from tramadol for worst pain, average pain, and the pain interference index, with exception of the pain interference index for tanezumab 10mg ($p=0.01$). Mean dose of tramadol was 203mg/day at week 16.

Tanezumab 10mg significantly ($p<0.05$) improved individual domains of the pain interference index (general activity, walking ability, sleep, and normal work) vs placebo and vs tramadol. Tanezumab 5mg significantly ($p<0.05$) improved pain interference with general activity and normal work vs placebo, and sleep vs placebo and vs tramadol. No statistical differences in any domain was observed for tramadol vs placebo.

Conclusion: Tanezumab 5mg and 10mg significantly improved worst pain, average pain, and overall pain interference index scores vs placebo in patients with CLBP. Tanezumab 10mg also significantly improved the overall pain interference index vs tramadol. Tanezumab 5mg significantly improved most individual domains of the pain interference index vs placebo, while tanezumab 10mg significantly improved all domains assessed vs placebo and vs tramadol.

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SLE, Sjögren's and APS clinical aspects

OP0091

A TWO-SCORE INTERFERON SIGNATURE AND MUSCULOSKELETAL IMAGING EXPLAIN THE ASSOCIATION BETWEEN INTERFERON AND ARTHRITIS IN SLE

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Background: Interferon (IFN) signature is associated with disease activity and flare in SLE. We previously described two independent IFN gene expression scores; IFN Score A (the most commonly measured ISGs) and IFN Score B (less commonly measured ISGs which may also respond to IFN-II or other immune mediators)[1]. Many more clinical outcomes are associated with IFN Score B than with a "classic" interferon signature. These include progression of At-Risk individuals to SLE, response to rituximab, and differentiation of IFN signature in RA and SLE.

In previous work, the relationship of IFN Signatures with arthritis was less clear than for other SLE features. This may be related to the local regulatory effects of IFN-beta in the synovium, contrasting with the pro-inflammatory effects of other interferons. Another reason may be the proven imprecision of clinical examination as a measure of MSK inflammation in SLE.

USEFUL was a multicentre longitudinal study including serial ultrasound assessment of SLE patients with inflammatory MSK pain receiving treatment with glucocorticoids (GC).

Objectives: To determine whether IFN scores A and B are associated with imaging-proven synovitis in SLE and measure the responsiveness of IFN scores to GC treatment.

Methods: 133 SLE patients were recruited into the USEFUL study if the referring physician deemed they had inflammatory pain warranting treatment. Participants received depomedrone 120mg IM then were assessed at 0, 2 and 6 weeks using clinical instruments and ultrasound (US). OMERACT US criteria were used to categorise patients as active (GS2 or PD1 in at least one joint or

tendon), active in both joints and tendons, or non-active (no GS1 and PD0 or better in all joints).

Expression of 26 interferon stimulated genes, normalised to PP1A was measured in whole blood collected in TEMPUS tubes using a custom Taqman array. IFN scores A and B were calculated as previously described[1]. Missing data was imputed using expectation-maximisation method. Parametric tests were applied with post hoc Tukey to compare scores between groups.

Results: At baseline, there was no significant difference in IFN Score A between ultrasound groups ($F = 1.045$, $p = 0.355$). In contrast, IFN Score B differed significantly between ultrasound groups ($F = 4.168$, $p = 0.018$). The greatest difference was between active ultrasound for both joints and tendons ($n=22$) and non-active ultrasound ($n=53$) (difference = 0.75, 95% CI 0.13, 1.37, $p=0.013$).

There was no significant change from baseline in IFN Score A at week 2 (mean difference 0.08, 95% -0.14, 0.31, $p = 0.45$) or week 6 (mean difference -0.03, 95% -0.25, 0.19, $p = 0.79$). Similarly, there was no significant change in IFN Score B at week 2 (mean difference -0.01, 95% -0.18, 0.17, $p = 0.93$) or week 6 (mean difference -0.07, 95% -0.21, 0.08, $p = 0.36$).

