and IL-6 protein production were measured using realtime PCR and ELISA at day 27/14 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 pMSCs 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 is needed to understand the H19 effector mechanisms on osteogenic differentiation androgenic potential of MSCs are in progress.

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