easily accessible, rich source of MSCs. Biology of MSCs in AS is poorly understood and data describing MSCs from adipose tissue (ASCs) are missing.

Objectives The aim of this study was to perform comparative basic characterisation of ASCs of AS patients (AS/ASCs) and ASCs line originating from healthy volunteers (hASCs).

Methods AS/ASCs of 15 AS patients and commercially available hASC lines were used. Phenotype and expression of adhesion molecules (ICAM-1, VCAM-1) on ASCs were evaluated by flow cytometry. Basal and cytokine (tumor necrosis factor +interferon γ or interleukin-23)-induced secretion of nine factors (TGF β , IL-6, galectin, LIF, IL-1Ra, PGE $_2$, sHLA-G, TSG6 and kynurenines) known to mediate immunomodulatory activity of MSCs was measured by ELISAs. ASCs were co-cultured with either anti-CD3/CD28-stimulated CD4 $^+$ T lymphocytes or mitogen-stimulated peripheral blood mononuclear cells (PBMCs) of allogeneic healthy volunteers. Expression of activation markers (HLA-DR, CD25, CD69) on T cells and these cells proliferation were evaluated by flow cytometry.

Results There were no significant differences in the phenotype, expression of adhesion molecules and secretory potential between hASCs and AS/ASCs. Inhibition of T cell proliferation was found in both co-culture systems, while modulation of activation markers expression (CD25 down-regulation, CD69 up-regulation) on CD4 $^+$ T and CD8 $^+$ T cell subsets was stated in ASCs-PBMCs co-cultures only and hASCs and AS/ASCs exerted similar effects.

Conclusions AS/ASCs have phenotype and basic biological features (i.e. secretory potential, some attributes related to immunomodulatory activities) comparable to hASCs.

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Disclosure of Interest None declared.

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VISFATIN DOWN-REGULATES GROWTH PROMOTING LNCRNA H19 DURING OSTEOGENIC DIFFERENTIATION OF MESENCHYMAL STROMAL CELLS

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Career situation of first and presenting author Student for a master or a PhD.

Introduction Osteoarthritis (OA) and osteoporosis are destructive bone diseases causing chronic pain and leading to disability. Mesenchymal stromal cells (MSC) are essential to bone health and tissue repair. Adipokines such as visfatin alter the osteogenic potential of MSCs and contribute to the loss of bone homeostasis. Long non-coding RNA H19 is one of the first lncRNAs discovered and relevant for distinct processes, e.g. during embryonic growth and tumor formation. lncRNAs interact directly with DNA, RNA as well as proteins, modulating transcription, protein expression and protein function. H19 upregulation was shown in osteogenic differentiation of MSCs.¹ H19 increases the osteogenic potential via TGF β 1 and Wnt/ β -catenin pathways.^{2,3}

Objectives To investigate the influence of visfatin on H19 expression during osteogenesis.

Methods Human MSCs from healthy donors (hMSC) and primary human MSCs from osteoarthritis patients (phMSC) after knee replacement surgery were treated with differentiation medium to induce osteogenic differentiation (OD). Matrix mineralization (MM) was quantified after 21 days by Alizarin Red. H19 expression by realtime PCR and IL-6 production by ELISA were measured.

Results lncRNA H19 was up-regulated during OD. Although the H19 upregulation was not altered by co-stimulation with resistin, leptin or TNF α , visfatin co-stimulation during OD down-regulated H19 expression up to 10-fold as compared to unstimulated MSCs. The effect was significant in phMSCs at two of three measured time points (day 7 $p=0.03$; day 14 $p=0.002$, $n=3$) and in hMSCs at day 14 ($p=0.0003$, $n=4$). Visfatin co-stimulation of MSCs in OD increased MM, as well as IL-6 levels. However, TNF did not alter H19 expression or increase MM.

Conclusions Visfatin co-stimulation during osteogenesis down-regulated lncRNA H19 expression, indicating a loss of the growthpromoting effects of lncRNA H19 in affected areas of destructive bone disease. This regulatory effect was specific to visfatin and did not occur upon co-stimulation with other adipokines or inflammatory stimuli such as TNF supporting a TNF-independent effect of visfatin.

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Disclosure of Interest None declared.

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PODOPLANIN REGULATES THE MIGRATION OF MESENCHYMAL STROMAL CELLS AND THEIR INTERACTION WITH PLATELETS

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Career situation of first and presenting author Young investigator.

Introduction Within the rheumatoid joint, mesenchymal stromal cells (MSC) up-regulate podoplanin with unknown consequences for disease pathogenesis. The function of podoplanin has been linked to enhanced migratory potential and interactions with platelets. However, it is unclear how these two cell types interact with one another, given that MSC and platelets are usually located in different anatomical compartments (tissue vs blood respectively) separated by the blood vascular endothelial cells (EC).

Objectives Here, we examined the functional consequences of podoplanin expression on the migratory potential of MSC and their interactions with circulating platelets.