

Frontline Science: Human bone cells as a source of IL - 27 under inflammatory conditions: role of TLRs and cytokines

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Abstract

IL - 27 regulates immune responses as well as hematopoiesis and bone remodeling, but its cellular sources in the bone remain unknown. In this study, we investigated whether osteoclasts and osteoblasts—the 2 cell types orchestrating bone homeostasis—could be a source of IL - 27 and identified stimuli that induce its expression in vitro. We observed that human monocyte - derived osteoclasts expressed a broader range of TLRs than did human primary osteoblasts and that both cell types exhibited a differential induction of IL - 27 expression in response to TLR or cytokine stimulation. Whereas several TLR agonists, notably TLR4 and TLR7/8 agonists, induced substantial expression of IL - 27 by osteoclasts, stimulation of osteoblasts with agonists of TLR3 and/or TLR4—the 2 TLRs selectively expressed by these cells—resulted in no or low IL - 27 expression. In addition, IL - 27 increased TLR3 expression in osteoclasts and enhanced poly(I:C) - mediated induction of IL - 27 in these cells. IFN - γ , when combined with either IL - 1 β plus TNF - α , IL - 11, or CNTF, induced significant levels of IL - 27 in osteoclasts but not in osteoblasts. In the latter cells, the addition of type I IFN, together with proinflammatory cytokines, was necessary to induce substantial levels of IL - 27. Immunohistochemical studies of inflamed and remodeling bone tissue, including cases of infectious osteomyelitis and bone metastases, provided evidence that osteoclasts, osteoblasts, and occasionally osteocytes or chondrocytes, could express IL - 27 in situ. This autocrine production of IL - 27 by TLR - or cytokine - activated bone cells might constitute a negative - feedback mechanism to limit bone erosion and to dampen T cell-mediated immune pathology during bone inflammation.

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