Conclusion: Previous studies were unable to demonstrate an association between a typical interferon signature and arthritis in SLE. Our study includes a homogenous patient population and therapy, objective measure of synovitis, and a more detailed assessment of IFN Status. We found that imaging-proven synovitis is associated with increased expression of a specific subset of ISGs (IFN score B), but not a the more typical interferon signature genes (IFN Score A).

This increases the body of evidence for the value of IFN score B in predicting clinical outcomes. GC treatment did not affect systemic IFN signature scores at follow up. Future analysis will explore the role of IFN Scores in predicting clinical responses to therapy in this study.

References:

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OP0092

OPTIMIZING A DEFINITION OF LUPUS LOW DISEASE ACTIVITY STATE (LDA) FOR DAILY CLINICAL PRACTICE: SLE-DAS LDA VS LLDAS

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Background: The Lupus Low Disease Activity State (LLDAS)[1] is a proposed target for the management of Systemic Lupus Erythematosus (SLE). However, the LLDAS definition is cumbersome to apply, as it requires comparison with manifestations in the previous visit, scoring of Systemic Lupus Erythematosus disease activity index (SLEDAI), Physician Global Assessment (PGA), treatment conditions and exclusion of additional features. The SLE disease activity score (SLE-DAS)[2] is a validated continuous measure with higher sensitivity to change and validity in predicting damage accrual as compared to SLEDAI-2K. SLE-DAS is quickly quantified with its online calculator. The SLE-DAS low disease activity (SLE-DAS LDA) was recently defined [3] and it is easier to apply than the LLDAS.

Objectives: To compare the performance of SLE-DAS and LLDAS for defining LDA state in a real-life clinical setting.

Methods: Cross-sectional study of SLE patients fulfilling ACR'97 and/or SLICC'12 classification criteria followed at an academic lupus clinic, from January to December 2019. Fulfilment of LLDAS and SLE-DAS LDA state was verified for each patient. The SLE-DAS LDA state was defined as (1) SLE-DAS ≤ 3.77 [3] with (2) prednisolone dose ≤ 7.5 mg/day. The proportion of cases in LDA state using LLDAS and SLE-DAS LDA was compared with McNemar's test. Agreement between LLDAS and SLE-DAS LDA was tested with Cohen's Kappa coefficient.

Results: We included 292 patients (86.6% female, mean age: 48.7 ± 14.4 years, mean disease duration: 14.4 ± 9.3 years). From these, 245 (83.9%)

and 248 (85%) were in LDA according to LLDAS and SLE-DAS LDA, respectively. There was no statistically significant difference between LLDAS and SLE-DAS LDA ($p=0.581$ regarding the identification of patients in LDA. Agreement between these two sets of criteria was almost perfect ($k=0.831$, $p<0.01$).

There were 13 discordant cases, fulfilling only the SLE-DAS LDA ($n=8$) or the LLDAS ($n=5$), and their manifestations of disease activity (excluding serologic features) are summarized in table 1.

Patients fulfilling only SLE-DAS LDA

Number of cases	PGA (range)	Active clinical manifestations (range)	SLEDAI-2K (range)	SLE-DAS (range)	Prednisolone (range, mg/day)
6	0.1-0.2	Leukopenia 2.2-2.7x10 ⁹ /L	1-3	1.46-3.03	0-2.5
2	0.2-0.3	Thrombocytopenia 71-96x10 ⁹ /L	1-3	1.97-2.86	0-5

Patients fulfilling only LLDAS

Number of cases	PGA (range)	Active clinical manifestations (range)	SLEDAI-2K (range)	SLE-DAS (range)	Prednisolone (range, mg/day)
2	0.2-0.4	Arthritis (2-4/28 swollen joints)	4	4.41-5.31	0-5
1	0.5	Panniculitis (face and torso)	3	5.53	0
1	0.1	Generalized rash	4	5.01	5
1	0.4	Leukopenia 2.1x10 ⁹ /L Thrombocytopenia 62x10 ⁹ /L	2	4.99	0

Conclusion: A LDA state, by either definition, was achieved by most patients in this real-life setting. LLDAS and SLE-DAS LDA identify almost exactly the same population. The SLE-DAS LDA definition is easier to apply and hence might be the optimal definition for use in daily clinical practice.

