Vitamin K stimulates osteoblastogenesis and inhibits osteoclastogenesis in human bone marrow cell culture.
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Abstract
Accumulating evidence indicates that menaquinone-4 (MK-4), a vitamin K(2) with four isoprene units, inhibits osteoclastogenesis in murine bone marrow culture, but the reason for this inhibition is not yet clear, especially in human bone marrow culture. To clarify the inhibitory mechanism, we investigated the differentiation of colony-forming-unit fibroblasts (CFU-Fs) and osteoclasts in human bone marrow culture, to learn whether the enhancement of the differentiation of CFU-Fs from progenitor cells might relate to inhibition of osteoclast formation. Human bone marrow cells were grown in alpha-minimal essential medium with horse serum in the presence of MK-4 until adherent cells formed colonies (CFU-Fs). Colonies that stained positive for alkaline phosphatase activity (CFU-F/ALP(+)) were considered to have osteogenic potential. MK-4 stimulated the number of CFU-F/ALP(+) colonies in the presence or absence of dexamethasone. The stimulation was also seen in vitamin K(1) treatment. These cells had the ability to mineralize in the presence of alpha-glycerophosphate. In contrast, both MK-4 and vitamin K(1) inhibited 1,25 dihydroxyvitamin D(3)-induced osteoclast formation and increased stromal cell formation in human bone marrow culture. These stromal cells expressed ALP and Cbfa1. Moreover, both types of vitamin K treatment decreased the expression of receptor activator of nuclear factor kappaB ligand/osteoclast differentiation factor (RANKL/ODF) and enhanced the expression of osteoprotegerin/osteoclast inhibitory factor (OPG/OCIF) in the stromal cells. The effective concentrations were 1.0 microM and 10 microM for the expression of RANKL/ODF and OPG/OCIF respectively. Vitamin K might stimulate osteoblastogenesis in bone marrow cells, regulating osteoclastogenesis through the expression of RANKL/ODF more than through that of OPG/OCIF.
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