Figure 2. Bootstrapped mean oral morphine equivalent dose per person per year for patients with inflammatory arthritis (above) and age and sex matched comparators (below). Box edges represent 25-75th percentiles and whiskers 95% confidence intervals.

Table 1. Baseline demographic data. Mean $\pm$ SD unless specified. * defined from diagnosis to baseline.

	All patients	Rheumatoid arthritis	Psoriatic arthritis	Ankylosing spondylitis	Undifferentiated arthritis
Total n (%)	940 (100)	359 (38)	251 (27)	217 (23)	113 (12)
Age (years)	49 $\pm$ 14	53 $\pm$ 14	49 $\pm$ 13	43 $\pm$ 13	44 $\pm$ 15
Disease duration (years)*	7.8 $\pm$ 8.5	8.2 $\pm$ 8.2	7.4 $\pm$ 7.8	8.3 $\pm$ 10.2	6.3 $\pm$ 6.6
Female	58%	73%	59%	34%	52%

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OP0089 EFFICACY OF CLOMIPRAMINE FOR CHRONIC LUMBAR RADICULAR PAIN A RANDOMIZED CLINICAL TRIAL

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Background: Lumbar radicular pain is the most common chronic neuropathic pain syndrome. Antidepressants are highly recommended for neuropathic pain, but there is no evidence for their efficacy.

Objectives: The aim of this double-blind, randomised, placebo-controlled trial is to determine whether Clomipramine (an antidepressant) is more effective than placebo in reducing pain in individuals with resistant chronic lumbar radicular pain.

Methods: A double-blind, randomized, clinical trial. Sixty-two patients with resistant chronic lumbar radicular pain were included. (The sample size was calculated on the assumption that clomipramine would reduce the incidence of lumbar radicular pain of 35%, compared with placebo, with a two-sided test, an alpha level of 0.05, and a power of 85%). Patients were randomly allocated to receive either Clomipramine by slow intravenous infusion for 10 days in a hospital setting with progressively increasing doses, 25mg on the first day, 50mg on the second day and 75mg on the third day until the tenth day, or placebo (500ml of physiological serum a day). For both groups, paracetamol is added intravenously at a dose of 3g per day for ten days, Parecoxib for 3 days and ten sessions of lumbar spine rehabilitation including analgesic massage, muscle strengthening and joint maintenance. At the exit, clomipramine was relayed with 25mg per day orally until the 90th day for

clomipramine group, and paracetamol was authorized in both groups, in case of severe pain. The primary outcome was pain intensity, measured at baseline, 5th day, 10th day and 90th day using VAS pain (10mm). Secondary outcome included DN4-questionnaire, lumbar radicular discomfort (VAS 10mm), pain-free perimeter of walking (min), disability assessed using the Roland Morris Disability questionnaire and severity of mood symptoms assessed using the Hospital Anxiety and Depression scale (HAD), measured on days 0, 5, 10 and 90.

Results: 31 patients were assigned to the clomipramine group and 31 to the placebo group. There were no differences between the groups in demographic characteristics. Treatment by Clomipramine had a significantly greater reduction in pain, discomfort and DN4 from the 5th day ($p = 0.000$, $p = 0.001$ and $p = 0.004$ respectively) than the placebo, with an improvement maintained until 90th day. There was a statistically significant improvement in pain-free walking distance and disability for the clomipramine group from the 10th day ($p = 0.02$ and 0.001 respectively) and that maintained until 90th day. However, there was no significant improvement in HAD between the 2 groups. ($p \geq 0.1$).

Conclusion: This double-blind, randomized, clinical trial shows that clomipramine is quickly effective and maintained over time in the management of resistant chronic lumbar radicular pain. It can therefore be part of the therapeutic arsenal in this sense.

Disclosure of Interests: None declared

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OP0090 EFFICACY OF SUBCUTANEOUS TANEZUMAB FOR THE TREATMENT OF CHRONIC LOW BACK PAIN: AN ANALYSIS OF BRIEF PAIN INVENTORY-SHORT FORM SCORES FROM A 56-WEEK, RANDOMIZED, PLACEBO- AND TRAMADOL-CONTROLLED, PHASE 3 TRIAL

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Background: Tanezumab, a monoclonal antibody against nerve growth factor, was recently evaluated in an 80 week placebo and tramadol-controlled trial in patients with chronic low back pain (CLBP) and a history of inadequate response to standard-of-care analgesics (NSAIDs, opioids, etc). Primary endpoint was change in Low Back Pain Intensity (LBPI) at week 16 vs placebo. Key secondary endpoints were the proportion of patients with $\geq 50\%$ improvement in LBPI at week 16, change in Roland Morris Disability Questionnaire score at week 16, and change in LBPI at week 2 (all vs placebo). Tanezumab 10mg met the primary and all key secondary endpoints. Tanezumab 5mg did not meet the primary endpoint, but improved 2 of 3 key secondary endpoints. Due to the primary endpoint result and the statistical gate-keeping approach to control for multiple comparisons, a conclusion of superiority over placebo could not be made for the 5mg dose.

Objectives: To further characterize tanezumab's effects on pain and function in this trial through analysis of Brief Pain Inventory-short form (BPI-sf) scores.

Methods: Patients received placebo (n=406), subcutaneous (SC) tanezumab 5mg (every 8 weeks; n=407), SC tanezumab 10mg (every 8 weeks; n=407) or oral tramadol prolonged-release (100-300mg/day; n=605). Pre-specified secondary endpoints included BPI-sf worst pain, average pain, the overall pain interference index, and selected individual domains of the index (general activity, walking ability, sleep, and normal work). Least squares (LS) mean (standard error [SE]) changes from baseline in BPI-sf scores were compared between groups (unadjusted for multiplicity) at week 16 using an analysis of covariance model. Scores range from 0-10 with higher scores indicating greater pain severity or functional impairment.

Results: LS mean (SE) differences from placebo for worst pain were -0.52 (0.19) for tanezumab 5mg ($p \leq 0.01$), -0.54 (0.19) for tanezumab 10mg (≤ 0.01), and -0.24 (0.17) for tramadol ($p=0.17$). LS mean (SE) differences from placebo for average pain were -0.37 (0.18) for tanezumab 5mg ($p=0.04$), -0.46 (0.18) for tanezumab 10mg (≤ 0.01), and -0.17 (0.16) for tramadol ($p=0.29$). LS mean (SE) differences from placebo for the pain interference index were -0.41 (0.18) for tanezumab 5mg ($p=0.03$), -0.58 (0.18) for tanezumab 10mg (≤ 0.01), and -0.15 (0.17) for tramadol ($p=0.39$). Effects of tanezumab were not statistically different ($p > 0.05$)

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:

[1] El-Sherbiny, Y.M., et al. Scientific Reports, 2018. 8(1): p. 5793.

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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. Fulfillment 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%)