IL-23 and IL-17 in ankylosing spondylitis

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Sir,

We read with interest the study published by Wang et al. demonstrating the elevated levels of IL-23 and IL-17 in the serum of patients with active Ankylosing Spondylitis (AS) [1].

This illustrates the potential central role of the IL-23/IL-17 axis in AS [2], and is concordant with other findings, such as IL-23 receptor polymorphisms in AS, as well as in Crohn's disease or in psoriasis [3], sharing the concept of spondyloarthropathy. Elevated IL-17 levels were found in the serum of AS patients [4], and increased numbers of Th 17 cells in the peripheral blood of patients with seronegative spondylarthritides (and not RA) were reported by Jandus [5], as well as increased frequencies of IL-17 positive CD4+ T cells in PBMC from patients with AS [6].

Moreover, for the first time, a higher expression of IL-23 p19 mRNA in PBMC of AS patients as well as enhanced production of IL-17 in the supernatants of PBMC cultured in the presence of recombinant IL-23 was found by Wang et al. [1]. Previous studies did not demonstrate elevated levels of p40 IL-23 in the serum of AS patients, compared with controls, whereas, in some of them, synovial fluid levels were higher than serum levels [7]. This gives the central role to p40 IL-23 in the pathophysiological axis, and a potential therapeutic target in AS. Nevertheless, it has been demonstrated that targeting p40 IL-12/23 with a

monoclonal antibody (ustekinumab) may be effective in treating psoriatic arthritis [8], psoriasis, and Crohn's disease.

According to the results of Wang et al. [1], one may speculate that p19 IL-23 blockade may be even more efficacious, and may represent a new therapeutic pathway in AS.

Conflict of interest statement None.

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