the roles of adipokines in pathogenesis of autoimmune disorders are still controversial due to their both pro-inflammatory and anti-inflammatory effects.

**Objectives:** The aim of this study was to evaluate adipokine levels in patients with RA and to assess their association with the activity of inflammatory process.

**Methods:** The study included 62 patients with RA and 35 practically healthy sex and age-matched persons of control group. The diagnosis of RA was established according to the ACR 2010. Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) were used to assess inflammation. Disease activity and functional impairment were evaluated using the Disease Activity Score (DAS28). Serum leptin and adiponectin levels were studied by immunoassay using standard sets (DRG, Germany and “Oregenium”, Finland). Results are expressed as mean ± standard error of the mean. Spearman’s ρ was used to calculate correlations between markers of disease activity (ESR, CRP, DAS28) and serum adipokine levels. A p value<0.05 was considered statistically significant for all tests.

**Results:** It was found that the mean value of leptin and adiponectin levels were 20.7±12.3 ng/ml and 2.47±1.34 ng/ml respectively in patients with RA and 6.47±3.17 ng/ml and 4.21±1.4 ng/ml respectively in the control group. Thus, the leptin level in patients with RA was 3.2 times higher, and adiponectin level was 1.7 times lower than in healthy individuals. Levels of adipokines were associated with the activity of the inflammatory process. Thus, serum concentration of leptin level was increased (r=0.33 and r=0.35) and adiponectin level was decreased (r=−0.25 and r=−0.24) with the increasing of ESR and CRP. Similar patterns were observed for the integral index of RA activity DAS28. In particular, DAS28 was 1.6 times higher in subjects with leptin levels above 44.7±5.4 ng/ml than in the group of patients with leptin levels below 44.7±9.4 ng/ml. The correlation analysis also confirmed the close association between the leptin and adiponectin levels DAS28 activity index (r=0.37 and r=−0.28, respectively).

**Conclusions:** Disadipokinemia in patients with RA is characterised by the increasing of serum leptin level and the decreasing of serum adiponectin level and is closely related to the activity of the inflammatory process.

**Disclosure of Interest:** None declared.

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**AB0131**

**RHEUMATOID FACTOR IS DETECTED ON CIRCULATING EXTRACELLULAR VESICLES IN A SUBPOPULATION OF RHEUMATOID ARTHRITIS PATIENTS WITH A MORE SEVERE DISEASE PHENOTYPE**

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**Background:** Extracellular vesicles (EVs) play a role in cell-cell communication and contain numerous signalling molecules inside and on their cell membrane. Although their function remains to be elucidated, evidence accumulates that EVs contain numerous signalling molecules inside and on their cell membrane. The data suggests that fraxinellone alleviated the clinical and histologic features of inflammatory arthritis in CIA mice. Fraxinellone suppressed the expression of interleukin-17, and T helper 17 cell-related transcription factors (RORγt and phosphorylated STAT3) in CD4+ T cells. CD19+B cells showed lower expression of activation-related markers were reduced in the presence of fraxinellone. Osteoclastogenesis after fraxinellone treatment was evaluated by staining with tartrate-resistant acid phosphatase (TRAP) and by measuring the mRNA levels of osteoclastogenesis-related genes.

**Disclosures:** Fraxinellone alleviated the clinical and histologic features of inflammatory arthritis in mouse. The therapeutic effect of fraxinellone was associated with the inhibition of cellular differentiation and activation. The data suggests that fraxinellone could be a novel treatment for inflammatory arthritis, including rheumatoid arthritis.

**REFERENCES:**


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**AB0132**

**FRAXINELLONE ATTENUATES RHEUMATOID INFLAMMATION IN MICE**

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**Background:** Fraxinellone is isolated from Dictamnus dasycarpus, a traditional herbal medicine that attenuates inflammatory conditions.1,2 Recent studies have suggested that fraxinellone has a potential therapeutic effect in animal models with inflammatory diseases.3,4

**Objectives:** We aimed to evaluate the therapeutic effect of fraxinellone on inflammatory arthritis and identify the underlying mechanisms.

**Methods:** Fraxinellone (7.5 mg/kg) or a vehicle control was injected into mice with collagen-induced arthritis (CIA). The severity of arthritis was evaluated clinically and histologically. The differentiation of CD4+ T cells and CD19+B cells was investigated in the presence of fraxinellone. Osteoclastogenesis after fraxinellone treatment was evaluated by staining with tartrate-resistant acid phosphatase (TRAP) and by measuring the mRNA levels of osteoclastogenesis-related genes.

**Results:** Fraxinellone attenuated the clinical and histologic features of inflammatory arthritis in CIA mice. Fraxinellone suppressed the expression of interleukin-17, and T helper 17 cell-related transcription factors (RORγt and phosphorylated STAT3) in CD4+ T cells. CD19+B cells showed lower expression of activation-related markers were reduced in the presence of fraxinellone. Osteoclastogenesis after fraxinellone treatment was evaluated by staining with tartrate-resistant acid phosphatase (TRAP) and by measuring the mRNA levels of osteoclastogenesis-related genes.

**Conclusions:** Fraxinellone alleviated synovial inflammation and osteoclastogenesis in mice. The therapeutic effect of fraxinellone was associated with the inhibition of cellular differentiation and activation. The data suggests that fraxinellone could be a novel treatment for inflammatory arthritis, including rheumatoid arthritis.

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