Table 1

	Negative n=35	CCP n=65	CCP_Carb n=36	CCP_Carb_Acet n=76
Female (%)	74.1	72.3	66.75	52.6
Age, mean	64.1	54.9	60.5	56.7
Disease duration, mean, months	6.6	14.7	11.7	13.2
Smoked (%)	54.3	67.7	77.8	67.1
DAS28-ESR, mean (complete data)	5.5 (30)	4.7 (56)	5.1 (30)	5.2 (64)
Radiographic Scores				
Baseline Erosion score, mean	2.9	2.8	7.2	3.3
Baseline JSN score, mean	3.3	3.4	6.6	3.5
Baseline Total SvH Score, mean	6.2	6.2	13.8	6.8
Mean change in Total SvH score over 12 months	0.5	0.5	1	1.8

with anti CCP antibodies (n=65), antibodies to anti CCP and anti Carb (n=36), antibodies to all three PTM (n=76) and antibody negative patients (n=35). Overall there was low radiographic progression in this sample of the SERA cohort. EULAR non-responders had greater progression compared to good responders (Least square mean difference in SvH over 12 m of 1.6, p=0.019).
Baseline SvH erosion scores are 4.4 points higher in the CCP_Carb group compared to CCP alone (p=0.009) (Table 2 + Fig 1).

Table 2

Comparison	LS mean difference in baseline erosion score	P value
CCP_Carb vs CCP	4.4	0.009
CCP_Carb vs CCP_Carb_Acet	3.9	0.017
CCP_Carb vs Negative	4.3	0.026
Comparison	**LS mean difference in SvH change over 12 months**	**P value**
CCP_Carb_Acet vs CCP	1.3	0.016
CCP_Carb_Acet vs Negative	1.3	0.046

Total SvH 12m progression is 1.3 points higher in the triple positive group compared to the CCP group alone (p=0.016) (Table 2 + Fig 2). Total SvH progression is also higher when comparing the triple positive to the negative group (p=0.046).

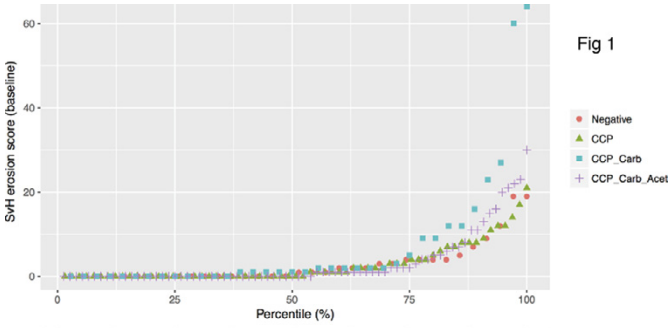

Fig 1

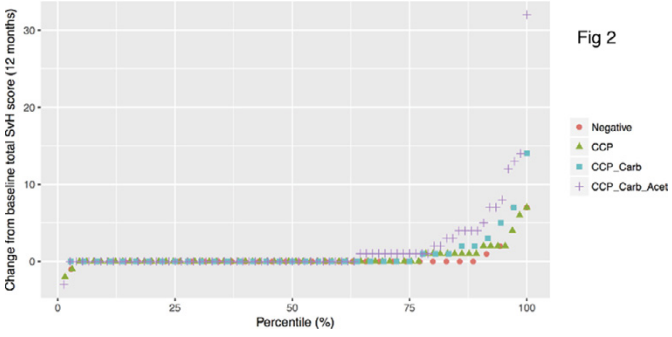

Fig 2

Conclusions: RA patients with antibodies to citrullinated peptides only have lower baseline erosions and less radiographic progression over 12m compared to those with a wider autoantibody repertoire. Baseline differences in erosion suggest that these antibodies may be pathogenic during the pre-RA disease process. Radiographic progression increases with autoantibody repertoire suggesting ongoing immune activation.
Disclosure of Interest: J. Nijjar: None declared, F. Morton: None declared, A. Gilmour: None declared, C. Paterson: None declared, H. Bang Employee of: Orgentec Diagnostike GmbH and holds patent for mutated citrullinated vimentin as diagnostic tool, D. van der Heijde Employee of: Director of Imaging Rheumatology, K. Raza: None declared, C. Buckley: None declared, D. Porter Grant/research support from: Pfizer co-funded the SERA cohort, I. McInnes: None declared
DOI: 10.1136/annrheumdis-2017-eular.2844

THU0112 STRESS AND DEPRESSION IN PATIENTS WITH EARLY INFLAMMATORY POLYARTHRITIS: NATURAL HISTORY AND ASSOCIATIONS WITH DISEASE ACTIVITY, DISABILITY AND PAIN OVER FIVE YEARS

J.M. Gwinnutt[1], D.P.M. Symmons[1,2], A.J. MacGregor[3,4], J.R. Chipping[3,4], T. Marshall[3,4], M. Lunt[1], S.M.M. Verstappen[1]. *[1]Arthritis Research UK Centre for Epidemiology, University of Manchester; [2]NIHR Manchester Musculoskeletal Biomedical Research Unit, Central Manchester University Hospitals NHS Foundation Trust, Manchester; [3]Norwich Medical School, University of East Anglia; [4]Rheumatology Department, Norfolk and Norwich University Hospitals NHS Trust, Norwich, United Kingdom*

