CD25 and PD-1. Evaluating the central site of pathology, CXCL13 was highly expressed in the inflamed facet joints (Figure 1).

**Conclusion:** The multiomics analysis revealed multiple cytokines to be activated in eSpA patients. The early disease is characterized by T cell activity in combination with macrophage and monocyte attractive chemokines. CXCL13 stands out as a chemokine of particular interest, being increased both in eSpA, at the central- and peripheral site of pathology, and associated with structural changes in time. The CXCR5-CXCL13 axis could be an important new axis in understanding SpA disease pathology, serving both as a biomarker for disease severity and progression, but also to lead to new targets of treatment.

**Methods:** Single-cell RNA sequencing of sorted synovial fibroblasts and imaging mass cytometry from different mouse models of arthritis (TNFtg, S. neobuchanan, S. gordonii). We aimed to reprogram fibroblasts, from pro-inflammatory and tissue destruction. Herein, persistent joint fibroblast activity leads to continuous destruction of the joint, whereas its inhibition/silencing prevents joint damage despite presence of a residual inflammation[1]. Persistent activation of the joint fibroblasts leads to destruction of the joint[1], and silencing both, inflammation and mesenchymal activation is required to protect joints from damage.

**Objectives:** To examine the influence of continuous mechanical load (CML) on the osteogenic behavior of MSc with or without INF-γ/TNF-α/IL-22 or testosterone exposure.

**Methods:** Adipose tissue-derived MCs, characterized by CD90, CD73, CD105, CD45, CD31, isolated from the adipose tissue of a healthy donor and kindly donated by Bonus BioGroup, Hafia, Israel, were cultured for two weeks without or under CML of 2 gr/cm^2^, applied by transparent glass cubes placed on the plates covering the well, in the following conditions: a. osteogenic differentiation medium (ODM) with no additions of cytokines or testosterone; b. ODM with the addition of interferon-γ (INF-γ), tumor necrosis factor-α (TNF-α), allowed IL-22-induced proliferation and migration of mesenchymal stem cells (MSC) [1].

**Results:** Mechanical load prevented MSC proliferation and migration in the presence of INF-γ/TNF-α/IL-22 (Figure 1).

**CONCLUSION:** This new treatment approach might have several advantages over anti-inflammatory drugs. (i) no interference with important defence mechanisms against pathogens and no increased risk of opportunistic infections, (ii) activation of a mechanism that retains tissue integrity, (iii) option to combine with anti-inflammatory drugs, making silencing of fibroblasts to a new tool of disease control.

**REFERENCES:**

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**REFERENCES:** NIL.