Interleukin 22, a Potential Therapeutic Target for Rheumatoid Arthritis

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To the Editor:

A recent report by Da Rocha, et al indicated that levels of interleukin 22 (IL-22) were increased in patients with rheumatoid arthritis (RA) compared with controls; levels of IL-22 correlated with Disease Activity Score (DAS28) and Clinical Disease Activity Index measures, rheumatoid factor positivity was correlated with higher levels of IL-22 in patients with RA, and the presence of bone erosions was associated with high IL-22 levels. These findings suggest that IL-22 may be a good biomarker for assessment of activity in RA, and IL-22 seems to be a potential therapeutic target for RA.

Other studies have indicated the similar relationship between IL-22 and RA, where serum IL-22 levels were found to be increased in patients with RA compared with controls,1,2,4,5 and high IL-22 levels correlated with bone erosions. The IL-22 concentration in synovial fluid was higher in patients with RA compared with controls.2,5 In patients with RA, Th17 cells were recognized to produce higher IL-22 levels. Th22 cells also produced IL-22 and the expression of Th22 cells. IL-22 were significantly elevated in RA patients.5,6 More importantly, Th17/22 cells showed positive correlations with IL-22, C-reactive protein, and DAS28 data.6 In addition, natural killer (NK)-22 cells in vitro can secrete higher levels of IL-22 and tumor necrosis factor-α (TNF-α), and NK-22 supernatant can induce the proliferation of RA fibroblast-like synoviocytes (FLS); however, addition of IL-22 antibody plus TNF-α antibody inhibited the proliferation of FLS induced by the NK-22 supernatant. In vitro, human recombinant IL-22 (rhIL-22) significantly increased proliferation of RA synovial fluid and FLS and production of monocye chemotactrant protein-1.7,8, but an inhibitory effect of anti-IL-22R antibody on proliferation of FLS induced by rhIL-22 was found in RA.7 Moreover, an experimental arthritis model (IL-1Ra−/−) demonstrated that IL-22−/− mice significantly increased proliferation of RA synovial fluid and FLS and production of monocye chemotactrant protein-1.7,8, but an inhibitory effect of anti-IL-22R antibody on proliferation of FLS induced by rhIL-22 was found in RA.7


These findings suggest therapeutic potential for patients with RA, and suggest a role for IL-22 in development of RA. Further studies are needed to clarify the role of IL-22 in RA. Therapeutic agents targeting IL-22 might result in innovative new therapies for RA.