Abstract
Although the precise pathogenic mechanisms that lead to Rheumatoid Arthritis (RA) joint damage remain unclear, macrophages and CD4 T cells and B cell are three major immune cellular components involved in this process. IL-6 levels are known to be significantly elevated in some RA patients, but it was considered as a marker of inflammation. IL-6 is a unique inducer of IL-21 production by naïve and memory CD4 T cells. IL-6 has recently emerged as a therapeutic target for RA and blocking IL-6 signals improved disease. The mechanisms however are unknown. We propose that IL-6 (provided by macrophage) induces the production of IL-21 in CD4 T cells, and IL-21 acts on B cells to promote autoantibody products. We will examine the effect of IL-6 on IL-21 expression in RA.