatively correlated to secretion of tears, IgG, the absolute number of B, Th1, and Th17, the percentage of B and Th17, and ratios of Th1/Th2 and Th17/Tregs. Raising serum VD levels by supplementation of VD, may be a potential Therapy for Sjogren's syndrome.

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Figure 1. Comparison of VD levels(ng/ml) between patients with PSS and healthy people

Results are given as median (P_{25}, P_{75}). **patients with PSS** 10.2 (9.00,15.11), **healthy people** 20.71(15.16,26.41) $Z=-7.37$ $P<0.001$

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SAT0204 LUPUS LOW-DISEASE ACTIVITY STATE VS SLE RESPONDER INDEX IN A "REAL-LIFE" SETTING

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Background: Systemic lupus erythematosus (SLE) is a chronic multi-organ autoimmune disease characterised by a heterogeneous pathogenic background and by protean clinical manifestations. Recent efforts in identifying novel agents for therapeutic interventions have led to disappointing results and designing a reliable way to assess drug efficacy and verify the achievement of remission, has become itself a major challenge. The SLE responder index (SRI) is a composite tool developed and validated in clinical trials to define in patients with active disease an acceptable response to investigational agents. The role of SRI outside clinical trials has not been defined. The lupus low disease activity state (LLDAS) has been originally validated in a cohort-based setting and it focuses on the achievement of fixed conditions rather than variations from baseline. A formal comparison of these tools outside clinical trials has not been performed.

Objectives: To prospectively assess the performance of SRI and LLDAS in a "real world" observational setting.

Methods: One hundred-thirty-one consecutive patients SLE were subdivided into two groups based on the need or not to escalate their immune suppressive treatment. Clinimetrics including Physician Global Assessment scale (PGA), SLE Disease Activity Index 2000 (SLEDAI-2K) and British Isles Lupus Assessment Group index (BILAG) 2004 version were measured at baseline and at six and 12 months, together with laboratory data and treatment changes. LLDAS and SRI were calculated at each time point.

Results: LLDAS achievement correlated with treatment de-escalation over 12 months ($\chi^2=5.2$, $p=0.034$, OR=4.1 with 95%CI=1.2-14.4), whereas SRI-4 achievement did not. LLDAS responses were more frequent in patients with lower basal SLEDAI-2K (i.e. SLEDAI-2K<6; $\chi^2=21.5$, $p<0.001$, RR=8.4 with 95%CI=3.2-25.0), while SRI-4 responses were more prevalent among patients with higher basal SLEDAI-2K and/or severe renal activity at baseline ($\chi^2=5.9$, $p=0.025$, RR=10.8 with 95%CI=1.1-103.1). Positive anti-

DNA antibodies associated with non-achievement of LLDAS at any time point. Serositis was associated with lack of LLDAS at baseline, but did not affect LLDAS achievement at 12 months. Normalising complement levels anticipated the achievement of LLDAS and SRI-4.

Conclusion: LLDAS is a valuable tool for assessing response to treatment in the daily rheumatology practice. SRI might be less informative, at least in patients with low basal SLEDAI.

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SAT0205 A CYTOKINE "SCAR SIGNATURE" CHARACTERIZES PATIENTS WITH FATIGUE IN SYSTEMIC LUPUS ERYTHEMATOSUS AND SJOJREN'S SYNDROME

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Background: Fatigue is highly prevalent in systemic lupus erythematosus patients (SLE) and primary Sjogren's syndrome (pSS) and represents one of its unmet needs.[1] Its pathogenesis is multifactorial, with the activity of the underlying disease exerting a prominent role, along with psychological factors and co-morbid conditions. Previous studies evaluating fatigue and cytokines in patients with SLE and pSS have yielded inconclusive results.

Objectives: We aimed to evaluate patient-reported outcome measures reflecting fatigue and their correlation to serum cytokines in patients with SLE and pSS and healthy volunteers (HV). A panel of circulating cytokines, chemokines and growth factors was compared between groups, correlated to the level of fatigue and within SLE and pSS to global disease activity. The objective was to identify cytokines reflecting the degree of fatigue, which could be exploited as biomarkers and therapeutic targets.

Methods: We performed a cross-sectional study on subjects included in the Swiss SLE Cohort Study (SSCS). All subjects were evaluated clinically and had a serum sample taken. Fatigue was assessed by FAS (Fatigue Assessment Scale) and by the vitality subscale (VT) of the Medical Outcomes Study 36-Items Short Form Healthy Survey. Clinical activity in SLE and pSS patients was determined by a 4-point Likert-scale Physician's Global Assessment (PGA). SLE activity was assessed with the SLE Disease Activity Index score with the Safety of Estrogens in SLE National Assessment modification (SELENA-SLEDAI). Serum cytokines were assessed by multiplex bead array analysis (ProcartaPlex, ThermoFisher Scientific, USA). P values were adjusted for multiple comparisons.

Results: Fifty-six patients with SLE, 18 with pSS and 18 healthy volunteers (HV) were included between November 2015 and June 2016. There were no significant differences between groups regarding to age, gender and body mass index (BMI). FAS and VT correlated strongly (Spearman's rho -0.87, $p<0.01$). FAS was significantly higher in patients than in healthy individuals (median FAS 23 [16-31], 28[20.5-35], 17 [15-27] in SLE, pSS and HV respectively; $p=0.02$). Patients with SLE and pSS displayed higher serum levels of interferon (IFN)-gamma (median [IQR] 10.29 [4.94-15.25] pg/mL in SLE, 9.64 [6.25-13.86] pg/mL, undetectable in HV; $p<0.01$). Interleukin (IL)-10 also was only detected in pSS and SLE. Hepatocyte growth factor (HGF) was more expressed in patients than in controls ($p=0.01$). The levels of most other cytokines (IFN-alpha, IL-1 alpha, IL-2, IL-4, IL-6, IL-17, IL-21 and IL-23) were not detectable. Only HGF displayed a significant correlation with FAS (P Pearson 0.29, $p<0.01$).

Conclusion: Patients with SLE and pSS display a molecular pattern of chronic inflammatory conditions, with higher serum levels of IFN-gamma, IL-10 and HGF. The latter two are both involved in regeneration and tissue repair ("scar signature")[2]. HGF levels correlated independently with