Macrophages are known to exhibit remarkable phenotypic plasticity and understanding the role of this characteristic in regulating inflammation and pathology remains a major challenge, as does the characterization of factors in the microenvironment such as the synovium that control such macrophage characteristics. Importantly, whether the infiltrating, inflammatory macrophages of the RA ST similarly exhibit such phenotypic plasticity, and whether this occurs during the process of reaching remission, remains to be studied.

**Objectives:** We investigated the phenotypic plasticity of inflammatory synovial macrophages from patients with RA in vitro, investigating their ability to convert from an inflammatory macrophage population into ‘regulatory’ CD206+MerkTK+ macrophages. These findings will provide a proof-of-concept as to the utility of these macrophages for a cell-based therapy in resolving inflammation in patients with RA, and will likely extend our understanding of the mechanisms of action of currently used therapeutics.

**Methods:** Synovial fluid (SF) mononuclear cells were obtained from patients with active early RA (<1 year; fulfilling 2010 ACR/EULAR classification criteria). Cryopreserved SFMCs were cultured for 48hr in the presence of 10ng/mL interferon(IFNγ), 50ng/mL dexamethasone, 10 µg/mL Infliximab, or diluent. Following culture, cells were immunostained and analysed using a Beckman Coulter CytoFLEX flow cytometer and FlowJo software. SF macrophages were characterised by expression of CD14, CD45, CD68 (Figure 1A), and proportions of CD206+MerkTK+ macrophages measured.

**Results:** Prior to culture, the CD68+ macrophage populations present in SF were found to be predominantly CD206+MerkTK+. After 48 hours of culture, in the absence of any stimulus, there was an increase in proportions of CD206+MerkTK+ macrophages. Treatment with either dexamethasone or anti-TNF (Infliximab) resulted in a further increase in proportions of CD206+MerkTK+ cells. In contrast, culture with IFNγ reduced this induction in a population. Importantly, we found that the generated CD206+MerkTK+ macrophages were phenotypically stable in culture following removal of these differentiating agents.

**Conclusion:** Our findings demonstrate that inflammatory SF cells are indeed able to polarize to regulatory, CD206+MerkTK+ macrophages in vitro. The findings provide further mechanistic insights into the role of the therapeutic benefits of glucocorticoids and TNF inhibitors, as well as providing initial proof-of-concept in the use of regulatory macrophages as a cellular-based therapy or therapeutic target for patients with RA.

**References:**

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POS0059 COMPLEMENT PROTEINS ARE ELEVATED IN PATIENTS WITH AXSPA COMPARED WITH RELEVANT CONTROL PATIENTS WITH LOW BACK PAIN AND SPA-FEATURES WITHOUT AXSPA.

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**Background:** Axial spondyloarthritis (axSpA) is associated with a certain genetic predisposition, i.e., with the presence of human leukocyte antigen (HLA)-B27. However, the pathogenesis remains largely unexplained. Animal models of ankyllosing spondylitis have shown inhibition of complement to be beneficial in terms of limiting structural damage (1). The lecin pathway of complement activation seems to be a key component of the innate immune system and plays a pivotal role in both homeostasis and development. The influence of complement in axSpA is mainly unexplored. We have, however, previously reported elevated plasma levels of the lectin pathway proteins L-ficolin and H-ficolin in patients with axSpA compared with blood donors (2).

**Objectives:** Our aim was to investigate plasma levels of lectin pathway proteins in a clinical cohort of patients with axSpA and compare them to relevant controls that often experience similar challenges in differentiating from axSpA.

**Methods:** Plasma samples were obtained from individuals in a cohort of patients suffering from low back pain (LBP) including: 1) 23 patients with axSpA, 2) 55 patients without axSpA experiencing SpA-features/symptoms, and 3) 64 patients suffering from low back pain (LBP) and fulfilling the diagnostic criteria for the "轴性脊柱关节炎(Low Back Pain)" by the ASAS (2016) task force.

**Results:** Plasma levels of C1r/C1s, C4, C5, C5b-9, C4d, C1s, C3, C3c/C3d, C5a, C6a, C7a, C8a, C9a, C8bp, and C9bp were significantly higher in patients with axSpA compared to LBP patients. The levels of C1r/C1s, C4, C5, C5b-9, C4d, C1s, C3, C3c/C3d, C5a, C6a, C7a, C8a, C9a, C8bp, and C9bp were also significantly higher in patients with axSpA compared to controls not fulfilling axSpA diagnostic criteria.

**Conclusion:** This study provides evidence that complement activation is increased in patients with axSpA compared to patients with LBP and controls not fulfilling axSpA diagnostic criteria. This finding suggests a potential role for complement in the pathogenesis of axSpA and highlights the need for further research to elucidate the mechanisms underlying this increased complement activation.