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Short Communication

Potential Role of Mirnas in Rheumatoid Arthritis

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Rheumatoid Arthritis (RA) is a chronic multifactorial disease which is affecting 1% of the world population. It is a persistent, debilitating synovial joint disease characterized by increased synovial cells division, inflammation, cartilage destruction and bone deformity [1-3]. Infiltration of inflammatory cells and growth factors plays a crucial role in formation of synovial pannus [4]. The abnormal activity of immune cells results in dysregulated immune response in the inflamed synovial membrane which ultimately contributes to the cartilage destruction and bone erosion. In spite of significant therapeutic development, diagnosis of RA still remains unexplained. Hence understanding the molecular mechanism of RA, as a holistic approach, becomes a key technique for researchers to connect it with various factors like environment, stochastic, transcription factors and miRNAs. Recent investigation reports that miRNAs have the potential to become the next generation diagnostics and therapeutics. Moreover, it is attracting tremendous attention from the biological and biomedical research communities. Understanding the functioning of the miRNAs is becoming crucial for identifying the underlying role in different disease pathogenesis [5]. MiRNAs are evolutionarily conserved sequences, approximately 22 nucleotides in length, which regulates about 30% of the mammalian protein-encoding genes. Specific miRNAs when bound with target mRNAs can inhibit its expression by a variety of mechanisms [6]. This type of repression is directly dependent on sequence complementarity between seed region of miRNA and target sequence of mRNA. Partial complementarity may result in translation repression or target mRNA instability, whereas perfect complementarity between the miRNA and mRNA heteroduplex will cause target mRNA destruction [7]. MiRNAs have been identified as key regulators of major cellular processes like cell division, death, cellular metabolism, intracellular signaling, immunity, and cell movement [8-12]. Any abnormal miRNA expression will affect the above critical processes, and will therefore lead to various disorders.

Report suggests that miRNAs plays important role in inflammatory responses, cell proliferation of synoviocytes, and production of MMPs in RA [13]. Watanabe et al., (2015) reports, up regulation of miR-146a and miR-155 in the synovium, as a result of inflammatory signals, apparently plays a role in RA pathogenesis by regulating the inflammation [14]. Also, macrophages and T cells which are key regulatory molecules in RA were regulated by MiR-146a. This miR-146a is found to be expressed in several CD3+ T cell subsets and CD79a+ B cells and it is associated with CD68+ macrophages and CD4+T cells (17). Therefore, identification of unique miRNAs that are expressed patterns in RA can be perceived as molecular diagnostic markers which can shed light in understanding the role of miRNAs in RA pathogenesis. It paves a path towards new gene therapy approaches for handling RA and other autoimmune arthritis.

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