Choloroquine reduces formation of bone resorbing cells in murine osteoporosis

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Bone homeostasis requires precise balance between deposition of new bone by osteoblasts and resorption of old bone by osteoclasts. Bone diseases, including osteoporosis and rheumatoid arthritis, are the result of increased osteoclast activity and formation, which allows bone resorption to outpace deposition.

In this issue of the *Journal of Clinical Investigation*, Brendan Boyce and colleagues at the University of Rochester evaluated the role of TNF receptor–associated receptor 3 (TRAF3) in promoting osteoclast formation. Mice lacking TRAF3 in osteoclast precursor cells had mild osteoporosis that was associated with increased osteoclast formation.

The authors found that chloroquine treatment increased TRAF3 in osteoclast precursor cells and limited osteoclast generation. Furthermore, treatment of mouse models of osteoporosis with chloroquine inhibited osteoclast formation.

These studies implicate that therapies aimed at increasing TRAF3 in osteoclast precursor cells may limit bone loss for those with bone diseases.

More information: Chloroquine reduces osteoclastogenesis in murine osteoporosis by preventing TRAF3 degradation, *J Clin Invest*. DOI: 10.1172/JCI66947

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