anti-proliferation effects of different treatments. To further study the potential mechanism, TNF-α-induced in vitro model was applied. With different treatments, cell proliferation was detected using MTS, meanwhile, cell cycle distribution and apoptosis were examined by flow cytometric analysis. Western blotting and real-time quantitative PCR were conducted to evaluate many molecules that involved in interested pathways like COX-2/TxA2 pathway and AKT/FOXO3a pathway.

**Results:** The paw swelling volume and histological data indicate that 18β-GA administration attenuates arthritis severity in rats with CIA. Lower level of IL-1β, IL-6, and TxB2 were observed in serum of 18β-GA group as compared with model group. In addition, synovial immunohistochemistry data shows that 18β-GA decreased about half of PCNA intensity induced by collagen. However, in vivo, all data exhibited no significant differences among groups with monotherapy and combination therapy. In vitro, 18β-GA inhibited the mRNA and protein levels of COX-2 and TxAS that induced by TNF-α in MHTA cell line. Both p-JNK and NF-κB (p50) were inhibited by 18β-GA as well as TxAS siRNA transfection. Moreover, 18β-GA inhibited MHTA proliferation in a time- and dose-dependent manner from MTS assay. Flow cytometric analysis revealed that 18β-GA induced cell apoptosis and caused G1-phase cell cycle arrest. Finally, AKT and FOXO3a were predominantly phosphorylated by TNF-α, whereas such effect was blocked by 18β-GA treatment.

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**References:**

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of adipokines. Many studies have shown increasing leptin level and reducing adiponectin level in patients with rheumatoid arthritis (RA) compared with healthy ones. On the other hand 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-matched persons of control group. The diagnosis of RA was established according to the ACR 2010. Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and albumin were measured. Serum leptin and adiponectin levels were studied by enzyme immuno assay using standard sets (DRG, Germany and ‘Organon’, 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 2.2 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 (ρ=0.33 and r=0.35) and adiponectin level was decreased (ρ=-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 patients with leptin levels above 44.7±9.4 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 with diabetic nephropathy.

Conclusions: Disadipokineemia 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


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 are involved in cell-cell communication and contain numerous signalling molecules inside and on their cell membrane. 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 (RORgamma and phosphorylated STAT3) in CD4+ T cells. CD19+B cells showed lower expression of activation-induced cytokine deaminase (AID) and Blimp1 after treatment with fraxinellone. The formation of FRAP-positive cells and the expression of osteoclastogenesis-related markers were reduced in the presence of fraxinellone. Inhibition of interleukin-17 and osteoclastogenesis was also observed in experiments using human peripheral mononuclear cells.

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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AB0132 FRAXINELLONE ATTENUATES RHEUMATOID INFLAMMATION IN MICE

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Background: Fraxinellone is isolated from Dictamus dasycarpus, a traditional herbal medicine that attenuates inflammatory conditions.1-3 Recent studies have suggested that fraxinellone has a potential therapeutic effect in animal models with inflammatory diseases.4,5

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 (RORgamma and phosphorylated STAT3) in CD4+ T cells. CD19+B cells showed lower expression of activation-induced cytokine deaminase (AID) and Blimp1 after treatment with fraxinellone. The formation of FRAP-positive cells and the expression of osteoclastogenesis-related markers were reduced in the presence of fraxinellone. Inhibition of interleukin-17 and osteoclastogenesis was also observed in experiments using human peripheral mononuclear cells.

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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