group (32.6% vs 21.8%), but without statistical significance. Seropositivity of various autoantibodies was comparable between those with and without LTBI. Basal and stimulated IFN-γ levels was lower in IGRA and autoantibodies positive group(p=0.014) compared to IGRA positive antibody negative group.

Conclusions This study showed higher prevalence of LTBI in antibody positive FDRs of SLE. Basal and stimulated IFN-γ levels were lower in antibody positive group, in contrast to SLE patients who have higher basal IFN-γ. Further longitudinal studies would be required to see the effect of these autoantibodies on LTBI and risk of progression to TB.

LP-170 PREDICTORS OF NEUROPSYCHIATRIC FLARES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: RESULTS FROM FIVE PHASE III CLINICAL TRIALS OF BELIMUMAB

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Background Belimumab has shown ability to reduce flare rates in systemic lupus erythematosus (SLE), but little is known about its potential benefits in neuropsychiatric (NP)SLE. We investigated predictors of NP flares in SLE patients under standard therapy with or without add-on belimumab.

Methods Data from five clinical trials of belimumab in SLE (BLISS-52, BLISS-76, BLISS-NEA, BLISS-SC, EMBRACE) were utilised (N=3638). Flares were defined using the British Isles Lupus Assessment Group (BILAG) activity index. Predictors of flares were investigated throughout a 52-week follow-up using univariable and multivariable Cox regression. A subgroup analysis in patients with baseline NP BILAG E was performed to determine predictors of de novo NP flare. Organ damage was assessed using the SLICC/ACR Damage Index (SDI).

Results In total, 105 (2.9%) NP flares were documented. In multivariable analysis, male sex (HR: 2.37; 95% CI: 1.31–4.28; p=0.004), baseline NP BILAG B-D (HR: 5.91; 95% CI: 3.86–9.06; p<0.001), and high baseline SDI score (HR: 1.35; 95% CI: 1.21–1.50; p<0.001) were highly associated with NP flare development. Belimumab use yielded no clear protection. In a separate analysis of SDI domains, the NP domain (HR: 3.25; 95% CI: 2.72–3.88; p<0.001) was the strongest predictor of NP flare, with cognitive impairment (HR: 14.29; 95% CI: 9.22–22.14; p<0.001), transverse myelitis (HR: 21.89; 95% CI: 5.40–88.72; p<0.001), and neuropathy (HR: 8.87; 95% CI: 5.59–14.09; p<0.001) mainly driving this association. In the subgroup analysis of the NP BILAG E population, male sex was the strongest predictor of de novo NP flare (HR: 3.26; 95% CI: 1.51–7.04; p=0.003).

Conclusions Current or former NPSLE activity and established organ damage in the NP domain at baseline were the strongest predictors of NP flare. Whether belimumab treatment protects against NP events remains unclear.

LP-171 FIBROMYALGIA AND GLUCOCORTICOIDS USE DRIVES SELF-PERCEIVED DEPRESSION IN SYSTEMIC LUPUS ERYTHEMATOSUS: INSIGHTS FROM A LARGE PROSPECTIVE AND MULTICENTER STUDY USING RELESSERPROS REGISTER'S DATABASE.

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Background Little is known about depression in SLE. To evaluate the prevalence of depression and associated factors in a large, multicenter SLE cohort (RELESSER-PROS).

Methods Prospective longitudinal study of SLE patients answering positively to the depression question of the Lupus Impact Tracker (LIT) questionary (question number 7, LITQ7 'I was depressed') over 5 consecutive annual visits (V1 to V5). Self-perceived depression was answered from 0 ('none of the time') to 4 ('most of time'). Covariates with potential impact in depression were considered. Friedman test and GEE models were used.

Results 1463 patients were included. Mean age 55 years, 90% female. Mean disease duration: 14 years. Fibromyalgia was present in 5.7%. Glucocorticoids use ranged from 49.4% to 57%, depending on the visit. SLEDAI ranged from 0 to 2 and SDI from 1 to 2. Prevalence of 'depression any time' was