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ANTI-CARBAMYLATED PROTEIN ANTIBODIES' LEVELS ARE NEGATIVELY CORRELATED WITH CIRCULATING EFFECTOR T-CELLS IN A COHORT OF PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

^{1,2}Silvia Piantoni, ¹Ilaria Cavazzana, ¹Francesco Poiatti, ¹Stefania Masneri, ^{1,2}Roberta Ottaviani, ^{1,2}Micaela Fredi, ^{1,2}Franco Franceschini. ¹Rheumatology and Clinical Immunology Unit, ASST Spedali Civili Brescia, Brescia; ²Dept. of Clinical and Experimental Sciences, University of Brescia, Brescia, Italy

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Background/Purpose Anti-carbamylated protein antibodies (anti-CarP) were detected in a large cohort of patients with Systemic Lupus Erythematosus (SLE) in correlation with erosive arthritis but not with disease activity indexes. In animal models, T-cells may be activated by carbamylated epitopes playing a role in the development of arthritis. The imbalance between circulating regulatory (Treg) and CD28- effector T-cells was described in active SLE patients, explaining its involvement in disease's pathogenesis. Actually, no data are available about the possible correlation with these T-cell subpopulations and anti-CarP levels in SLE.

Methods Eight SLE patients with a median (10th-90th percentile) SLEDAI-2K=0 (0-4), anti-dsDNA levels=34.1 (15.6-427.4) UI/ml (nv<7), SDI=1 (0-1.3) were enrolled. Serum anti-CarP levels were evaluated using a home-made ELISA (nv<340 AU/ml) and peripheral blood T cell immunophenotyping was done using Flow Cytometry (Beckman Coulter). Treg were defined as CD4+CD127lowCD25high T-cells.

Results Enrolled patients showed levels of anti-CarP=189.38 (93.5-341.1) AU/ml, Treg=2.2 (% of CD4+), CD4+CD28- =7.7 (4.8-22.5) (% of CD4+) and CD8+CD28- =30.3 (17.2-35.6) (% of CD8+). Analyzing possible correlations among different T-cell subtypes and anti-CarP levels, a significant inverse correlation was found between these autoantibodies and CD4 +CD28- T cells ($r=-0.8$, $p<0.01$; Spearman rank correlation). No correlations were found between autoantibodies and other T-cell subpopulations or disease activity/damage indexes.

Conclusions In a small cohort of patients with serologically active SLE, anti-CarP autoantibodies were found as negatively correlated to circulating CD4+CD28- T-cells, which were described in association with disease damage, independently of age, gender, disease duration and activity. This suggest a potential role of anti-CarP as marker of SLE with a minor extent of T-cell activation and, consequently, with a possible better prognosis.

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THE COMBINED TYPE-I INTERFERON AND NEUTROPHIL GENE SCORES IDENTIFY HIGHLY ACTIVE SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS AND PERFORMED BETTER THAN CLASSICAL SEROLOGICAL MARKERS

¹François Chasset, ²Camille Ribi, ³Marten Trendelenburg, ⁴Uyen Huynh-Do, ¹Pascale Roux-Lombard, ⁵Delphine S Courvoisier, ¹Carlo Chizzolini. ¹Dept. of Pathology and Immunology, School of Medicine University of Geneva, Geneva; ²Division of Immunology and Allergy, University Hospital Center of Lausanne, Lausanne; ³Dept. of Biomedicine and Division of Internal Medicine, University Hospital of Basel, Basel; ⁴Division of Nephrology and Hypertension, Inselspital, Bern University Hospital, Bern; ⁵Division of Rheumatology, University Hospital and School of Medicine, Geneva, Switzerland

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Background In SLE, heterogeneous clinical expression and activity may reflect diverse pathogenic and/or effector

mechanisms. We dwelled into SLE heterogeneity by assessing the expression of three gene sets representative of type I interferon (IFN-I), neutrophil-(PMN) and plasmablast-(PB) signatures in a well characterized, multidisciplinary cohort of SLE patients. We further assessed whether individual gene products could be representative of these three signatures.

Methods Whole blood, serum and clinical data were obtained from 140 SLE individuals. Gene expression was assessed by NanoString® technology, using a panel of 37 probes allowing the computation of six IFN-I, one PMN and one PB scores. Protein levels were measured by ELISA.

Results High IFN-I gene expression was found in 45 to 50% of SLE individuals, depending on the score used. All 6 IFN-I scores were significantly associated with active skin involvement and 2 of 6 with arthritis. Interferon-induced GTP-binding protein Mx1 (MX1) correlated with IFN-I score ($p<0.0001$) and was associated with a similar clinical phenotype. High PMN gene expression was found in 25% of individuals in association with SLE fever, serositis, leukopenia and glucocorticoid use. PB gene expression was highly influenced by immunosuppressant agents with no association with SLE features. The combined IFN-I and PMN gene expression was significantly associated with high disease activity and outperformed anti-dsDNA, anti C1q and complement levels to predict SLE activity.

