Moreover, it has been reported that pre-mRNA or mRNA levels did not correlate with the degree of each DEGs was obtained by analyzing the topological structure of STRING online software. Cytoscape software was utilized to visualize PPI and analyze Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis of DEGs. Interactions of proteins encoded by DEGs were discovered by Protein-protein interaction network (PPI) using STRING online software. Cytoscape software was utilized to visualize PPI and the degree of each DEGs was obtained by analyzing the topological structure of the PPI network.

**Results:** A total of 611 DEGs were found to be differentially expressed in psoriasis. GO analysis revealed that up-regulated DEGs were mostly associated with defense and response to external stimuli while down-regulated DEGs were mostly associated with metabolism and synthesis of lipids. KEGG enrichment analysis suggested they were mainly enriched in IL-17 signaling, Toll-like receptor signaling and PPAR signaling pathways. Cytokine-cytokine receptor interaction and lipid metabolism. In addition, top 9 key genes (CXCL10, OASL, IFIT1, IFIT3, RSAD2, MX1, OAS1, IFI44 and OAS2) were identified through bioinformatics analysis of GEO databases.

**References:**

**Conclusion:** This study represents a comprehensive evaluation of the role of differentially expressed genes in psoriasis, leading to a loss of function phenotype and, as a consequence, reduced levels of IFI1 protein and activity that protect against autoimmunity. Structural analysis of rare shared genetic susceptibility or protection loci may provide insight into our understanding of the pathophysiology of autoimmune diseases and the research findings may affect the better management of the diseases under study.

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**Figure 1.** Structural representation of the interferon-induced helicase C domain-containing protein 1 (Homo sapiens) structure (4GL2 from Protein Data Bank) (in green) bound to the double stranded RNA (in orange-yellow). The location of the mutation (I923V) is highlighted in pink. The proximity of the aminoacid 923 to the nucleotide is apparent.
Methods: The genes were selected according to previous studies describing their possible contribute in the modulation of clinical and laboratory features.

Objectives: We confirm the associations between five SNPs, already studied in RA, and PsA susceptibility, suggesting a common inflammatory pathway in chronic inflammatory rheumatological diseases. Moreover, we show how the genotyping of only few associated SNPs could help to define a genetic risk profile for PsA development.

REFERENCES:


Conclusion: We confirm the associations between five SNPs, already studied in RA, and PsA susceptibility, suggesting a common inflammatory pathway in chronic inflammatory rheumatological diseases. Moreover, we show how the genotyping of only few associated SNPs could help to define a genetic risk profile for PsA development.

Disclosure of Interests: None declared

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Figure 1: Number of risk alleles in patients and controls: rs7574865 (STAT4), rs33980500 (TRAF3IP2), rs6920220 (TNFAIP3) and rs27524 (ERAP1) SNPs.

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