Figure 2. A heatmap shows the correlation between the intestinal microbiota and CD4+ T cells in patients with RA, and Ruminococcus torques at the genus level was negative related with Treg cells. (Colors indicate the Spearman rank correlation, *** P < 0.001).

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POS0397 AGGREGATED SURVIVIN BINDING AROUND HISTONE H3 EPIGENETIC MODIFICATIONS IN RISK LOCUS ASSOCIATED WITH RHEUMATOID ARTHRITIS

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Background: Survivin is an integral part of the Chromosomal Passenger Complex (CPC) which plays a vital role in mitosis. Experiments have demonstrated that survivin can physically bind to DNA. Crystallographic studies show that survivin binds to Threonine-3 of histone H3. In patients with autoimmune diseases, increased survivin expression contributes to an aggravated disease phenotype. Thus, functional, and mechanistic data point to a potential chromatin regulatory role for survivin, possibly in combination with activated disease phenotype. Thus, functional, and mechanistic data point to a potential therapeutic strategy to attenuate revascularization in RA. The increased aggregation of survivin around histone H3 EMs point to its potential regulatory function in gene transcription. Since regions around RA risk SNPs overlap with survivin peaks, survivin’s nuclear function could have immunologically important effects in mechanisms of autoimmune diseases.

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POS0398 ADIPONECTIN INDUCES SYNOVIAL ANGIOGENESIS IN RHEUMATOID ARTHRITIS THROUGH METABOLIC REMODELING

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Background: Our team have previously reported that Adiponectin correlates well with synovial inflammation and progressive bone erosion in rheumatoid arthritis (RA). Angiogenesis is another important part, which plays a critical role in the pathogenesis of RA.

Objectives: We hypothesized that adiponectin induces synovial angiogenesis in RA.

Methods: Single-cell RNA sequencing (scRNA-Seq) was used to screen cellular changes in local knee joint of collagen-induced arthritis (CIA) after intraarticularly injected of adiponectin. Chimeras models of synovium-cartilage-NOD/SCID mice, matrigel plug assay and rat aortic ring assay were performed to demonstrate the pro-angiogenesis role of adiponectin. Cellular experiment, including proliferation, migration, apoptosis, tube formation and angiogenesis related gene expression profile, were detected with Human Umbilical Vein Endothelial Cells (HUVEC) and Mice Lung Microvessel Endothelial Cell (MLMEC) after adiponectin stimulation. Seahorse was performed to clear the influence of adiponectin to cell metabolism.

Results: The synovium and pannus hyperplasia worse in CIA model after intraarticularly injected of adiponectin, along with more serious synovitis and bone erosion. ScRNA-Seq of synovial tissues separated from CIA reminded that endothelial cell barbarically grows via metabolic remodeling after stimulated with adiponectin. Synovial chimeras, matrigel plug and rat aortic ring shows adiponectin accelerates angiogenesis significantly in different background conditions. In vitro, endothelial cell proliferation detecting by RCTA and CCK8, migration by wound healing and transwell, apoptosis by FACS, tube formation and angiogenesis related gene expression profile by PCR-ARRAY were promoted by adiponectin in both HUVEC and MLMEC. Seahorse showed HUVEC made more use of glycolysis after co-cultured with adiponectin, a method of cell energy supply that tumor cells possess called warburg effect, that drives endothelial cell hyperplasia in severe environment.

Conclusion: As a classic metabolic regulator, adiponectin exacerbates CIA by promoting angiogenesis through metabolic remodeling. The findings not only provide a novel insight into the pathogenic role of adiponectin, but also reveals a potential therapeutic strategy to attenuate revascularization in RA.

Disclosure of Interests: None declared
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POS0399 TMT-BASED QUANTITATIVE PROTEOMICS ANALYSIS OF SYNOVIAL FLUID-DERIVED EXOSOMES IN RHEUMATOID ARTHRITIS, AXIAL SPONDYLOARTHRITIS, GOUT AND OSTEOARTHRITIS

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Background: The pathogenesis of the joint diseases rheumatoid arthritis (RA), axial spondyloarthritis (axSpA), gout, and osteoarthritis (OA) are still not fully elucidated. Exosomes in synovial fluid (SF) has a critical role in the pathogenesis of arthritis. None of study has compared the proteomics of SF-derived exosomes in RA, axSpA, gout and OA.

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