Conclusions The IFN-I and PMN gene scores segregate with distinct SLE clinical features and their joint expression identify high disease activity. MX1 protein levels perform similarly to IFN-I gene expression.

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ANTI-NEURONAL ANTIBODIES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: PREVALENCE, CLINICAL ASSOCIATIONS AND SEARCHING FOR A SPECIFIC TARGET

¹Elisabetta Chessa, ¹Maria Maddalena Angioni, ¹Silvia Pinna, ¹Mattia Congia, ¹Alberto Floris, ²Elias Manca, ²Giulia Corda, ³Lorena Loreface, ²Gian Luca Ferri, ²Cristina Cocco, ¹Alessandro Mathieu, ¹Alberto Cauli, ¹Matteo Piga. ¹Reumatologia, Policlinico Universitario AOU e Università di Cagliari, Cagliari; ²NEF-Lab, Dipartimento di Scienze Biomediche, Università di Cagliari, Cagliari; ³Neurologia, Ospedale Binaghi, ATS, Italy

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Background The aim of this study was to evaluate the prevalence and clinical significance of Anti-neuronal antibodies (AnAb) in a cohort of neuropsychiatric (NP) Systemic Lupus Erythematosus (SLE) patients.

Methods Overall, 28 SLE patients (age 45.6 ± 14.4 years) with inflammatory neuropsychiatric syndromes, classified according to existing criteria (1, 2) were enrolled and serum samples were collected before starting treatment for the specific NP event; 41 consecutive SLE patients (44.2 ± 14.2 years) without NP involvement, 22 Multiple Sclerosis patients (37.9 ± 11.0 years) and 82 healthy subjects (44.8 ± 15.0 years) served as controls. Search for AnAb was performed by tissue-based immunohistochemistry assays (IHC) on male Sprague Dawley rat's brain sections and confirmed by cell-

based immunocytochemistry assays on SH-SY5Y (human neuroblastoma) cell cultures. The association between serum positivity for AnAb by IHC and a large panel of data (demographic, serologic, SLEDAI, conventional brain MRI, treatment) was investigated by univariate analysis. Multivariate models were fitted with covariates with $p < 0.05$ to identify factors independently associated with serum positivity for AnAb; $p < 0.05$ were considered statistically significant.

Results AnAb were detected in 23 (82.1%) NPSLE patients and in 16 (39.0%) SLE patients without NP involvement resulting in 82% specificity (95%CI 71%-90%) and 61% sensitivity (95%CI 48%-72%) in differentiating NPSLE from SLE without NP involvement. None of the sera from MS patients (0%) and healthy subjects (0%) showed AnAb. Serum AnAb by IHC were independently associated with NPSLE ($p < 0.01$) and higher SLEDAI ($p < 0.01$). No association with specific NPSLE syndrome and brain conventional MRI abnormalities was identified.

Conclusion AnAb are significantly more frequent in patients with NPSLE than SLE. Further studies are needed to identify the unknown neuronal antigens targeted by AnAb in SLE patients.

P18 PRESENCE OF RHEUMATOID FACTOR WAS ASSOCIATED WITH A DECREASED RISK OF LUPUS NEPHRITIS IN PATIENTS WITH JUVENILE SYSTEMIC LUPUS ERYTHEMATOSUS

^{1,2}Yasmin Mahfouz, ¹Anastasia Vasiliki Madenidou, ^{1,2}Oliver Chang, ^{1,2}Farah El-Sharnoubi, ¹Charlene Foley, ¹Coziana Ciurtin. ¹Centre for Adolescent Rheumatology, University College London, London; ²University College London Medical School, London, UK

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Background Estimated 10- 20% of all patients with systemic lupus erythematosus (SLE) develop clinical disease before the age of 18 years and are therefore classified as juvenile-onset SLE (JSLE). JSLE is characterised by a higher prevalence of lupus nephritis, compared to adult-onset SLE. Chronic kidney disease (CKD) refers to a state of irreversible kidney damage and/or reduction of kidney function that is associated with progressive loss of function over time. Lupus nephritis does not always lead to CKD. However, when it does it is associated with increased morbidity and mortality.

Objectives We aimed to identify clinical and laboratory predictors of CKD development in JSLE patients by comparing the baseline characteristics of JSLE patients with and without CKD to ascertain if there are any significant differences between the two groups.

