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MiR-146a Upregulation of Phagocytosis in Human Macrophages

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Research & Scholarship SYMPOSIUM

MiR-146a Upregulation of Phagocytosis in Human Macrophages

Sjögrens Syndrome (SjS) is an autoimmune disease that attacks exocrine glands such as salivary and lacrimal glands resulting in severe dryness of the mouth and eyes. Previous studies have linked increased microRNA-146a (miR-146a) expression in peripheral blood mononuclear cells in SjS patients compared to healthy controls. MicroRNAs (miRNAs), small non-coding RNA molecules that post-transcriptionally regulate gene expression, are known to play key regulatory roles in immune responses and have been implicated in a growing number of autoimmune disorders. Further investigation into the role of increased miR-146a expression in SjS revealed links to several immune functions including phagocytosis. Our goal was to further examine the relationship between miR-146a expression and the rate of phagocytosis in human macrophages by using apoptotic human cells as a phagocytic target. We hypothesized that upregulation of miR-146a would increase phagocytic activity of differentiated THP-1 human monocytes. To quantify phagocytic activity, a pH-sensitive fluorescent dye (pHrodo) was used to indicate the E. coli or apoptotic Jurkats that had been phagocytosed. THP-1 cells were transfected with miR-146a and differentiated into macrophages. Phagocytic activity was observed by incubating fluorescently labeled E.coli or apoptotic Jurkat cells with miR-146a transfected and mock transfected THP-1 cells for 2-4 hours. Fluorescence intensity was quantified using a fluorescent plate reader (E. coli) and microscopy (apoptotic Jurkats). MiR-146a-transfected THP-1 cells exhibited significantly increased phagocytic activity of fluorescently labeled E. coli ($P < 0.001$) and apoptotic Jurkats. Knockdown of TRAF6, a gene target of miR-146a, did not impact the phagocytic activity. MiR-146a appears to upregulate phagocytic activity in human THP-1 cells through an unknown mechanism. Further studies are in progress to determine the mechanism by which miR-146a upregulates phagocytosis.