disease activity group; 9 from remission and 19 from HC) the level of IL-6 in serum was evaluated.

Results: Patients with RA in comparison to HC have had a lower *FCER1G* methylation (0.98 [0.73-1.46] vs 1.96 [1.44-3], $p<0.00001$; median [interquartile range]) and miR-106b (0.79 [0.49-1.68] vs 1.54 [0.88-2.51], $p=0.008$) and miR-17 (1.26 [0.41-2.04] vs 2.44 [2.09-3.47], $p=0.0001$) expressions. No difference in methylation between high and remission RA groups was found. MiR-106b and miR-17 expressions were different between RA patients with high disease activity and remission ($p=0.009$ and $p=0.003$, respectively), however a high disease activity group was not different to HC ($p=0.82$ and $p=0.12$, respectively). Detailed results are presented in Table 1. A strong correlation between IL-6 levels and *FCER1G* methylation ($r_s = -0.46$) was found.

Table 1. Methylation and expression between patients divided by disease activity in compare to controls.

	High disease activity, n=29	Remission, n=21	HC, n=24
<i>FCER1G</i> methylation	1.11 [0.83-1.52]	0.96 [0.61-1.18]	1.96 [1.44-3]
miR-106b expression	1.36 [0.63-1.76]	0.54 [0.19-1.19]	1.54 [0.88-2.51]
miR-93 expression	0.63 [0.49-1.21]	0.59 [0.15-1.5]	1.03 [0.65-1.38]
miR-17 expression	1.46 [1.05-2.54]	0.34 [0.11-1.26]	2.44 [2.09-3.47]

Data are given by median [interquartile range]. *FCER1G*, Fc receptor gamma chain gene; HC, healthy controls; miR, micro-RNA; RA, rheumatoid arthritis patients.

Conclusion: *FCER1G* methylation was found as a new epigenetic marker of RA, which is independent of disease activity and may be associated with IL-6 production. Plasma miR-17 and miR-106b can be considered as a novel molecular biomarkers of disease severity in RA.

FcRγ may plays a significant role in RA pathogenesis and *FCER1G* gene methylation was found as a new, epigenetic and promising marker of RA.

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AB0097

TISSUE CYTOKINES AS NEW DIAGNOSTIC BIOMARKER OF BONE METABOLISM DISORDERS IN RHEUMATOID ARTHRITIS

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Background: Bone mineral density and proteins/peptides determination in blood and urine as markers of bone resorption and formation are currently used to diagnose osteoporosis (OP) and metabolic bone diseases. Recent evidence suggests that in RA changes in the secretion of hormones of white adipose tissue can be revealed [1,2,3,4].

Objectives: To study the clinical and diagnostic value of serum fetuin A, nesfatin, hemerin, leptin, adiponectin, resistin, visfatin determination in RA patients complicated by OP.

Methods: We examined 88 women with documented diagnosis of RA and mean disease duration of 6.56±0.88 years. We used EULAR/ARA 2010 criteria to diagnose the patients. Female patients with II degree of disease activity (DAS28), Steinbrocker stage II (erosive), rheumatoid factor- and anti-cyclic-citrullinated peptide antibody-positive were prevalent. We excluded patients who had surgery or developed an infection within the last 8 weeks, pregnant and breast-feeding women, those with severe heart, liver or kidney disease, immune deficiency, leucopenia or chronic infection.

A control group of 45 healthy females aged of 25 and 59 years were included in the study. There were no reported findings of joint pain and RA symptoms in the group. The groups were adjusted for age ($p>0.05$) and showed no statistically significant differences.

We measured serum fetuin A, nesfatin, hemerin, leptin, adiponectin, resistin, visfatin levels (μg/ml) using ELISA commercial test systems. We used spectrophotometer with wavelength of 450nm to detect the test results («Multiskan» immunoenzyme analyzer, Finland). We plotted a curve using computer software. We diagnosed OP using dual-energy X-ray absorptiometry with LUNAR DPX PRO (GE, USA).

Results: At the first stage, the level of pro-inflammatory cytokines was studied in a group of healthy individuals. Then, the reference values of these indicators were measured as $M \pm 26$. Patients with OP and RA had significantly higher levels of serum pro-inflammatory cytokines ($p<0.001$). For example, mean serum Adiponectin levels in RA patients who had normal bone density and had no OP were 35.21±0.6 μg/ml. Mean serum Adiponectin levels in RA/OP patients with low bone mineral density were 52.42±0.69 μg/ml. Adiponectin levels of 44 μg/ml and higher were associated with osteoporosis. Adiponectin levels of 43.9 μg/ml and lower were associated with normal bone density. Other pro-inflammatory cytokines have demonstrated similar dynamics of level serum.

Conclusion: Thus, we revealed that fetuin A, nesfatin, hemerin, leptin, adiponectin, resistin, visfatin levels depend on osteoporosis presence in RA patients. The test may be used to reduce the risk of low-energy fractures and to improve the quality of life in RA.

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AB0098

THE EFFECT OF MALT1-DEFICIENCY ON THE EFFECTOR PHASE OF EXPERIMENTAL AUTOIMMUNE ARTHRITIS

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Background: The paracaspase Malt1 is a cysteine protease, which forms a complex leading to the activation of the in proinflammatory transcription factor NF-κB in lymphocytes with CARMA1 and Bcl10. Previously, we showed that the myeloid equivalent of CARMA1, Card9 is important in neutrophils in Fcγ receptor-mediated cytokine release together with Bcl10 and Malt1. In line with these findings, we observed a significant decrease in the severity of autoantibody-triggered arthritis in the absence of Card9 and Bcl10.

Objectives: Our aim was to directly investigate whether the genetic deficiency of Malt1, the third component of the complex altered the process of the K/BxN serum transfer arthritis (that resembles to the effector phase of rheumatoid arthritis).

Methods: We used wild type and Malt1^{-/-} mice for our experiments. Autoantibody-mediated arthritis was induced by a single intraperitoneal injection of K/BxN serum. Clinical signs of joint inflammation were scored on a scale based on the cardinal inflammatory clues for two weeks. Ankle thickness was measured by a spring-loaded caliper.

Results: Similar to the deficiency of the other two components of the complex, Malt1^{-/-} mice showed a partial, but significant decrease in the macroscopic joint inflammation compared to arthritic serum-treated wild type animals during the entire experimental process. In line with this phenomenon, Malt1^{-/-} animals had reduced autoantibody-triggered ankle thickening.