Methods This is a single-centre retrospective study, who included patients reviewed in our young adult and adolescent clinics. Mann-Whitney U or Chi-square test were performed to compare the characteristics between the patients with and without CKD. We used the Pearson's (r) or Kendall's τ (tau) correlation to examine if there is any association between the CKD and the baseline characteristics.

Results We identified 44 JSLE patients, out of which 17 (39%) fulfilled the diagnostic criteria for CKD at their last clinical review. The stages of CKD varied from 2 to 5. All patients with CKD also had lupus nephritis, while 5/44 patients (11%) had lupus nephritis without CKD. The baseline characteristics are detailed in the table 1 below. There were statistically significant differences in the treatments used for patients with and without CKD. As expected, the highest

Abstract P18 Table 1 Baseline characteristics of JSLE patients in the Arthritis UK Centre for Adolescent Rheumatology

	CKD	Without CKD	P
Number of patients, n (%)	17 (39%)	27 (61%)	
Female, n (%)	14 (80%)	23 (85.2%)	$p=0.47$
Age at diagnosis, years	12 (10- 11)	12 (11- 15)	$p=0.62$
Disease duration, years	12 (8- 14)	12 (7- 13)	$p=0.78$
Highest dsDNA	215 (34- 644)	32 (6.8- 104)	$p=0.03^*$
SLEDAI at last assessment	2 (0- 4)	0 (0- 2)	$p=0.33$
Arthritis, n (%)	13 (76%)	16 (59%)	$p=0.24$
Rituximab, number of courses	1.5 (0- 2.3)	0 (0- 1)	$p=0.04^*$
Mycophenolate Mofetil, months	44 (21- 96)	17 (0- 70)	$p=0.04^*$
Steroids, months	48 (37- 82)	25 (11- 50)	$p=0.03^*$

Numbers are medians (interquartile ranges) unless otherwise stated.

* $p < 0.05$ is significant

dsDNA levels were higher in patients with CKD ($p=0.03$). There was also a positive moderate correlation ($\rho=0.32$) between raised levels of dsDNA and the development of CKD ($p=0.008$). We also found a negative moderate correlation ($\tau=-0.439$) between the presence of RF and CKD ($p=0.04$).

Conclusion Acknowledging the limitations posed by this small study, we identified a negative moderate correlation between the presence of RF and CKD, which has also been reported in the literature before. We cannot conclude that RF exerts a protective effect against renal disease in SLE, because of the many confounders that might account for a decreased RF in JSLE. Further research using a large JSLE cohort enabling multivariate logistic regression is recommended. DsDNA antibody levels are a measure of disease activity in lupus nephritis and therefore this might explain why patients who developed CKD were noted to have higher anti-dsDNA levels, in comparison with the patients who did not develop CKD.

P19 AUTOANTIBODY PROFILE ANALYSIS IN SLE PATIENTS

¹Marta De-la-Rubia-Navarro, ¹Elena Grau-García, ¹Samuel Leal-Rodríguez, ¹Cristóbal Páez-Perales, ¹Cristóbal Alcañiz-Escandell, ¹Inés Cánovas-Olmos, ¹Inmaculada Chalmeta-Verdejo, ¹Jorge Juan Frago-Gil, ¹Roxana González-Mazarío, ¹Luis González-Puig, ¹José Ivorra-Cortés, ¹Isabel Martínez-Cordellat, ¹Carmen Nájera-Herranz, ¹Rosa Negueroles-Albuxech, ¹José Eloy Oller-Rodríguez, ¹Francisco Miguel Ortiz-Sanjuán, ¹Elvira Vicens-Bernabeu, ²Daniel Hervás-Marín, ³Meritxel Fernández Matilla, ³Nagore Fernández-Llanio Comella, ³Juan Antonio Castellano Cuesta, ¹José Andrés Román-Ivorra. ¹Rheumatology Dept., HUP-La-Fe, Valencia; ²Biostatistics Unit, IIS-La-Fe, Valencia; ³Rheumatology Section, Hospital-Arnau-de-Vilanova, Valencia, Spain

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Background/Purpose In Systemic Lupus Erythematosus (SLE) the presence of some autoantibodies is related to specific clinical manifestations. We aimed to define SLE patient groups according to an autoantibody profile and to analyze the correlation of these profiles to clinical manifestations.

Methods A cross-sectional observational study of SLE (SLICC 2012 criteria) was conducted. A clinical and analytical evaluation was performed. Clinical manifestations were described according to RELESSER study.

We selected 8 autoantibodies to classify SLE patients: anti-dsDNA, anti-Sm, anti-RNP, anticardiolipin IgG/IgM (aCL IgG/M), anti- β 2microglobulin IgG/IgM ($\alpha\beta$ 2M IgG/M), lupus anti-coagulant (LA), anti-Ro and anti-La. Immunological profiles