0.005) or HC (4.81%) ($p: 0.04$); between the 3 groups, pSTAT3 was differently expressed in CD4-lymphocytes and CD14-monocytes (figure 1). We also demonstrated a correlation between pSTAT3 level in CD14 +cells and ESR ($p: 0.026$; $r: 0.4625$). We found no correlation between pSTAT3 expression and Body Mass Index or disease duration.

Conclusions: In this study, STAT3 seems to be activated more in RA than SA or HC. Monocytes (as macrophage) and T-cells have important role in RA. Isomaki et al. demonstrated a correlation between IL-6 level and STAT3 activation so it might be suggested a possible role of pSTAT3 as a biomarker of disease activity. However, further studies are needed to explore the important role of JAK-STAT pathway in RA.

REFERENCES:

- [1] Harris TJ, et al. Cutting Edge: An In Vivo Requirement for STAT3 Signaling in Th17 Development and Th17-Dependent Autoimmunity. *J Immunol* 2007.
- [2] Nurieva R, et al. Generation of Inflammatory Helper T Cells STAT3 Regulates Cytokine-mediated. *J. Biol. Chem.* 2007.
- [3] Walker JG, Smith MD. The Jak-STAT Pathway in Rheumatoid Arthritis. Editorial of The Journal of Rheumatology 2005.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.6968

AB0135 CIRCADIAN RHYTHMS OF IMMUNE CELLS IN HEALTHY INDIVIDUALS AND PATIENTS WITH RHEUMATOID ARTHRITIS

S. Wilantri^{1,2}, on behalf of AG Buttgerit, C. Strehl^{1,2}, on behalf of AG Buttgerit, D. Abdiram^{1,2}, on behalf of AG Buttgerit, T. Gaber^{1,2}, on behalf of AG Buttgerit, F. Buttgerit¹, on behalf of AG Buttgerit. ¹Rheumatology and Clinical Immunology, Charité Universitätsmedizin Berlin/DRFZ Berlin; ²Glucocorticoid and Bioenergetic, Deutsches Rheuma Forschungszentrum, Berlin, Germany

Background: Clinical symptoms of rheumatoid arthritis (RA), such as joint stiffness, swelling and pain, manifest in circadian pattern with the highest intensity occurs at early dawn. This is known to correlate with the circadian expression of IL-6, which peaks before the onset of the symptom. Despite this finding, the circadian behaviour of immune system in cellular and molecular level in RA patients has not yet been extensively investigated.

Objectives: Our previous study suggested that immunological circadian rhythms in patients with RA were altered when compared to the healthy individuals. Currently, we are performing 24 hours study involving RA patients and healthy individuals to further monitor the dynamic occurrence of diverse immune cells in the periphery.

Methods: Up to this time five eligible RA patients aged 45–75 years and twelve eligible healthy controls were recruited to join the study. On the study day, the blood was drawn in two hours interval throughout 24 hours. The participants were provided with regular meal, allowed to eat snacks ad libitum and carry passive activities. The absolute number of circulating immune cells was determined using TruCount. RNA were isolated from CD14⁺ monocytes and analysed by real-time PCR.

Results: The major populations of immune cells in the periphery of healthy controls, including CD4 T cells, CD8 T cells, regulatory T cells, B cells and monocytes, displayed circadian rhythm that peaks during the rest phase. The rhythms are in general shifted a few hours later in the RA patients. Noteworthy, CD14 monocyte, which is one of the major sources of IL-6 in RA, showed a more pronounced rhythm with higher amplitude in RA patients compared to healthy individuals. Furthermore, the following clock genes are rhythmically expressed in CD14 monocytes of both groups: *Rorα*, *Per1*, *Per2*, *Per3* and *DBP*. The peak of *Rorα*, *Per1*, *Per3* *DBP* and *CRY1* is shifted a few hours later in RA patients. Interestingly, circadian variation is not observed in the expression of *RevErbα* in healthy individuals, while in the RA patients a rhythm is established.

