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# *IL-12* CYTOKINE FAMILY: NEW PLAYERS IN PATHOGENESIS OF OSTEOPOROSIS

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The interleukin 12 *(IL-12)* family is unique in having the only heterodimeric cytokines, including *IL-12*, *IL-23*, *IL-27* and *IL-35*. Despite sharing many structural features and molecular partners, *IL-12* family cytokines have diverse functional effects. Both *IL-12* and *IL-23* are pro-inflammatory, and have been implicated in a number of autoimmune diseases. Monoclonal antibody against *IL-12/IL23* (Ustekinumab) has been used for treatment of psoriatic arthritis. *IL-23* deficient mice were found to be protected against development of collagen induced arthritis and moreover *IL-17* producing $CD4^+$ T cells were absent in *IL-23* deficient mice. The remaining two cytokines of the family, *IL-27* and *IL-35* are pre-dominantly anti-inflammatory in nature. Studies have shown that deletion of *IL-27* receptor in mice leads to excessive *Th17* responses in experimental autoimmune encephalomyelitis (EAE). *IL-27* also has been reported to provide protection against collagen-induced arthritis and rheumatoid arthritis by suppressing *Th17* cells and augmenting T regulatory cell differentiation. Apart from *IL-27*, *IL-35*, serum levels are found to be quite low in conditions of rheumatoid arthritis patients. While all these cytokines have diverse functions, their role in post-menopausal osteoporosis was not looked into. Our group has determined the role of *IL-27* cytokine and *IL-23* neutralizing antibody in estrogen deficiency induced bone loss conditions. Our studies revealed the osteoprotective effects of *IL-27* and anti-*IL 23* antibody by suppression of *Th17* differentiation, the major osteoclastogenic T helper subset and restoration of ovariectomy induced deterioration of trabecular bone microarchitecture. Our studies form a strong basis for using humanized *IL-27* or neutralizing *IL-23* antibody towards the treatment of postmenopausal osteoporosis.

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