effect of recArg1 regarding an *in vivo* model called serum transfer arthritis, where we treated C57BL/6J wildtype mice with the recombinant enzyme. Disease severity was then assessed using clinical scores and paw histology.

Results We observed that *ARG1* mRNA expression was down-regulated from the progression of a precursor to a mature osteoclast. We incubated day 3 osteoclast precursors with recArg1 and observed that addition of 1000 ng/ml recArg1 abolished osteoclastogenesis. L-Arginine deprivation led to a decrease in the oxygen consumption rate of osteoclast precursor cells, assessed 48 hour after RANKL addition. Using serum transfer arthritis, an established murine *in vivo* model, recArg1 treated mice showed reduced disease severity combined with a significant decrease in the presence of osteoclasts. Treatment efficiency was evaluated using an L-Arginine ELISA, where the amino acid was found to be absent in the serum of treated mice.

Conclusions We propose that the amino acid L-Arginine is critical for the development of osteoclasts from myeloid precursors and hypothesise that its abundance, influenced by recArg1 addition, influences development and severity of osteoclast driven diseases.

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P114

IONISING RADIATION INHIBITS INFLAMMATION IN PATIENTS WITH MUSCULOSKELETAL DISEASES: RADON TREATMENT VS LOW-DOSE RADIATION THERAPY

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Introduction Rheumatoid arthritis (RA) and osteoarthritis (OA) are the most common musculoskeletal diseases (MSD) that affect the joints. Reduced mobility and quality of life are the consequences of the cartilage and bone tissue destruction and the chronic inflammation process, caused by release of bone destruction markers and inflammatory factors including adipokines in the joint. Besides medications, an additional pain relief is achieved by the treatment of patients with low-dose ionising radiation, either as local low-dose radiation therapy (LDRT) or whole-body exposure to radon in radon baths or galleries.

Objectives In the previous work we showed the decrease of serum levels of visfatin and serum carboxy-terminal collagen crosslinks of type-I collagen (CTX-I) in patients treated in radon baths.¹ In the present study, we analysed serum samples of patients with MSD, who had been treated locally with photon radiation (LDRT). In addition, we analysed differentiation

and activity of osteoclasts that were differentiated *in vitro* from patient-derived monocytes.

Methods Serum samples were collected from patients before and after treatment. Levels of visfatin and CTX-I were measured by ELISA. Monocytes were isolated from blood samples of patients and cultivated with M-CSF and RANKL on bone slices for 2 weeks. Osteoclasts were defined as TRAP and F-actin positive cells. TRAP activity was measured in the cell supernatants using TRAP Staining Kit.

Results In the serum of patients treated with LDRT, a trend to reduced concentration of CTX-I was observed directly after the therapy. Further, osteoclasts, differentiated *in vitro* from LDRT patient-derived monocytes, showed reduced TRAP activity.

Conclusions The observations made in this study so far substantiate that the radiation-induced decrease of CTX-I levels could be one main factor that is related to the attenuation of inflammation and to the decrease of disease activity in the patients with MSD. This hypothesis is endorsed by the observed reduced differentiation and activity of *in vitro* cultivated patient-derived osteoclasts.

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P115

LOW DOSE RADIATION HAS A POSITIVE IMPACT ON BONE METABOLISM IN AN EXPERIMENTAL MODEL OF INFLAMMATORY ARTHRITIS

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Introduction Rheumatoid arthritis (RA) is, next to inflammation and infiltration of activated immune cells into the synovial joint, characterised by a progressive destruction of cartilage and bone. Although today's treatment options are very effective for many patients, not all of them respond properly or have to reduce medications due to adverse effects. In these patients it is crucial to slow down bone loss and inflammation in a timely manner to prevent further damage. Here, low-dose radiotherapy (LD-RT) could be an option, as it has been shown to ameliorate inflammation and to reduce pain. Using the human *TNFα* transgenic (*hTNFα* tg) mouse model as an experimental model of inflammatory arthritis, we revealed that locally applied LD-RT attenuates inflammation in the joints.

Objectives As little is known about the impact of LD-RT on bone metabolism, we thus focused on the effects of LD-RT on bone homeostasis.

Methods Bone marrow-derived osteoclasts (OC) of *hTNFα* tg mice were differentiated using M-CSF and RANK-L and