absence of fibrin deposition in Fg−/− mice was accompanied by significantly lower synovial inflammation, chondro-synovial adhesion and cartilage damage than in WT mice. Chondro-synovial adhesion correlated with cartilage damage in the WT and led to apparent mechanical stripping of the superficial cartilage, while this phenomenon was not observed in the Fg−/− mice. In vitro, autologous RA synoviocytes adhered to cartilage explants exclusively in the presence of fibrin deposition. Fibrinogen chains were not expressed by primary chondrocytes, indicating passive deposition from synovial fluid or tissue. In human RA cartilage explants, we found colocalization and a significant positive correlation between fibrin and calcific deposits. Fibrinogen caused exacerbated calcification in CPP-treated primary murine chondrocytes and induction of genes involved in chondrocyte calcification (Pc1, Pit1). Cartilage-oligomeric matrix protein (Comp) gene was also highly induced suggesting a pro-catabolic role of fibrinogen.

**Conclusion:** Fibrin deposition is an active trigger of cartilage degeneration in RA via induction of chondro-synovial adhesion (mechanical aspect) and induction of calcification (catabolic aspect). Newer therapeutic approaches may not merely focus on fibrinolysis but protect cartilage from fibrin-induced adhesion or calcification e.g. by fibrin-targeted immunotherapy.

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**Table 1.**

<table>
<thead>
<tr>
<th>Patient’s groups</th>
<th>RA</th>
<th>OA</th>
<th>HC</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Porphyromonas gingivalis positive</td>
<td>24 (80%)</td>
<td>9 (35%)</td>
<td>17 (72%)</td>
<td>RA → OA (p = 0.001)</td>
</tr>
<tr>
<td>Porphyromonas gingivalis negative</td>
<td>6 (24%)</td>
<td>17 (65%)</td>
<td>17 (71%)</td>
<td>RA → HC (p = 0.001)</td>
</tr>
</tbody>
</table>

**Conclusion:** The results of our study indicate that PG is found more frequently in periodontal pockets of patients with rheumatoid arthritis, which implies the important role of oral microbiota in RA pathogenesis, treatment and prevention.

**References:**

**Disclosure of Interests:** Pavel Selimov: None declared, Elena Firkova: None declared, Ljubinka Damjanovska-Krstikj Consultant of: Roche, Speakers bureau: Pfizer, Anastas Batalov: None declared, Ana Maneva: None declared, Rositsa Karaillova: None declared, Girma Delcheva: None declared, Katia Stefanova: None declared, Teodora Stankova: None declared

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**AB0094**

**FUNCTIONAL TREG CELLS MAY BE CONVERTED INTO T EFFECTOR PHENOTYPE ON EXPOSURE TO INFLAMMATORY MILIEU IN RHEUMATOID ARTHRITIS (RA) SYNOVIAL FLUID: IN-VITRO STUDY**

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**Background:** The debate on functional versus numerical difference in T regulatory cell population among patients with Rheumatoid arthritis (RA) is not clear. Tregs expressing Inflammatory subset phenotype markers, such as Th1(Ifnγ, Th2(Tsl), Th17(βet), and Th0(Treg) are reported. (1) Though the reported numbers of synovial fluid tregs are higher in RA, (2) the fate of Tregs on entering the synovial inflammatory milieu from peripheral blood (PB) has not yet been investigated.

**Objective:** To compare Treg frequencies in PB and synovial fluid between osteoarthritic (OA) and RA patients.

To compare cytokine levels in PB and SF between OA and RA
To study the effect of autologous synovial fluid on RA and OA Treg isolated from peripheral blood

**Methods:** The Peripheral Blood (PB) and synovial fluid (SF) of RA (n=80) and OA (n=30) patients were analyzed for CD4+T-cell subset frequencies and phenotypes by flow cytometry. Cytokine concentrations in plasma and SF were measured by cytometric bead array. Tregs from 5 RA-PB and 5 OA-PB were isolated and cultured in autologous synovial fluid for 24 hrs. Phenotypic expression of Th1 and Th17 chemokines on the cell surface were analyzed by flow cytometry and expression levels of T-bet, RORγ and FOXP3 in those Treg cells were measured with quantitative real-time PCR (RT-qPCR).

**Results:** The PB and SF frequencies of Th1, Th17 and Tregs are shown in Table 1. The pro-inflammatory cytokines were high in the plasma and SF of RA but the anti-inflammatory cytokines were similar (Fig 1A&B). Treg cells were isolated from RA and OA PB and cultured in autologous SF for 24 hrs. RA Treg showed increased cell surface expression of CXCR3+ and CCR6+ (Fig 1C) and there was no difference in OA Treg. Gene expression studies showed an increased expression of T-bet, RORγ and decreased expression of Foxp3 in RA Tregs while there was no difference in OA Treg before and after in vitro culture (Fig 1D).