



Aluminum inhibits the growth of hydroxyapatite crystals developed on a biomimetic methacrylic polymer.

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Résumé en anglais

PROJECT: Aluminum (Al) is an increasing problem in biomedicine since it can interact with phosphates. Bone is one of the preferential target tissues of Al deposition: Al interacts with mineralization and/or bone cell activities. We searched the influence of Al deposition in hydroxyapatite developed on a biomimetic polymer (carboxymethylated poly(2-hydroxyethyl-methacrylate)) which mimics bone mineralization in the absence of cells.

PROCEDURES: Pellets of polymer were incubated for 5 days in a synthetic body fluid (SBF) to induce mineralization, then 21 days in SBF containing 20, 40 and 60 µg/L Al(3+). Other pellets were incubated in SBF containing commercial Al foil (33 mg/vial) either in 1, 2 or 6 pieces. The mineral deposits were dissolved in HCl and Ca(2+), PO(4)(3-) and Al(3+) content was measured. Hydroxyapatite was characterized by SEM and X energy-dispersive X-ray analysis (EDX).

RESULTS: The amount of Al(3+) was dose-dependently increased in Ca/P deposits on the polymer pellets. At high concentration (or with the 6 Al foils) growth of hydroxyapatite calcospherite was inhibited; only calcified plates emerging from the polymer were observed. Pellets incubated with 1 and 2 Al foils exhibited a reduction in calcospherite diameter and an increase in the Al(3+)/Ca(2+) ratio. EDX identified Al in the mineral deposits.

CONCLUSIONS: In this acellular model, Al(3+) altered the growth of calcospherites at low concentration and inhibited their development at high concentration. In SBF, a release of Al(3+) from aluminum foils also inhibited mineralization. This study emphasizes the importance of Al in bone pathology and stresses the question of its release from biomaterials.

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