References:

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OP0093

FREQUENCIES AND PREDICTORS OF THE LUPUS LOW DISEASE ACTIVITY STATE AND REMISSION IN TREATMENT-NAÏVE PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS -- A REAL-WORLD COHORT STUDY

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Background: After the introduction of treat-to-target strategy in systemic lupus erythematosus (SLE), Lupus Low Disease Activity State (LLDAS) and definitions of remission in SLE (DORIS) were developed and validated. Several studies had demonstrated that the achievement and maintenance of LLDAS or DORIS was associated with good prognosis.

Objectives: To evaluate the attainability of LLDAS and DORIS in a treatment-naïve cohort of SLE.

Methods: LDAS5 was defined as LLDAS with a prednisone dose ≤ 5 mg/d. There were 4 definitions in DORIS: clinical remission on treatment (RONT), complete RONT, clinical remission off treatment (ROFT) and complete ROFT. The treatment-naïve patients from Peking University First Hospital SLE cohort were enrolled. The time to each state and their annual cumulative

probabilities were estimated by Kaplan-Meier approach. The frequencies of patients who achieved each component of LLDAS or DORIS during follow-up were determined.

Results: A total of 218 treatment-naïve patients were included, with a median follow-up of 4.48 years. Respectively, 190 (87.2%), 160 (73.4%), 148 (67.9%), 94 (43.1%), 23 (10.6%) and 18 (8.3%) patients achieved LLDAS, LLDAS5, clinical RONT, complete RONT, clinical ROFT and complete ROFT at least once during the follow-up time. The median time to LLDAS, LLDAS5, clinical RONT and complete RONT were 1.4, 2.3, 2.6 and 4.7 years, respectively.

Figure 1 Kaplan-Meier curves of time to LLDAS, LLDAS5 and each state of DORIS.

Table 1. Frequencies, time to achieve and annual cumulative probabilities of each state by Kaplan-Meier approach

States	Achieved patients Number (%)	Time to achieve (years)	Cumulative probabilities of achievement (%)				
			Year 1	Year 2	Year 3	Year 4	Year 5
LLDAS	190 (87.2)	1.4	18.8	69.7	86.7	89.1	92.6
LLDAS5	160 (73.4)	2.3	6.9	40.7	63.3	76.0	82.3
Clinical RONT	148 (67.9)	2.6	5.5	36.1	56.1	68.8	76.6
Complete RONT	94 (43.1)	4.7	4.1	22.6	37.5	45.3	50.4
Clinical ROFT	23 (10.6)	NA	1.4	2.9	5.4	6.7	10.6
Complete ROFT	18 (8.3)	NA	0.9	2.5	4.8	4.8	8.8

Table 2. Patients who achieved each component of LLDAS or DORIS during follow-up

Components	Number (%)
SLEDAI-2K ≤ 4 , with no activity in major organ systems (renal, central nervous system, cardiopulmonary, vasculitis, fever), and no haemolytic anaemia or gastrointestinal active	213 (97.7)
Clinical SLEDAI-2K = 0	210 (96.3)
PGA ≤ 1	217 (99.5)
PGA < 0.5	199 (91.3)
Serology (anti-dsDNA and complement) negative	148 (67.9)
Prednisone dose ≤ 7.5 mg/day	201 (92.2)
Prednisone dose ≤ 5 mg/day	171 (78.4)
No prednisone dose	40 (18.3)
No prednisone dose and Immunosuppressants	32 (14.7)

Conclusion: Our data confirmed that LLDAS is an attainable early treatment target for SLE. Though with more difficulty, RONT can be achieved in two-thirds of our patients. ROFT may not be an ideal treatment target at present as it is only attained in few patients.

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