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**INTERLEUKIN-6 IS NOT A CRUCIAL REGULATOR IN
TUMOUR NECROSIS FACTOR-MEDIATED BILATERAL
SACROILIITIS**

S Hayer, B Niederreiter, I Nagelreiter, J Smolen, K Redlich *Department of Internal Medicine III, Division of Rheumatology, Medical University of Vienna, Vienna, Austria*

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Objective To investigate the role of interleukin 6 (IL6) in the pathogenesis of bilateral erosive sacroiliitis in hTNFtg mice, an animal model of ankylosing spondylitis (AS).

Methods We crossed IL6^{-/-} mice with hTNFtg animals to generate IL6^{-/-}hTNFtg mice. Histopathological sections of bilateral sacroiliac joints were evaluated in 14-week-old IL6^{-/-}hTNFtg mice and compared with age-matched hTNFtg mice using H&E, TRAP and toluidine blue stainings. Serum levels of mRNA isolated from sacroiliac joints from wild-type, hTNFtg, IL6^{-/-} and IL6^{-/-}hTNFtg mice were measured. hTNF and mIL6 were also measured from these animals. Quantitative PCR was used to determine expression levels of genes related to inflammation and tissue degradation.

Results Overexpression of hTNF in hTNFtg mice led to the upregulation of mRNAs from tissue degrading enzymes such as matrix metalloproteinase (MMP)-3, -9 and -13 as well as osteoclast-specific cathepsin K and TRAP at sites of bilateral sacroiliitis. In IL6^{-/-}hTNFtg mice, the expression levels of these marker genes did not differ in inflamed sacroiliac joints when compared with hTNFtg mice. Moreover, the absence of IL6 did not alter the histopathology of bilateral sacroiliitis characterised by inflammation, bone erosion and cartilage damage when compared with hTNFtg littermates.

Conclusion IL6 is not a crucial regulator in TNF-mediated bilateral sacroiliitis, and this finding questions the potential of IL6 blockade as a new therapy in patients with AS.