Background: Stress and depression are common in patients with inflammatory polyarthritis (IP). There is little research on long term patterns of depression and stress or how these variables relate to disease activity, disability and pain.
Objectives: To describe the natural history of stress and depression over five years and to assess the association of baseline, one year prior and current disease activity, disability and pain with longitudinal stress and depression.
Methods: Patients recruited to the Norfolk Arthritis Register (NOAR) (inclusion criteria: ≥2 swollen joints for ≥4 weeks) from 2005–2008 were included in this analysis. Demographics, medication use, 51 swollen/tender joint counts (SJC51/TJC51), pain visual analogue scale, HAQ, comorbidities and the Arthritis Impact Measurement Scales 2 (AIMS2) depression and stress subscales (range 0–10; high score = worse health status) were collected at baseline and years 1, 2, 3 and 5. Rheumatoid factor (RF), anti-cyclic citrullinated peptide antibodies (anti-CCP2) and C-reactive protein (CRP) were measured in baseline blood samples. ACR/EULAR 2010 RA criteria were applied to baseline characteristics. Depression and stress over five years were described using descriptive statistics. Multivariate random effects models were applied to assess the association of baseline disease activity (SJC51/TJC51), pain and disability with depression and stress over time adjusting for baseline age, gender, RF, anti-CCP2, CRP, sDMARD use, comorbidities, depression and stress. Similar methods were used to assess one year prior and current disease activity, pain and disability's association with stress and depression. Missing data were imputed using multiple imputation.
Results: 509 patients were included (median (IQR) age: 57 (45, 68) years; 321 (63.1%) female; 305 (59.9%) ACR/EULAR RA). Baseline median (IQR) depression and stress were 2.5 (1.5, 4.5) and 4.0 (2.5, 5.5) respectively and remained constant over five years. Baseline SJC51, TJC51, pain and HAQ were not independently associated with depression or stress over five years. Current HAQ and pain, but not SJC51/TJC51, were independently associated with depression and stress (per unit increase in HAQ: depression β 0.83, 95% CI 0.69, 0.97; stress β 0.76, 95% CI 0.61, 0.90; per 1cm increase in pain: depression β 0.09, 95% CI 0.06, 0.12; stress β 0.09, 95% CI 0.05, 0.12). Higher HAQ was independently associated with increased depression and stress one assessment later (per unit increase in HAQ: depression β 0.21, 95% CI 0.09, 0.32; stress β 0.21, 95% CI 0.10, 0.33) but not pain, SJC51 or TJC51.
Conclusions: There were no associations between measures of disease activity and depression or stress. Prospectively higher HAQ scores were associated with worse psychological health a year later. This may have implications for holistic management of IP.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.3432

THU0113 SERUM CALPROTECTIN MAY REFLECT INFLAMMATORY ACTIVITY IN PATIENTS WITH ACTIVE RHEUMATOID ARTHRITIS DESPITE NORMAL C-REACTIVE PROTEIN

J. Hurnakova[1], H. Hulejova[1], J. Zavada[1], M. Komarc[2], L. Andres Cerezo[1], H. Mann[1], J. Vencovsky[1], K. Pavelka[1], L. Senolt[1]. *[1]Institute of Rheumatology, First Faculty of Medicine, Charles University, Prague, Czech Republic; [2]Department of Methodology, Faculty of Physical Education and Sport, Charles University, Prague, Czech Republic*

Background: About a half of patients with rheumatoid arthritis (RA) have normal or low C-reactive protein (CRP) levels[1–3]. Calprotectin is a promising and probably more specific biomarker of disease activity than conventionally used acute phase reactants.
Objectives: The aim of this study was to analyse levels of serum calprotectin in RA patients with clinically active disease and with low CRP (<10 mg/L).
Methods: A total of 160 RA patients and 32 healthy subjects were enrolled in this study. All patients underwent clinical examination (DAS28). The levels of calprotectin were analyzed in patients with moderate to high disease activity with low CRP levels and in healthy subjects. The discriminatory capacity of calprotectin to identify clinically active patients in spite of normal CRP was assessed using ROC curves.
Results: Out of all RA patients, 74/160 (46.3%) had low CRP and were in remission or had low activity at the same time. However, 51/160 (32%) had low CRP levels despite moderate to high disease activity according to DAS28. In these patients, calprotectin levels were significantly higher than in patients with low CRP in remission or with low disease activity at the same time (mean 2.7±1.5 vs. 2.1±1.2 μg/mL, p=0.043) and differed from that in healthy subjects (mean 2.7±1.5 vs. ± 1.9±1.2 μg/mL, p=0.011) (Figure 1). The discriminatory capacity for calprotectin to distinguish clinically active vs. inactive patients in spite of low CRP