Conclusions: In general, circadian rhythm of immune system in cellular and molecular level in RA patients appears to undergo phase shift and peaks a few hours later in comparison to healthy individuals. New established rhythms were also observed in cellular and molecular level. Another round of study involving seven RA patients is planned this spring to complete the project. Considering our data, we will continue to investigate circadian rhythms in expanded immune cell population using mass cytometry, immunoassay and microarray. Identification of immunological circadian rhythms in patients with RA and healthy individuals will help us to expand our knowledge in autoimmunity and provide an outlook on potential future implications.

Acknowledgements: We thank our clinical study team: Dr. Robert Biesen, Dr. Edgar Wiebe, Dr. Kim-Nikola Zeiner, Dr. Desiré Freier, Manuela Jakstadt, Lisa Ehlers, Annemarie Lang, Moritz Pfeifferberger, Alexandra Damerou, Pierre-Louis Krauß, Gabriela May and Marius Ibach for their help and contribution on the study days.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.2525

AB0136 THE EFFECT OF IL-6 RECEPTOR ANTIBODY FOR THE TREATMENT OF MCH-LPR/LPR-RA1 MICE THAT SPONTANEOUSLY DEVELOPED ANKYLOSING ARTHRITIS

T. Izumiya¹, Y. Mori¹, S. Mori², K. Takeda³, S. Horie³, T. Kodama³, E. Itoi¹. ¹Orthopaedic Surgery, Tohoku University Graduate School of Medicine; ²Oral and Maxillofacial Surgery, Tohoku University Graduate School of Dentistry; ³Laboratory of Biomedical Engineering for Cancer, Department of Biomedical Engineering, Graduate School of Biomedical Engineering, Miyagi, Sendai, Japan

Background: Mch-lpr/lpr-RA1 (RA1) mice are new strain mice which spontaneously developed arthritis in ankle and finally leads to ankylosis. There is no report that drug treatment has been applied to these mice.

Objectives: To examine the effect of mouse IL-6 receptor antibody MR16-1 for the treatment of RA-1 mice.

Methods: Male RA1 mice were randomly divided into treated and control groups. MR 16-1 was applied from 10 weeks of age for the treatment group. Saline was applied for the control group. The drug was administered every two weeks with the initial dose of 2 mg, then 0.5 mg. The effects were evaluated by histological synovitis score, *in vivo* imaging using ICG-encapsulated liposomes and the expression of serum SAA and IL-6.

Results: The tissue evaluation was carried out at 14 weeks, 17 weeks and 20 weeks of age. The histological score of treated groups were significantly improved compared with control group at every age. The interclass correlation coefficient was 0.771. *In vivo* imaging using ICG-encapsulated liposomes showed that significant signal decrease in treated groups at 14 weeks, but no significant difference was observed after 16 weeks. Blood SAA was significantly improved at 17 weeks of age.

Abstract AB0136 – Figure 1

Conclusions: IL-6 receptor antibody is effective for the treatment of ankylosing arthritis of RA1 mice. IL-6 might be a new potential target of treatment for ankylosing arthritis.

REFERENCE:

- [1] Mori S, Tanda N, Ito MR, et al. Novel recombinant congenic mouse strain developing arthritis with enthesopathy. *Pathol Int* 2008;58:407–414.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.1420

AB0137 ARTHRITIS-ASSOCIATED EGGERHELLA LENTA MODULATE DISEASE VIA METABOLIC AND MICROBIAL ALTERATIONS

V. Taneja. Mayo Clinic, Rochester, USA

Background: Role of environmental factors in predisposition to develop rheumatoid arthritis (RA) has gained interest due, in part, to the studies showing an association of gut microbiota with immune homeostasis. Although the etiology of RA is unknown, recent studies on the role of gut microbiota in inflammatory adaptive immune response have led to the concept that interaction between the host microbiome and genetic factors influences autoimmunity. We have shown an association of rare lineage commensals, *Eggerthella lenta*, with RA.

Objectives: In this study, we aimed to determine how human gut commensal *E. lenta* modulates gut epithelial integrity and inflammation via metabolites in humanised mice expressing RA-susceptible HLA-DQ8.

Methods: DQ8 mice following immunisation with type II collagen develop arthritis and antigen-specific cellular and humoral response. DQ8 mice orally gavaged