Genomic approaches to bone and joint diseases. New insights into molecular mechanisms underlying protective effects of vitamin K on bone health

[Article in Japanese]

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Abstract

Vitamin K is a nutrient originally identified as an essential factor for blood coagulation. Accumulated evidence indicates that subclinical non-hemostatic vitamin K deficiency in extrahepatic tissues, particularly in bone, exists widely in the otherwise healthy adult population. Both vitamin K1 and K2 have been shown to exert protective effects against osteoporosis. The new biological functions of vitamin K in bone are considered to be attributable, at least in part, to promotion of gamma-carboxylation of glutamic acid residues in vitamin K-dependent proteins, which is shared by both vitamins K1 and K2. A recent evidence of significant correlation between polymorphism of gamma-glutamyl carboxylase gene and bone mineral density supports the role of gamma-carboxylation-dependent actions of vitamin K. In contrast, vitamin K2-specific,gamma-carboxylation-unrelated functions have recently attracted scientific attention. Recent findings of vitamin K2-specific transactivation of steroid and xenobiotic receptor (SXR/PXR) may lead to new research avenue. The impact of genotype of apoE, a major vitamin K transporter, on ostepporosis as well as Alzheimer disease and atherosclerosis, raises a question whether vitamin K is involved in the pathogenesis of these diseases. Molecular bases of coagulation-unrelated pleiotropic actions of vitamin K and its implications in bone health deserve further investigations.

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