and IL-6 protein production were measured using realtime PCR and ELISA at day 2/7/14 of OD.

Results: H19 up-regulation in MSCs during OD could be confirmed in a time dependent manner. Visfatin-stimulation of MSC during OD increased matrix mineralization over time as well as IL-6 production (day 7, 14, 21: 46-, 93-, 78-fold). Visfatin stimulation down-regulated H19 expression up to 10-fold over the course of OD compared to non-stimulated control. The effect was significant in phMSCs in 2/3 measured time points (day 7, $p=0.03$; day 14, $p=0.002$, $n=3$) and in hMSCs on day 14 ($p=0.0003$, $n=4$).

Conclusions: During osteogenic differentiation of MSCs, visfatin showed pro-inflammatory and mineralization promoting effects. However, H19 was significantly down-regulated by visfatin during osteogenic differentiation. This may contribute to the loss of osteogenic potency of MSCs in inflamed tissues with increased visfatin concentration as observed in affected areas of destructive bone disease. Further research to understand the H19 effector mechanisms on osteogenic differentiation and osteogenic potency of MSCs are in progress.

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SAT0031 INTERLEUKIN 29 INHIBITS OSTEOCLAST DIFFERENTIATION AND FUNCTION IN RANKL-INDUCED OSTEOCLASTOGENESIS

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Background: Interleukin-29 (IL-29) is a new member of the recently discovered interferon (IFN) λ family and known to modulate immune functions of monocyte or macrophage. We have demonstrated that IL-29 is dysregulated in patients with rheumatoid arthritis (RA) and contributes to RA pathogenesis via inducing proinflammatory cytokines, chemokines or matrix metalloproteinases production in synovial fibroblasts,^{1,2} as well as stimulating inflammation and cartilage degradation in osteoarthritis disease.³ However, the role of IL-29 in bone resorption is unclear.

Objectives: Bone erosion in RA is associated with increased production of pro-inflammatory cytokines and accelerated osteoclastogenesis in affected joints.⁴ IL-29 is an important proinflammatory cytokine in RA. We investigated the effect of IL-29 on receptor activator of nuclear factor κ B ligand (RANKL)-induced osteoclastogenesis *in vitro* to determine whether IL-29 can stimulate or attenuate osteoclast-mediated bone resorption that is a hallmark of RA.

Methods: The viability and apoptosis of RAW264.7 cells after IL-29 different treatment were assessed by Cell Counting Kit-8 and flow cytometry, respectively. Osteoclasts were generated from RAW264.7 cells, bone-marrow-derived monocyte/macrophage precursor cells (BMMs) and blood-derived human PBMC cells. The effects of IL-29 on osteoclast formation were evaluated by tartrate-resistant acid phosphatase (TRAP) staining and counting the number of TRAP + multinucleated cells. Bone resorption experiment was assessed by pit formation. The expression of key molecules implicated in osteoclastogenesis (TRAP, CTSK, MMP-9, NFATC1 and c-Fos) was measured by real time RT-PCR.

Results: Although IL-29 did not show significant effect on the viability and apoptosis of RAW264.7 cells, it inhibited multinucleated cells in the differentiation of murine and human osteoclastogenesis, the bone-resorbing activity of mature osteoclasts. In addition, IL-29 downregulated osteoclast specific genes expression of TRAP, CTSK, MMP-9, NFATC1 and c-Fos.

Conclusions: IL-29 inhibits murine and human osteoclastogenesis by a direct mechanism suppressing responses of osteoclast precursors to RANKL. Our findings suggest that IL-29 may play a previously unrecognised role in the osteoclast formation.

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SAT0032 MESENCHYMAL STEM CELLS ALLEVIATE EXPERIMENTAL AUTOIMMUNE CHOLANGITIS THROUGH IMMUNOSUPPRESSION MEDIATED BY GALECTIN-9

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Background: Mesenchymal stem cells (MSCs) play anti-inflammatory role by secreting some kinds of bioactive molecules. However, the effect of MSCs on chronic autoimmune liver disease, such as primary biliary cholangitis (PBC) and its underlying mechanism remains elusive.

Objectives: The aim of this study was to assess the efficacy of UC-MSCs treatment (UC-MSCT) in 2OA-BSA-induced murine autoimmune cholangitis and explore its underlying mechanisms.

Methods: UC-MSCs were transplanted into experimental autoimmune cholangitis mice. Biochemical and histological analysis were performed based on the blood and liver tissues. The immunomodulatory effects of UC-MSCs and its cytoprotective function were also investigated.

Results: We found that UC-MSCT significantly ameliorated liver inflammation in 2OA-BSA induced autoimmune cholangitis mice, primarily by diminishing Th1 and Th17 responses, and modifying liver chemokine activity. We also found that UC-MSCs significantly repressed the proliferation of CD4⁺T cells and suppressed the differentiation of Th1 and Th17 cells, both of which were dependent on galectin-9 (Gal-9). Furthermore, we determined the signal transducer and activator of transcription (STAT) and c-Jun N-terminal kinase (JNK) signalling pathways were involved in the production of Gal-9 in MSCs.

Conclusions: The present study shows that UC-MSCs exert profound inhibitory effects on inflammatory responses and that they ultimately alleviate the liver injury in experimental autoimmune cholangitis mice. Further, we demonstrate that UC-MSCs inhibit Th1 and Th17 cell responses as well as aberrant chemokine activity through Gal-9 mediated immunosuppression. Additionally, our research reveals that the induction of Gal-9 in MSCs is mediated by the involvement of the STAT and JNK signalling pathways. These findings may help in the development of stem cell therapies for the treatment of PBC.

Disclosure of Interest: None declared

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SAT0033 TIME-DEPENDENT RELATIONSHIPS BETWEEN BIOLOGICAL PARAMETERS AND DISEASE ACTIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterised by high inter-patient variability of clinical features, pathology, and disease time-course. Relationships between biomarkers and disease remission/relapse cycles are especially complex and poorly understood.

Objectives: To investigate the relationship between disease activity and biomarker expression in a longitudinally-followed SLE cohort.

Methods: We measured 4 candidate protein biomarkers implicated in SLE (MIF, CCL2, CCL19 and CXCL10) and 13 routinely collected serum and urine biological parameters, and assessed disease activity (SLEDAI-2k) on each clinic visit. We analysed these data by first focusing on the magnitude of expression levels of the 17 biological markers and then on the temporal dimension, to untangle their relationship to disease activity.

Results: Data from 843 visits in 110 SLE patients (median age 47, 83% female, 49% Asian ethnicity) were analysed. We demonstrated highly heterogeneous time-dependent relationships between disease activity and the measured