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ACP-Mg particles for treatment of dental hypersensitivity: a mode of action study

Camilla Berg, Erik Unosson, Håkan Engqvist, Wei Xia

Introduction/purpose:
Dental hypersensitivity is a common clinical condition usually associated with exposed dentinal tubules. Occlusion of those, hindering fluid movement as a response to external stimuli, is one approach to reduce pain.[1] The occluding agent should induce mineralization and have a good filling effect. In this study, particles (200-400 nm) of Amorphous Calcium Phosphate with Mg (ACP-Mg) were used aiming to evaluate the mode of action with in-vitro studies on dentine as well as degradation properties in buffered systems.

Methods:
Degradation properties and ion-release was monitored by dispersing the particles in Tris-HCl (10 mg/mL) storing the dispersions at 37 °C. In-vitro testing was performed on 1 mm thick dentine discs cut from extracted human molars. Specimens were etched in 35 % phosphoric acid, rinsed with DI-water followed by treatment by applying a gel formulation containing the particles using a soft bristled toothbrush, brushing 1 min on each side and leaving the specimen to rest for 3 min. Treatment was repeated four times during one day and samples were incubated at 37 °C in artificial saliva.

Results:
In vitro tests and degradation studies showed that ACP-Mg particles induced formation of Hydroxyapatite (HA). ICP-OES, XRD and SEM showed that there was a rapid release of all ions up to 6 hours followed by a re-precipitation of HA at 24 hours with a lower Mg-content. The crystallinity increased with time as the concentration of all ions decreased in the solutions. Morphological evaluation for the in-vitro tests showed that HA-like structures formed already after 24 hours on the dentine surface. Cross-sections revealed that the particles reached as far as 80 µm from the surface and tubules, fully occluded by HA-like structures, was visible at comparable depths after 7 days.

Conclusions:
ACP-Mg particles can be used to reduce dentine hypersensitivity by effective occlusion of dentine tubules via rapid formation of surface and intra-tubular HA.

References: