
Abstract
Zinc is essential for the growth of the human and other animals. Bone growth retardation is a common finding in various conditions associated with zinc deficiency, suggesting a physiological role of zinc in the growth and mineralization of bone tissue. Bone zinc content is decreased by development with aging, skeletal unloading, and postmenopausal conditions. Zinc deficiency may play a pathophysiological role in the deterioration of bone metabolism. Zinc has been demonstrated to have a stimulatory effect on bone formation and mineralization; the metal directly activates aminoacyl-tRNA synthetase in osteoblastic cells, and it stimulates cellular protein synthesis. Moreover, zinc inhibits osteoclastic bone resorption by inhibiting osteoclast-like cell formation from marrow cells. Zinc may act on the process of bone-resorbing factors-induced protein kinase C activation, which is involved in Ca\(^{2+}\) signaling in osteoclastic cells. Zinc plays a role in the preservation of bone mass. AHZ is a zinc compound, in which zinc is chelated to β-alanyl-L-histidine. The stimulatory effect of AHZ on bone formation was more intensive than that of zinc sulfate. It is confirmed that bone-forming effect of AHZ is a greater than that of various bone-regulating hormones and other factors. The oral administration of AHZ has a fine restorative effect on osteopenia with various pathophysiological conditions. Zinc compound may be a new drug in the therapy of osteoporosis.