of most common therapies used in RA patients and has been shown to have beneficial effects on RA-associated cardiovascular pathologies in these patients.¹ Initial studies in the collagen-induced arthritis (CIA) model revealed an increase in cardiac fibrosis marker galectin-3, both locally in perivascular adipose tissue (PVAT) and systemically in blood plasma during inflammatory arthritis and correlated with impaired vascular function.²

Objectives This study aims to investigate the impact of anti-TNF treatment on galectin-3 expression and vascular function during CIA.

Methods Male DBA/1 mice underwent CIA induction and received either Enbrel or phosphate buffered saline (PBS) by intravenous injection. Vascular function was determined by measuring the constriction response of the thoracic aorta at termination using a myograph. Galectin-3 was measured locally in thoracic PVAT by immunohistochemistry and quantitative polymerase chain reaction (qPCR), and systemically by ELISA.

Results Anti-TNF treatment during CIA resulted in a decrease in arthritis severity and progression compared to PBS treated mice, and partially restored vascular function. Galectin-3 expression was significantly reduced in thoracic PVAT, but not in blood plasma, in CIA mice treated with Enbrel.

Conclusions In conclusion, decreased galectin-3 expression in Enbrel treated mice is associated with an improvement in arthritis-associated disease activity including arthritis clinical score, joint swelling and vascular function.

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VISFATIN IN BONE METABOLISM OF OSTEOPOROSIS AND OSTEOARTHRITIS PATIENTS

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Introduction Osteoporosis predominantly affects elderly people and is characterised by bone loss, increased fracture risk and reduced regeneration ability. Age-related bone loss correlates with increased bone marrow adiposity due to a shift of osteogenic towards adipogenic differentiation of bone marrow-derived mesenchymal stem cells (MSC). Adipose tissue is metabolically active. Through the release of adipokines with immunomodulatory properties adipose tissue might influence differentiation of bone marrow-derived MSC and contribute to bone loss in osteoporosis.

Objectives Thus we analysed the presence of adipokines (visfatin, resistin and leptin) in the bone marrow cavity and their effects on MSC differentiation.

Methods MSC and RNA were isolated out of spongiosa from femoral heads (hip replacement surgery of osteoarthritis patients (OA) or after osteoporotic femoral neck fracture (FF). Adipogenic as well as osteogenic MSC differentiation was performed with/without adipokines. For the transfer and differentiation of MSC on cancellous bone, cell-free bone fragments were prepared and sterilised. Proinflammatory factors were measured by immunoassays. Gene expression was evaluated by Realtime PCR. Matrix mineralization was assayed using Alizarin red S staining.

Results While resistin was reduced, visfatin and leptin levels were increased in FF bone vs. non-osteoporotic OA bone (FF mean visfatin 7.77 ± 1.86 , $n=11$; OA 2.25 ± 0.36 , $n=13$, $p=0.002$). In contrast to leptin and resistin, visfatin induced the secretion of proinflammatory factors (IL-6, IL-8, MCP-1) during both, osteogenic and adipogenic differentiation, (e.g. adipogenic differentiation IL-6, 21d; control: 270.3 ± 140.2 pg/ml; visfatin: $3,223 \pm 600.4$ pg/ml, $p=0.0006$, $n=12$). In osteogenically differentiated cells matrix mineralization was significantly increased, while collagen type 1 expression was downregulated (d21: -4.2 -fold/ $p=0.0001$; $n=7$) after visfatin stimulation. The expression of MMP2, -13, TIMP1 and -2 (e.g. d21: -2.4 -fold; -3.2 -fold; -3.2 -fold; -4.3 fold, respectively) was also reduced by visfatin during osteogenesis. Interestingly, visfatin significantly induced MMP13 expression (e.g. d21: 104-fold) during adipogenic differentiation under cell culture conditions. However, when differentiated on autologous cancellous bone, visfatin-induced MMP13 expression, as well as IL-6 and IL-8 release was markedly reduced (e.g. MMP13 21d: 13.8-fold, $p=0.0156$, $n=7$).

Conclusions Taken together, visfatin expression was not only elevated in osteoporotic bone, it induced release of proinflammatory factors and dysregulated the MMP/TIMP balance during MSC-differentiation. Therefore visfatin might influence bone remodelling specifically at the bone/adipose tissue interface. The visfatin-mediated increase of matrix mineralization and the observed reduction of organic component of ECM – Coll.1, essential for the bone plasticity might contribute to bone fragility and therefore to the pathogenesis of osteoporosis.

Disclosure of interest None declared

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INTENSIVE 24-WEEK PHYSIOTHERAPY PROGRAMME IN PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES – PRELIMINARY DATA FROM A SINGLE-CENTRE CONTROLLED STUDY

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Introduction Presence of muscular inflammation, followed by atrophy in idiopathic inflammatory myopathies (IIM) leads to impaired function, reduced muscle strength, endurance and aerobic capacity, decreasing quality of life. Data on the efficacy of non-pharmacologic care in IIM is very limited due to variety in studied interventions/outcomes.