References:

may plays a significant role in RA pathogenesis and FcRγ gene methylation (0.98 [0.73-1.48]) vs 1.96 [1.44-3], p=0.008) compared to healthy controls; miR, micro-RNA; RA, rheumatoid arthritis patients.

Results: 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.

Conclusions: FcER1G Fc receptor gamma chain gene; HC, healthy controls; miR, micro-RNA; RA, rheumatoid arthritis patients.

Disclosure of Interests:

Marek Ciesla: None declared, Bogdan Kolarz: None declared, Magdalena Dryglewska: None declared, Maria Majdan Consultant of: PRO (GE, USA).

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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 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 Fcy 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.