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Identification of the Expression of Top Deregulated MiRNAs in Rheumatoid Arthritis and Osteoarthritis

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Abstract : Introduction: Rheumatoid arthritis (RA) is an inflammatory, autoimmune disorder with progressive joint damage. Osteoarthritis (OA) is a degenerative disease of the articular cartilage that shows multiple clinical manifestations or symptoms resembling those of RA. Genetic predisposition is believed to be a principal etiological factor for RA and OA. In this study, we aimed to measure the expression of the top deregulated miRNAs that might be the cause of pathogenesis in both diseases, according to our latest NGS analysis. Six of the deregulated miRNAs were selected as they had multiple target genes in the RA pathway, so they are more likely to affect the RA pathogenesis.Methods: Eighty cases were recruited in this study; 45 rheumatoid arthiritis (RA), 30 osteoarthiritis (OA) patients, as well as 20 healthy controls. The selection of the miRNAs from our latest NGS study was done using miRwalk according to the number of their target genes that are members in the KEGG RA pathway. Total RNA was isolated from plasma of all recruited cases. The cDNA was generated by the miRcury RT Kit then used as a template for real-time PCR with miRcury Primer Assays and the miRcury SYBR Green PCR Kit. Fold changes were calculated from CT values using the ΔΔCT method of relative quantification. Results were compared RA vs Controls and OA vs Controls. Target gene prediction and functional annotation of the deregulated miRNAs was done using Mienturnet. Results: Six miRNAs were selected. They were miR-15b-3p, -128-3p, -194-3p, -328-3p, -542-3p and -3180-5p. In RA samples, three of the measured miRNAs were upregulated (miR-194, -542, and -3180; mean Rq= 2.6, 3.8 and 8.05; P-value= 0.07, 0.05 and 0.01; respectively) while the remaining 3 were downregulated (miR-15b, -128 and -328; mean Rq= 0.21, 0.39 and 0.6; P-value= <0.0001, <0.0001 and 0.02; respectively) all with high statistical significance except miR-194. While in OA samples, two of the measured miRNAs were upregulated (miR-194 and -3180; mean Rq= 2.6 and 7.7; P-value= 0.1 and 0.03; respectively) while the remaining 4 were downregulated (miR-15b, -128, -328 and -542; mean Rq= 0.5, 0.03, 0.08 and 0.5; P-value= 0.0008, 0.003, 0.006 and 0.4; respectively) with statistical significance compared to controls except miR-194 and miR-542. The functional enrichment of the selected top deregulated miRNAs revealed the highly enriched KEGG pathways and GO terms. Conclusion: Five of the studied miRNAs were greatly deregulated in RA and OA, they might be highly involved in the disease pathogenesis and so might be future therapeutic targets. Further functional studies are crucial to assess their roles and actual target genes.

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