

Response to: 'Denosumab, cortical bone and bone erosion in rheumatoid arthritis' by Rossini *et al*

Dear Editor,

We thank Rossini *et al* for their comments¹ on our recent study on inhibitory effect by denosumab on the progression of bone erosions in Japanese patients with rheumatoid arthritis (RA).² They addressed their observation from point of view of bone in patient with RA. We think their view brings treatment of RA closer to treatment of osteoporosis.

Activated osteoclasts decrease bone mineral density (BMD) and stimulate bone erosion in patients with RA.³ Receptor activator of nuclear factor kappa-B ligand (RANKL) promotes osteoclast differentiation, maturation, and activation.^{4–7} Denosumab is a fully human monoclonal antibody against RANKL that inhibits osteoclast formation, function, and survival. Denosumab treatment increases BMD in cortical and trabecular bone. In addition, denosumab has been shown to improve cortical bone microstructure in subjects with osteoporosis or low bone mass.^{8–9}

Increases in lumbar spine and total hip BMD and inhibition of progression of bone erosion have been observed with denosumab treatment in subjects with RA, and beneficial effects on cortical bone similar to those described in subjects with osteoporosis/low bone mass might be expected also in those with RA.^{1–10} Denosumab improves bone microstructure of both cortical and trabecular bone, its effects on osteitis as well as bone erosion are expected in patients with RA.

Denosumab is a unique treatment for patients with RA and low bone mass.

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Competing interests None declared.

Provenance and peer review Commissioned; internally peer reviewed.



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To cite Takeuchi T, Tanaka Y, Ishiguro N, *et al*. *Ann Rheum Dis* 2016;**75**:e71.

Accepted 3 August 2016



► <http://dx.doi.org/10.1136/annrheumdis-2016-210022>

Ann Rheum Dis 2016;**75**:e71. doi:10.1136/annrheumdis-2016-210022

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