Table 2. Response to first TT and drug survival at 12 months (Δ= M12-M0)									
	RA (n=1602)			AxSpA (n=1306)			PsA (n=493)		
	M	F	p	M	F	p	M	F	p
Δ VAS pain (0-100)	-30.0 (-55.0; -15.0)	-35.0 (-55.0; -15.0)	0.566	-40.0 (-60.0; -20.0)	-40.0 (-60.0; -20.0)	0.109	-40.0 (-55.0; -25.0)	-40.0 (-60.0; -20.0)	0.364
Δ VAS fatigue (0-100)	-30.0 (-50.0; -10.0)	-30.0 (-55.0; -10.0)	0.734	-35.0 (-50.0; -20.0)	-35.0 (-55.0; -20.0)	0.738	-30.0 (-55.0; -5.0)	-40.0 (-60.0; -20.0)	0.174
Δ HAQ	-0.5 (-0.9; -0.1)	-0.5 (-0.9; -0.1)	0.463	-0.5 (-0.9; -0.1)	-0.5 (-0.9; -0.1)	0.929	-0.5 (-0.9; -0.1)	-0.5 (-0.9; -0.1)	0.232
Δ EQ-5D (0-1)	0.4 (0.0; 0.6)	0.5 (0.0; 0.7)	0.156	0.5 (0.1; 0.6)	0.5 (0.1; 0.6)	0.602	0.5 (0.1; 0.7)	0.5 (0.1; 0.7)	0.648
Δ CRP	-11.0 (-25.9; -1.0)	-7.9 (-20.7; -1.0)	0.091	-14.9 (-26.6; -7.2)	-10.0 (-19.5; -2.7)	< 0.001	-11.0 (-27.0; -3.1)	-8.2 (-20.0; -1.7)	0.003
Survival (%)	80 (76; 84)	76 (73; 78)	0.182	89 (87; 91)	79 (75; 83)	< 0.001	88 (84; 92)	83 (78; 88)	0.146

Acknowledgments: Supported by project 00023728 of Ministry of Health, CZ
Disclosure of Interests: Jakub Zavada Speakers bureau: Abbvie, UCB, Sanofi, Eli-Lilly, Novartis, Zentiva, Accord, Lucie Nekvindova: None declared, Karel Pavelka Consultant of: Abbvie, MSD, BMS, Eisai, Roche, UCB, Medac, Pfizer, Biogen, Speakers bureau: Abbvie, MSD, BMS, Eisai, Roche, UCB, Medac, Pfizer, Biogen, Pavel Horak Speakers bureau: Pfizer, Abbvie, Eli Lilly, Novartis, Roche, Sanofi, Jiří Vencovský: None declared
DOI: 10.1136/annrheumdis-2020-eular.2849

Validation of outcome measures and biomarkers

AB1234 MICRO-RNA 155 AND MIR-34A: POSSIBLE BIOMARKERS OF INFLAMMATORY BURDEN AND DISEASE ACTIVITY IN ANCA-ASSOCIATED VASCULITIS WITH RENAL INVOLVEMENT

D. Bruno¹, P. G. Cerasuolo¹, C. Di Mario¹, S. L. Bosello^{1,2}, L. Gigante¹, A. Musto¹, G. Vischini³, S. Costanzi³, S. Alivernini^{1,2}, B. Toluoso², G. Grandaliano^{3,4}, E. Gremese^{1,2}. ¹Division of Rheumatology, Università Cattolica del Sacro Cuore, Rome, Italy; ²Division of Rheumatology, Fondazione Policlinico Universitario A. Gemelli-IRCCS, Rome, Italy; ³Division of Nephrology, Fondazione Policlinico Universitario A. Gemelli-IRCCS, Rome, Italy; ⁴Division of Nephrology, Università Cattolica del Sacro Cuore, Rome, Italy

Background: Predicting clinical outcomes in ANCA-related glomerulonephritis remains a major challenge. To date, there is no reliable biomarker able to predict renal prognosis in patients with ANCA-associated vasculitis (AAV). Micro-RNA (miRNA) are non-coding RNAs involved in the fine tuning of immune cells biology and this epigenetic modulation associates with different phenotypes and prognosis in several diseases.

Objectives: To investigate the expression of miR-155 and miR-34a in kidney biopsies of AAV patients according with renal outcome.

Methods: Fifteen patients with AAV and renal involvement (mean age 63.0 ±13.3 years, disease duration 4.9±2.2 months), who underwent renal biopsy. Demographic, clinical and autoimmune parameters were recorded for each patient. Each kidney biopsy was classified according to the Berden Classification, Risk group (according to the ANCA Renal Risk Score) and the chronicity Classification of the Mayo Clinic's proposed score.

MiR-155 and miR-34a expression was investigated on kidney biopsy tissue using the miRNeasy FFPE kit (Qiagen). The quantitative expression of miR-155, miR-34a and housekeeping gene U1, used as control, was assessed by Real Time-PCR. MiR-155 and miR-34a expression was correlated with histopathological and clinical-laboratory parameters.

Each patient was followed for 12 months and renal outcome was considered according to KDIGO CKD Classification. Markers of inflammation (ESR, CRP) and urine analysis data were recorded at baseline and after 12 months.

Results: Six (40%) patients were p-ANCA positive and 9 (60%) c-ANCA positive. Eight patients (53%) also had pulmonary involvement. The mean baseline GFR was 30.7±28.8 ml/min/1.73 m² and 10 patients (66%) showed an active urinary sediment.

