Methods CD14-positive monocytes from healthy donors were cultured in presence of M-CSF, IFN- γ , IL-4 or IL-10. The expression of the *PADI2* and 4 genes was measured by RT-qPCR and the expression of PAD2 and 4, evaluated by immunoblotting of total cell extracts.

Results *PADI2* and 4 mRNAs are expressed in monocytes while PAD2 and 4 proteins are also immunodetected in these cells. In the different M Φ subsets, *PADI2* mRNAs are less detected than in the related monocytes except for the IFN- γ M Φ subset where it is detected at a higher relative rate. By contrast, *PADI4* gene expression is suppressed in all M Φ subsets except the IFN- γ M Φ where *PADI4* mRNAs are weakly detected. Consistently with mRNA analysis, PAD2 is detected in all polarised M Φ and more strongly expressed in IFN- γ M Φ whereas PAD4 is no longer detectable except faintly for the IFN- γ M Φ .

Conclusions This study shows that PAD2 is expressed at various degrees in the four analysed M Φ phenotypes but PAD4, only in the IFN- γ M Φ , both enzymes being expressed in monocytes. This reinforces the hypothesis of a role for monocytes and M Φ in genesis of ACPA epitopes in the ST.

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Acknowledgements The technical assistance of Céline Thomann, Géraldine Offer and Imène Belhaouane is gratefully acknowledged.

Disclosure of interest None declared

P006

ANTI-RA33 (HNRNP-A2/B1) AUTOANTIBODIES ARE ASSOCIATED WITH THE THERAPEUTIC RESPONSE TO METHOTREXATE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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10.1136/annrheumdis-2018-EWRR2018.32

Introduction Besides the determination of rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA), anti-RA33 antibodies (which are directed to the nuclear antigen hnRNP-A2) could be of additional diagnostic and/or prognostic value in patients with rheumatoid arthritis (RA) because they are also found in RF/ACPA negative patients.¹

Objectives So far, published data on anti-RA33 antibodies refer only to the IgG isotype.² It was therefore the aim of this study to measure the prevalence of anti-RA33 IgG, IgM and IgA antibodies in patients with RA and to determine their potential prognostic value regarding prediction of response to treatment.

Methods To determine the diagnostic sensitivity and specificity of anti-RA33 subtypes sera from 255 RA patients, 258 disease controls and 100 healthy subjects were tested by a prototype anti-RA33 EliA (Thermo Fisher Scientific) for the presence of anti-RA33 IgG, IgA and IgM antibodies. ACPA and RF were routinely measured by EliA and nephelometry, respectively. All RA patients had initially been treated with conventional synthetic drugs (mostly methotrexate, MTX) and were subsequently treated with at least one TNF inhibitor (TNFi). Therapeutic responses to MTX and TNF blocking biologicals

were analysed in an inception cohort (n=104) who had started their DMARD therapy at our clinic. To define therapeutic responses the simplified disease activity index (SDAI) and American College of Rheumatology (ACR) responses were calculated.

Results Diagnostic specificity was >96% for all 3 anti-RA33 subtypes. Among the 255 RA patients, 11% tested positive for anti-RA33 IgG antibodies, 15% for IgM antibodies and 6% for IgA antibodies. Altogether, 62 patients (24%) had at least one type of anti-RA33 antibody: among these, 24 patients were RF-negative, 26 were ACPA-negative and 18 were RF/ACPA double negative. Thus, in 32 patients (13%), anti-RA33 was the only antibody specificity. Regarding responses to MTX therapy, the percentage of SDAI50 or ACR20 responders, respectively, was significantly higher (p=0.034 for SDAI50 and p=0.005 for ACR20) among anti-RA33 positive patients (with or without RF/ACPA) compared to anti-RA33 negative (but RF/ACPA positive) patients and RF/ACPA negative patients. Thus, 60% of the anti-RA33 positive patients as compared to 37% anti-RA33 negative patients showed a SDAI50 response; similar values were seen for ACR20 responses.

Conclusions Apart from their added diagnostic value anti-RA33 antibodies may have also prognostic value for prediction of therapeutic responses to MTX treatment. Therefore anti-RA33 antibodies may be taken into consideration as additional markers that might become helpful tools in therapeutic decision making.

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Disclosure of interest D. Sieghart: None declared, P. Studenic: None declared, M. Grundhuber Employee of: Thermo Fisher Scientific-Phadia GmbH, S. Swiniarski Employee of: Thermo Fisher Scientific-Phadia GmbH, A. Platzer: None declared, J. Smolen: None declared, G. Steiner: None declared

P007

IN RA, BECOMING SERONEGATIVE OVER THE 1ST YEAR OF DMARD TREATMENT DOES NOT TRANSLATE TO BETTER CHANCES OF DRUG-FREE REMISSION IN THE LONG-TERM

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10.1136/annrheumdis-2018-EWRR2018.33

Introduction Patients with RA harbour autoantibodies of various isotypes and directed against several post-translational modifications. Baseline seropositivity is a poor prognostic factor for sustained drug-free remission (SDFR). However, autoantibody levels may change and patients may become seronegative under treatment. It is unknown how often this happens, and whether decreasing levels or becoming seronegative early in disease, indicating disappearance of serological autoimmunity, improves chances of SDFR.

Objectives To longitudinally characterise the levels and presence of autoantibodies in RA patients and to investigate