containing target protein, we established stable MH7A cell lines overexpressing GFP-tagged PLCH2. We also conducted *in vitro* and *in vivo* experiments using a recombinant adenovirus expressing the full length form of PLCH2 or C2 domain of PLCH2 (C2-PLCH2).

Results Several C2 domain-containing proteins were identified whose over-expression resulted in reduced proliferation and NF- κ B activity of RA-FLS. Among those, we focused on PLCH2 in this study. PLCH2 levels were significantly decreased in RA-FLS and synovium than OA. PLCH2 and C2-PLCH2 suppressed cell invasion and migration, cytokines production, and matrix metalloproteinase secretion in RA-FLS and stable MH7A cell lines compared to control. PLCH2 and C2-PLCH2 sensitised RA-FLS to apoptosis *in vitro* and *in vivo* model of matrigel implants engrafted into immunodeficient mice. PLCH2 and C2-PLCH2 further decreased arthritis severity in collagen-induced arthritis (CIA) mouse model.

Conclusions Our results showed that invasive characteristics of RA-FLS and inflammatory arthritis of CIA are reduced by PLCH2 and C2 domain of PLCH2. Therefore C2 domain of PLCH2 may have therapeutic potential for RA.

REFERENCES

- Farah CA, Sossin WS. The role of C2 domains in PKC signalling. *Adv Exp Med Biol* 2012;**740**:663–83.
- Yáñez M, Gil-Longo J, Campos-Toimil M. Calcium binding proteins. *Adv Exp Med Biol* 2012;**740**:461–82.
- Friedrich R, Yehekel A, Ashery U. DOC2B, C2 domains, and calcium: A tale of intricate interactions. *Mol Neurobiol* 2010 February;**41**(1):42–51.
- Zhou Y, Sondek J, Harden TK. Activation of human phospholipase C- ϵ 2 by Gbetagamma. *Biochemistry* 2008 April 15;**47**(15):4410–7.
- Popovics P, Beswick W, Guild SB, Cramb G, Morgan K, Millar RP, Stewart AJ. Phospholipase C- η 2 is activated by elevated intracellular Ca(2+) levels. *Cell Signal* 2011 November;**23**(11):1777–84.

Acknowledgements This work was supported by grants (NRF-2014R1A2A1A11051360 and NRF-2015R1A5A2008833) from the National Research Foundation of Korea (NRF) funded by the Korean government (MSIP).

Disclosure of interest None declared

P038

RECENT THYMIC EMIGRANT T CELLS INCREASE IN RHEUMATOID PATIENTS TREATED WITH ABATACEPT

^{1,2}S Piantoni*, ¹F Regola, ¹A Tincani, ¹P Airò. ¹Rheumatology and Clinical Immunology Unit, Spedali Civili and University of Brescia; ²Department of Molecular and Translational Medicine, University of Brescia, Brescia, Italy

10.1136/annrheumdis-2018-EWRR2018.60

Introduction CD4⁺ T cells in rheumatoid arthritis (RA) display a peculiar restriction of the T-cell receptor (TCR) repertoire which compromises their ability to react to novel antigens.¹ We demonstrated that this process could be partially reverted by abatacept (ABA), which is a blocker of T lymphocyte co-stimulation, used in the treatment of RA.²

To better understand the mechanisms underlying this phenomenon, we speculated that ABA could influence the frequency of peripheral T cell subpopulations, even at early stages of maturation.

The homeostasis of the *naïve* T cell pool is maintained not only by thymus production, but also by mechanisms of peripheral replication involving TCR activation revealed by CD31 down-modulation. Indeed, CD4⁺CD45RA⁺CD31⁺ T cells have been described as *recent thymic emigrants* (RTE) newly produced by the thymus

and CD4⁺CD45RA⁺CD31⁺ T cells as *central naïve*, involved in a self-renewal peripheral process.

Objectives We aimed at profiling circulating *naïve* CD4⁺CD45RA⁺ T cells, by assessing their expression of CD31 (PECAM-1).³

Methods Thirty-one RA patients (median age [10°–90° percentile] 42 [25–64] years) were evaluated before and after 6 month of ABA therapy. Phenotypic analysis of peripheral blood T lymphocytes was made by flow-cytometry. Wilcoxon-signed rank test and Spearman rank correlation were used for the comparisons.

Results After ABA therapy, the absolute number of total T CD4⁺ increased from 780 [423–1351] to 1000 [658–1566] cells/mm³ (p=0.01). Total *naïve* CD4⁺ increased in percentage (33 [18–56] vs 40 [20–61]% of CD4⁺; p=0.02) and in absolute number (257 [82–568] vs 344 [62–689] cells/mm³; p=0.03). In parallel, the number of RTE increased in percentage (10.6 [2–26] vs 11.6 [3–25]% of CD4⁺; p=0.04) and in absolute number (51 [15–194] vs 110 [23–271] cells/mm³; p=0.01). The *central naïve* counterpart did not show significant variations in percentage (29 [23–40] vs 27 [20–38]% of CD4⁺; p=0.20) nor in absolute number (191 [41–404] vs 242 [76–526] cells/mm³; p=0.20). No correlation was found between age and RTE number at baseline.

Conclusions The number of total *naïve* T cells increases after therapy with ABA together with the number of RTE, suggesting a thymic output boost. Besides the peripheral effect in reducing the number of effector T cells which was showed by previous studies,⁴ ABA could have a role in promoting the immune reconstitution at the early stage of T cell development.

REFERENCES

- Wagner UG, et al. *PNAS* 1998.
- Imberti L, et al. *J Transl Med* 2015.
- Kohler S, et al. *Blood* 2009.
- Airò P, et al. *Trends Mol Med* 2013.

Disclosure of interest None declared

P039

ROTATOR CUFF CALCIFIC TENDINOPATHY: CHONDROCYTE-LIKE CELLS SURROUNDING CALCIFIC DEPOSITS EXPRESS TNAP AND ENPP1, TWO KEY ENZYMES OF THE MINERALIZATION PROCESS

^{1,2}C Darrieurt-Laffite*, ^{1,2}A Najm, ²T Garraud, ²P Layrolle, ²F Blanchard, ^{1,2}B Le Goff. ¹Rheumatology, CHU de Nantes; ²INSERM U1238, Phy-Os Laboratory, Nantes, France

10.1136/annrheumdis-2018-EWRR2018.61

Introduction Calcific tendinopathy is a frequent cause of chronic painful shoulders. It is caused by deposits of carbonated apatite in rotator cuff tendons. Although the disease is frequent, molecular and cellular mechanisms involved in this pathological mineralization process are not currently identified. **Objectives** The objective of the study was to analyse calcified tendon samples to understand the organisation of the deposits and to characterise the cells potentially involved in their formation.

Methods Samples were collected from cadaveric subjects. They were fixed in formalin 4%, decalcified in EDTA, dehydrated and embedded in paraffin. Some samples were not decalcified to allow a better characterisation of the calcific deposits. Hematoxylin and eosin (HE), Safranin O/Fast Green (SO/FG), Von Kossa (no decalcified samples) and Tartrate-Resistant Acid Phosphatase (TRAP)