ONLINE ISSN : 1880-1404
PRINT ISSN : 0916-717X

Biomedical Research on Trace Elements
Vol. 15 (2004) , No. 1 9-14

[PDF (650K)] [References]

Role of Zinc in Regulation of Osteoclastogenesis

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Abstract:
Zinc administration prevents bone loss in rats with ovariectomy, skeletal unloading, hydrocortisone treatment, adjuvant arthritics and diabetic condition in rats. Zinc has been shown to play a role in the preservation of bone mass by stimulating osteoblastic bone formation and inhibiting osteoclastic bone resorption in rat bone tissues. The cellular mechanism of zinc action in inhibiting osteoclastic bone resorption is reviewed. Zinc inhibits bone resorption which is stimulated by various bone-resorbing factors including parathyroid hormone (PTH), 1, 25-dihydroxyvitamin D_3 [1, 25$(OH)_2D_3$], and prostaglandin E_2 (PGE_2) in rat bone tissue culture in vitro. Also, zinc has an inhibitory effect on the bone-resorbing factors-induced osteoclast-like cell formation in mouse marrow culture in vitro. The stimulatory effect of bone-resorbing factors on osteoclastogenesis is mediated through receptor activator of NF-κB ligand (RANKL), which plays a key role in development of osteoclasts from preosteoclasts. Zinc inhibits RANKL stimulation in osteoclastogenesis. RANKL is expressed in osteoblasts. In addition, zinc may inhibit RANK expression which is mediated through Ca^{2+} signaling (protein kinase C) by stimulation of PTH, 1, 25$(OH)_2D_3$ or PGE_2 in osteoblasts. Zinc plays an inhibitory role in the regulation of osteoclastogenesis.

Key words: zinc, osteoclastogenesis, bone resorption, osteoporosis

To cite this article:

Masayoshi Yamaguchi, “Role of Zinc in Regulation of Osteoclastogenesis”, Biomedical Research on Trace Elements, Vol. **15**, pp.9-14 (2004) .

JOI JST.JSTAGE/brte/15.9