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The cross-talk between inflammatory cytokine and osteoclast in rheumatic arthritis

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Abstract

Rheumatic arthritis (RA) is a chronic, systemic inflammatory disorder that affects many tissues and organs, but principally attacks synovial joints. A variety of cytokines, such as TNF-α, and B cells play crucial role in the pathogenesis of rheumatic arthritis. The balance between ostoblasts and osteoclasts derived from monocytes/macrophages fusion keeps the homeostasis of bone, and now over-activation of osteoclasts, which leads to the destruction of articular cartilage and bone, has been found in rheumatic arthritis. The symptoms are significantly relieved in the patients treated with TNF-α inhibitors such as Anti-TNF-α Ab, while there are still limits using this treatment in clinic because of the side effects and drug tolerance. New therapeutic targets need to be identified to improve the treatment efficiency of rheumatic arthritis. Our investigation is try to clarify the detail molecular mechanisms in the cross-talk between cytokines or immune cells and osteoclast in rheumatic arthritis, and we found that TNF-α promotes up-regulation of the membrane calcium tunnel protein which can induce formation and activation of osteolasts. Our data will provide mechanistic insight to the pathogenesis of rheumatic arthritis and also outline new potential target for the development of the drug to treat rheumatic arthritis.