At disease onset, the mean expression of miR-155 was 9.5±21.1, while the expression of miR-34a was 13.1±46.2. Considering the autoimmune profile, kidney tissue of p-ANCA positive patients was enriched of miR-155 (19.6±30.6 fold) compared to c-ANCA positive patients (1.9±2.9 fold; p=0.001). Particularly, considering the renal function, kidney tissue of patients with greater impairment of renal function (KDIGO stage 5) was enriched of miR-155 (21.5±38.3 fold) compared to patients with less renal impairment (KDIGO stage 1-4) (4.72±8.16 fold, p=0.004).

Tissue expression of miR-155 and miR-34a did not correlated with the above-mentioned histopathological classifications. After 12 months from kidney biopsy, 3(20%) patients had a worsening of renal function, 5 (33%) still presented elevated markers of inflammation and 3 (20%) still had proteinuria at urine analysis. At baseline, kidney tissue of patients with higher CRP plasma levels and proteinuria at follow-up presented higher

expression of miR-155 (p=0.002 and p=0.001), whereas no significant differences were found about miR-34a kidney tissue expression.
Conclusion: MiRNAs may play a potential role in the pathogenesis of ANCA-related glomerulonephritis. MiR-155 kidney enrichment seems to mirror the disease inflammatory burden and activity at the onset and after 12 months representing a possible biomarker in ANCA vasculitis with renal involvement. This finding may represent the basis for further studies on miRNA expression in blood samples, aiming to identify a non-invasive biomarker of kidney damage, predicting disease's relapses and patients' prognosis.

References:
[1] Renauer et al, Clin Rev Allergy Immunol. 2016
Disclosure of Interests: Dario Bruno: None declared, Pier Giacomo Cerasuolo: None declared, Clara Di Mario: None declared, Silvia Laura Bosello Speakers bureau: Abbvie, Pfizer, Boehringer, Laura Gigante: None declared, Alessia Musto: None declared, Gisella Vischini: None declared, Stefano Costanzi: None declared, Stefano Alivernini: None declared, Barbara Toluoso: None declared, Giuseppe Grandaliano: None declared, Elisa Gremese Speakers bureau: Abbvie, BMS, Celgene, Janssen, Lilly, MSD, Novartis, Pfizer, Sandoz, UCB
DOI: 10.1136/annrheumdis-2020-eular.6011

AB1235 SALIVARY LEVEL OF CALPROTECTIN (MRP 8/14) AS A BIOMARKER FOR SJÖGREN SYNDROME – A PROSPECTIVE OBSERVATIONAL STUDY

P. Chebbi¹, J. Kabeer¹, P. Sandhya¹, M. Gowri¹, D. Danda¹. ¹CMC Vellore, Tamil Nadu, India

Background:
Objectives: Calprotectin (MRP 8/14) is secreted by neutrophils and monocytes in an inflammatory milieu(1). Increased levels of calprotectin in blood and faeces has been previously shown in patients with primary Sjogren's syndrome(pSS)(2). We hypothesised Calprotectin in saliva may therefore potentially reflect inflammation in salivary glands. In the present study, we aimed to assess the utility of salivary calprotectin as a biomarker in pSS and to evaluate the potential association of calprotectin with clinical features, laboratory markers and disease activity indexes in pSS.

Methods: Consecutive patients attending rheumatology clinic between September 2016 and July 2017 of age more than 18 years fulfilling either American European Consensus Group (AECG) 2002 or 2016 American College of Rheumatology/European League Against Rheumatism Classification Criteria for Primary Sjogren's Syndrome, constituted the case group. Patients with sicca non-pSS and rheumatoid arthritis patients satisfying ACR/EULAR criteria 2010 were recruited as disease controls. Age and sex-matched healthy subjects were recruited as a controls. Patient history, signs and symptoms, laboratory investigations, ESSDAI and ESSPRI scoring were noted for cases and disease controls. Unstimulated saliva was collected by the spitting method. Salivary calprotectin levels were quantified by commercially available ELISA kit(R&D Systems, MN, USA).

Results: A total of 42 pSS cases (41.5911.21 years, 40 women), 40 disease controls (47.629.81 years, 39 women) and 30 healthy controls (36.10.65 years, 29 women) were recruited. In pSS and disease controls, the median levels of salivary calprotectin [38.5(81) and 52(69.5 ng/ml)] were significantly higher compared to healthy controls [26.5(29) {p=0.001}]. Salivary calprotectin levels in pSS was positively associated with oral symptoms and negatively associated with immunosuppression of 1 year and more(P=0.04 and 0.03 respectively),but not ESSDAI,ESSPRI scores or lab parameters.

Conclusion: Salivary calprotectin was elevated in pSS compared to healthy subjects and was found to be positively associated with oral symptoms and with immunosuppressive treatment.

References: [1]Stroncek DF, Shankar RA, Skubitz KM. The subcellular distribution of myeloid-related protein 8 (MRP8) and MRP14 in human neutrophils. J Transl Med. 2005 Sep 28;3:36.

[2] Fagerhol MK, Larsen A, Brun JG, Hammer HB, Angel K, Kvien TK, et al. Large molecular size EDTA-resistant complexes containing S100A12, ERAC, in serum during inflammatory conditions. Scand J Clin Lab Invest. 2012 Apr;72(2):129–36.

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2020-eular.6216

AB1236 KL-6 AS A BIOMARKER FOR SJÖGREN SYNDROME-ASSOCIATED INTERSTITIAL LUNG DISEASE: METHODOLOGY MATTERS

Y. H. Chiu¹, C. C. Lu¹, F. C. Liu¹, S. E. Tang², S. J. Chu¹, S. Y. Kuo¹, H. C. Chen¹. ¹Tri-Service General Hospital, National Defense Medical Center, Division of Rheumatology/Immunology/Allergy, Taipei, Taiwan, Republic of China; ²Tri-Service General Hospital, National Defense Medical Center, Division of Thoracic Medicine and Critical Care, Taipei, Taiwan, Republic of China