Effects of fluoride during osteoblasts mineralization

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Fluoride is a trace element that can be incorporated during bone formation and substitutes hydroxyapatite by fluorapatite. Effects of fluoride ions in biological systems can promote advantages and problems. On one hand, fluoride could be a mitogenic stimulus for osteoblasts. However, high concentrations of this element can cause apoptosis in rat and mouse osteoblasts, and inhibition of the osteogenic function. There are correlations with fluoride and expression of some proteins/factors involved in cellular functions (osteoblast, odontoblast, and ameloblast): collagen, matrix metalloproteinases (MMPs), core-binding factor α1 (Cbfa1), osteoprotegerin (OPG), alkaline phosphatase (ALP), and others. Furthermore there are relations between fluoride and mineralization processes, where fluoride can modulate this process negatively or positively. The most common fluoride effects related to bone cells are: induction of apoptosis, induction of oxidative damage/stress, and regulation of the mitogen-activated protein kinase (MAPK). In addition, fluoride can promote osteoblast differentiation and proliferation through activation of Bone Morphogenetic Protein (BMP)/Smad pathway. However, in general, there are some controversial points that remain unclear, like fluoride concentration and effect, influence of the genetic background, molecules modulated and susceptibility cellular to fluoride. In conclusion, fluoride affects the production and degradation of the extracellular matrix during early onset and probably during the mineralization period. Additionally, the genetic factors may contribute to the variation in cell response to fluoride exposure.