anti-proliferation effects of different treatments. To further study the potential mechanism, TNF- α -induced *in vitro* model was applied. With different treatments, cell proliferation was detected using MTS, meanwhile, cell cycle distribution and apoptosis were examined by flow cytometric analysis. Western blotting and real-time quantitative PCR were conducted to evaluate many molecular targets involved in interested pathways like COX-2/TxA2 pathway and AKT/FOXO3a pathway.

Results: The paw swelling volume and histological data indicate that 18 β -GA administration attenuates arthritis severity in rats with CIA. Lower level of IL-1 β , IL-6, and TxB2 were observed in serum of 18 β -GA group as compared with model group. In addition, synovial immunohistochemistry data shows that 18 β -GA decreased about half of PCNA intensity induced by collagen. However, *in vivo*, all data exhibited no significant differences among groups with monotherapy and combination therapy. *In vitro*, 18 β -GA inhibited the mRNA and protein levels of COX-2 and TxAS that induced by TNF- α in MH7A cell line. Both p-JNK and NF- κ B1 (p50) were inhibited by 18 β -GA as well as TxAS siRNA transfection. Moreover, 18 β -GA inhibited MH7A proliferation in a time- and dose- dependent manner from MTS assay. Flow cytometric analysis revealed that 18 β -GA induced cell apoptosis and caused G1-phase cell cycle arrest. Finally, AKT and FOXO3a were predominantly phosphorylated by TNF- α , whereas such effect was blocked by 18 β -GA treatment.

Abstract AB0127 – Figure 1

Conclusions: This study has for the first time shown that 18 β -GA has an inhibitory role in synovial cell inflammation and proliferation, which is, at least in part, dependent on the regulation of COX-2/TxA2 pathway and AKT/FOXO3a pathway. Thus, 18 β -GA should be regarded as a new potential drug candidate for RA therapy.

REFERENCES:

- [1] Korcowska I. Rheumatoid arthritis susceptibility genes: An overview. *World J Orthop* 2014;5(4):544–9.
- [2] Mahmoud AM, et al. Methotrexate hepatotoxicity is associated with oxidative stress, and down-regulation of PPARgamma and Nrf2: Protective effect of 18beta-Glycyrrhetic acid. *Chem Biol Interact* 2017;270:59–72.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.5898

AB0128 CXCL1, BUT NOT AUTO-ANTIBODIES OR CD4+CCR6+ MEMORY TH CELLS WITHIN BLOOD, IS A MARKER TO DIFFERENTIATE MICE INTO COLLAGEN INDUCED ARTHRITIS POSITIVE OR NEGATIVE PRIOR TO CLINICALLY MANIFEST DISEASE

M. Molendijk, A.M. Mus, P.S. Asmawidjaja, E. Lubbers, Erasmus MC, Rotterdam, Netherlands

Background: There is currently a knowledge gap on early pathogenesis prior to Rheumatoid Arthritis (RA) diagnosis. Additionally, current medication available for RA treatment has not been developed for prevention. Collagen induced arthritis (CIA) could aid in extending knowledge on early RA pathogenesis and testing the preventive effects of medicines.

Objectives: In this study we sought a marker that can differentiate mice prior to clinically manifest disease into their future CIA status with the aim to facilitate research into early disease processes and preventive treatment strategies.

Methods: Blood was obtained at time points prior (days 12 and 19) and after clinically manifest disease (days 27 and 35) during CIA. Antibodies against bovine and mouse collagen type II (mCII) were measured from plasma by ELISA. CD4+CCR6+ memory Th cells as well as other T cell types were determined in

blood. Cytokines and chemokines were detected in plasma by Luminex. Mice were divided into CIA negative and CIA positive groups based on CIA score reached on day 35.

Results: Antibodies against mCII of the IgG2a isotype differed prior to clinically manifest disease but are not suitable as a differentiation marker. CD4+CCR6+ memory Th cells in blood differed only at day 35. The same holds for IL-6, TNF α and CXCL2. In contrast, CXCL1 differed prior to clinically manifest disease with an AUC significantly better (p=0.003) than random.

Conclusions: Here we identified CXCL1 as a marker that can differentiate mice prior to clinically manifest disease into CIA positive and CIA negative mice. This might help facilitate research into early disease processes and preventive pre-clinical treatment strategies.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.6554

AB0129 ASSESSMENT OF MORPHOLOGY OF THE EARLY AND LATE STAGE OF JUVENILE RHEUMATOID ARTHRITIS

M. Salokhiddinov¹, U. Anarmetova¹, G. Haydarova¹, I. Xodjanazarov². ¹Tashkent Medica Academy; ²Traumatology, Tashkent Medical Academy, Tashkent, Uzbekistan

Background: One of the current problems of modern rheumatology is chronic inflammatory diseases of the knee joint in children. With juvenile rheumatoid arthritis (JRA), an uncontrolled inflammatory process can lead to the formation of contractures and deformities of the limbs

Objectives: Our aim is to study morphology of the early and late stage of juvenile rheumatoid arthritis

Methods: In total, 81 knee joint surgery was performed on 71 children of the child age in connexion with the JRA. The average age of the patients was 11 years (6–14). To verify the diagnosis during diagnostic arthroscopy, a multifocal biopsy from 7 points was performed. Pathomorphological study of the material was performed according to the conventional histological method of studying soft tissues

Results: The results of the pathomorphological examination were analysed for the time frame of the appearance of the JRA. Pathomorphological early and late synovitis criteria were found. Early criteria (typical for the first three months after the JRA debut) – the phenomenon of necrosis in synoviocytes and the subintimal layer, palisade-like cell structures in the sub-synovial layer, synovocyte proliferation, fibrinoid superimpositions on the surface of the cover layer, productive endovascular endo-lytic endotheliosis, lymphocyte infiltration and plasmocytes. Late criteria (duration of the disease – more than 3–6 months): marked plasmacytic infiltration with the formation of lymphoid nodules with a hermetic centre, activation of fibrinoid and sclerotic processes with the formation of extensive fibrinoid necrosis with perifocal sclerosis, the formation of rheumatoid nodules, productive synovial hyperplasia, deposition of amyloid masses, formation of pannus granulation tissue with destructively invasive growth articular cartilage and synovium

Conclusions: Determining the stage of JRA is of great clinical importance for the early initiation of treatment and prevention of irreversible destructive complications. The proposed new method for determining the prevalence of pathological changes in the synovial membrane of the knee joint in children with JRA using a combined arthroscopic and pathomorphological evaluation of pathological changes in synovium in 7 joints allows to accurately determine the prevalence of the pathological process in the synovium, which has macroscopically only local manifestations

REFERENCES:

- [1] Gormley GJ, Steele WK, Gilliland A, et al. Can diagnostic triage by general practitioners or rheumatology nurses improve the positive predictive value of referrals to early arthritis clinics? *Rheumatology* 2003;42(6):763–768.
- [2] Houssien DA, Scott DL. Early referral and outcome in rheumatoid arthritis. *Scandinavian Journal of Rheumatology* 1998;27(4):300–302.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.2151

AB0130 SERUM LEPTIN AND ADIPONECTIN LEVELS IN RHEUMATOID ARTHRITIS PATIENTS, THEIR ASSOCIATION WITH INFLAMMATORY PROCESS

O. Galiutina, S. Shevchuk, Y. Sehed, I. Kuvikova. Institute of Invalid Rehabilitation, Vinnitsya, Ukraine

Background: It is well known that such bioactive substances as leptin and adiponectin are involved in different pathologic process including inflammation. At the same time, in a number of studies it was demonstrated anti-inflammatory properties