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

[1]Leonardo Palazzo, [1]Julius Lindblom*, [1]Nursen Cetrez, [1]Henri Ala, [1,2]Ioannis Parodis. *[1]Division of Rheumatology, Department of Medicine Solna, Karolinska Institutet, Sweden; [2]Department of Rheumatology, Faculty of Medicine and Health, Örebro University, Sweden*

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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.

[1]Iñigo Rúa-Figueroa, [2]Julia Martínez-Barrio, [3]Norman Jiménez, [4]María Galindo-Izquierdo, [5]Esther Uriarte-Isacelaya, [6]Antonio Fernandez-Nebro, [7]Jaime Calvo, [6]Sara Manrique-Arija, [8]Jose Rosas, [9]Rocio Caño-Alameda, [10]J. Narváez, [11]Inmaculada Ros, [12]Elena Aurrecoechea, [13]Vicenç Torrente-Segarra, [14]Clara Sanguesa, [15]Mercedes Freire-González, [16]Eva Tomero-Muriel, [17]Loreto Horcada, [18]Clara Moriano, [19]Mireia Moreno, [20]Carlota Laura-Iñíguez, [21]Ricardo Blanco, [22]Ana Pérez-Gómez, [23]José Luis Andréu-Sánchez, [24]Sandra Garrote-Corral, [25]Santiago Muñoz-Fernández, [26]Gema Bonilla, [27]Nuria Lozano-Rivas, [28]Carlos A Montilla-Morales, [29]O Ibarguengoitia, [30]Lorena Expósito, [31]Elia Vals-Pascual, [32]Angela Pecondon-Español, [33]Sergio Machín, [34]Eva Salgado-Perez, [1]Maria Celia-Erausquin, [35]Tarek Carlos Salman-Monte, [36]Raúl Menor-Almagro, [37]Alejandro Muñoz-Jimenez, [38]Irene Altabás-González, [39]Jorge Juan Fragío-Gil, [38]Jose María Pego-Reigosa*. *[1]Rheumatology, Hospital De Gran Canaria Doctor Negrin, Spain; [2]Rheumatology, Hospital Gregorio Marañón, Spain; [3]Rheumatology, Instituto De Investigación Sanitaria Galicia Sur, IRIDIS Group, Spain; [4]Rheumatology, University Hospital October 12, Spain; [5]Rheumatology, Donostia Unibertsitate Ospitalea, Spain; [6]Rheumatology, Hospital Regional Universitario de Málaga, Spain; [7]Rheumatology, Araba University Hospital, Spain; [8]Rheumatology, Hospital Marina Baixa, Spain; [9]Rheumatology, General University Hospital of Alicantet, Spain; [10]Rheumatology, Bellvitge University Hospital, Spain; [11]Rheumatology, Son Llàtzer Hospital, Spain; [12]Rheumatology, Hospital Sierra Llana, Spain; [13]Rheumatology, Hospital de Sant Joan Despí Moisès Broggi, Spain; [14]Rheumatology, Germans Trias i Pujol Hospital, Spain; [15]Rheumatology, University Hospital of A Coruña, Spain; [16]Rheumatology, Hospital de La Princesa, Spain; [17]Rheumatology, Navarra Hospital Complex, Spain; [18]Rheumatology, University Hospital of León, Spain; [19]Rheumatology, Hospital Parc Taulí de Sabadell, Spain; [20]Rheumatology, Hospital Lugo, Spain; [21]Rheumatology, Marqués de Valdecilla University Hospital, Spain; [22]Rheumatology, Hospital Universitario Príncipe De Asturias, Spain; [23]Rheumatology, Puerta de Hierro Majadahonda University Hospital, Spain; [24]Rheumatology, Ramón y Caja Hospital, Spain; [25]Rheumatology, Infanta Sofia University Hospital, Spain; [26]Rheumatology, La Paz University Hospital, Spain; [27]Rheumatology, Hospital Clínico Universitario Virgen de la Arrixaca, Spain; [28]Rheumatology, Salamanca University Hospital, Spain; [29]Rheumatology, Basurto University Hospital, Spain; [30]Rheumatology, Hospital Universitario De Canarias, Spain; [31]Rheumatology, Doctor Peset University Hospital, Spain; [32]Rheumatology, Miguel Servet University Hospital, Spain; [33]Rheumatology, Hospital Materno Infantil De Gran Canaria, Spain; [34]Rheumatology, Complexo Hospitalario Universitario De Ourense, Spain; [35]Rheumatology, Hospital Del Mar, Spain; [36]Rheumatology, Hospital Universitario De Jerez, Spain; [37]Rheumatology, Virgen Del Rocío University Hospital, Spain; [38]Rheumatology, Complejo Hospitalario Universitario De Vigo, Spain; [39]Rheumatology, La Fe University and Polytechnic Hospital, Spain*

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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