Comparison between the altered peripheral replicative study of GWAS-associated gene expression levels of the studied miRNAs were calculated by 2-ΔΔCt technology and compared to healthy controls (HCs). Relative changes of levels of miR-146a and miR-155 in whole PB were determined by qPCR (SybrGreen). 29 (46.03%) of the RA patients showed overexpression of miR-146a in PB (p=0.074). In the group of SLE patients the PB levels of miR-146a were overexpressed (0.5 - 0.8 fold) were SERBP1, ATXN7, PSIP1, ZNF385D, SIPA1L1.

Conclusion: This combined genetic and functional meta-analysis elucidated novel genes, functional networks and pathways for psoriasis. These results will lead to important insights into the immunopathogenesis and treatment of psoriasis.

REFERENCES

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AB0017 COMPARISON BETWEEN THE ALTERED PERIPHERAL BLOOD MiRNA EXPRESSION IN PATIENTS WITH RHEUMATOID ARTHRITIS AND SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: MicroRNAs (miRNAs) are a class of small, non-coding RNAs that negatively regulate gene expression at posttranscriptional level. Altered miRNA expression in the circulation has been described in inflammatory joint diseases such as rheumatoid arthritis (RA) as well as in systemic rheumatic diseases, such as systemic lupus erythematosus (SLE). miR-146a and miR-155 have been found to regulate key signaling pathways involved in the pathogenesis of RA and SLE [1-2].

Objectives: To compare the expression levels of miR-146a and miR-155 in peripheral blood (PB) from RA and SLE patients in regard to their use as disease biomarkers.

Methods: 63 RA patients and 40 SLE were included in the study. The expression levels of miR-146a and miR-155 in whole PB were determined by qPCR (SybrGreen technology) and compared to healthy controls (HCs). Relative changes of gene expression levels of the studied miRNAs were calculated by 2^-ΔΔCt method. SPSS was used for statistical analysis.

Results: 29 (46.03%) of the RA patients showed overexpression of miR-146a in PB when compared to HCs, but the levels were not statistically significant to differentiate patients from HCs (p=0.365). 34 (53.97%) of the RA patients didn’t show a statistically significant expression of miR-155 in the PB when compared to HCs and PB expression of miR-155 couldn’t be differentiated for RA from HCs (p=0.074). In the group of SLE patients the PB levels of miR-146a were overexpressed in 25 (62.5%) and levels of miR-155 were increased in 20 (50.0%) of the patients (p=0.06).

Conclusion: Although miR-146a and miR-155 are involved in key signaling pathways in the pathogenesis of RA their whole PB expression could not fully reflect the local pathological process and thus to differentiate patients from HCs. The expression of both miRNAs in whole PB of SLE samples showed a possibility for discriminating patients from HCs. Further analysis with larger sets is needed to confirm if altered systemic miRNA expression levels could be used in the clinical practice as disease biomarkers.

REFERENCES

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Disclosure of Interests: None declared


AB0018 TRANSCRIPTOMIC CHARACTERIZATION OF SINGLE PATHOGENIC MEMORY B CELLS IN RHEUMATOID ARTHRITIS

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Background: Autoantigen-specific B lymphocytes are crucial players in the development of rheumatoid arthritis (RA). Despite the fact that ACAs+ autoreactive memory B cells have been observed in the majority of RA patients for a long time, their properties still remain enigmatic.

Objectives: In this study, we aimed to reveal transcriptomic nature of single pathogenic memory B cells from RA patients.

Methods: Single-cell full-length RNAseq data was generated from ACAs+ and control memory B cells from 7 human donors with established rheumatoid arthritis and subjected to transcriptome analyses as well as B cell receptor (BCR) assembly.

Results: Both transcriptome and BCR data was successfully generated from the majority of the sequenced cells. The success rate depended on chosen sequencing parameters as well as on a BCR assembling algorithm that was used. We observed expression of genes defining memory B cell population in our data as well as pathways crucial for their function.

Conclusion: Taken together, gene expression and BCR sequence data obtained from the same single cell can give novel insights into the biology and role of B cells in the development of the disease.

AB0019 REPPLICATIVE STUDY OF GWAS-ASSOCIATED CANDIDATE GENE LOCI IN PATIENTS WITH OSTEARTHRITIS FROM THE BASHKORTOSTAN REPUBLIC

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Background: Osteoarthritis (OA) is one of the most common joint diseases and a global socially significant problem. Currently, new methods of early diagnosis of the disease are being developed, before the development of destructive processes in various tissues of the joint. The leading role in determining the pathophysiology of OA is given to the identification of genetic and epigenetic mechanisms.

Objectives: The main goal of the study was replicative analysis of the associations of loci associated with OA according to the data of genome-wide analysis of associations (GWAS) located near the DOT1L, ALDH1A1, GN3L, GLT8D1, ASTN2, FILIP1, SENP5, NCOA3, DWVA, CHA1 genes with various localization OA.

Methods: DNA samples from 410 women were used (mean age 45.45 ± 2.35). Patients with OA was carried out in accordance with the criteria of the American Association of Rheumatology (1990), the debut of the disease before the age of 55 years and radiographic confirmation. For genotyping, PCR-RFLP analysis using KASP® technology was used. As a calculation tool, MS Office Excel 2007 was used. The results were presented in the form of a table. Statistica v.8.2 (StatSoft), SPSS v.13 (SPSS Inc) software packages are used.

Results: We conducted replicative analysis of the 12 loci that were the most significantly associated with OA in the results of GWAS study, among which rs12982744 and rs2302061 (DOT1L), rs4836732 (ASTN2), rs9350591 (FILIP1 &