

**BONE APATITE: BUILDING INTERFACES FROM THE INSIDE OUT.** Jill D. Pasteris, Brigitte Wopenka, and John J. Freeman, Department of Earth and Planetary Sciences, Washington University, Campus Box 1169, St. Louis, MO 63130-4899 USA. pasteris@levee.wustl.edu

**Introduction:** Bone provides a fascinating challenge to both materials scientists and mineralogists who study nanocrystals and mineral interfaces. Bone is a composite material dominated by nanocrystals of carbonated apatite (bioapatite) 10s of nm long, which are interfaced with orderly arrays of the fibrous protein, type I collagen. Bundles of mineralized collagen fibrils (100s of nm long) are organized into a plywood-like superstructure [1], which contributes to bone's remarkable combination of strength and flexibility. Despite the importance of bone apatite to human health, many questions remain concerning its chemistry and crystallography, which are, however, essential in determining the nature and strength of bonding between the mineral and collagen; the nano-scale properties of this interface, in turn, probably are critical to the bone's strength.

Among the ongoing controversies about the crystal-chemistry of bone apatite is the question of whether its several wt.% carbonate substitute for  $(\text{PO}_4)^{3-}$  groups, reside in the "OH-channels", or occupy both sites [2-4]. Moreover, despite decades-long reference in the medical literature to bone apatite as "hydroxyapatite" or "carbonated hydroxyapatite", various analytical techniques, including NMR, IR, and Raman spectroscopy show no evidence of OH<sup>-</sup> in bone apatite [5]. In addition to the crystallographic ramifications of those possible ionic substitutions, they also entail different charge-balance needs, which strongly affect the bonding between mineral and collagen.

**Chemically Imposed Nanocrystallinity:** Bioapatite crystals are plate-like; they have in-plate dimensions on the order of 10s of nm but thicknesses of only a few nm. Such nano-scale dimensions result in high surface area:volume ratios, which promote a rapid response of the crystal to changes in its chemical environment (from adjacent fluid or collagen) and also ensure that changes in mineral chemistry have a strong effect on the crystal-collagen bonding.

In many ways, bone exhibits the "novel" properties recognized in nanocrystalline materials compared to their coarse-grained equivalents, *e.g.*, significantly enhanced strength, ductility, wear-resistance, and chemical reactivity. These properties typically are ascribed to the high surface area:volume ratio of nanocrystals, *i.e.*, physical conditions can be imposed to create nano-scale grains whose bulk properties are enhanced with respect to those of their macro-scale analogs. In contrast, in the case of bone, we believe that nanocrystallinity is imposed chemically through the body's control on the composi-

tion of bioapatite--which dictates not only a small grain size, but also an apparently desirable defect structure (two different aspects of "crystallinity").

Under low-temperature (*e.g.*, body-temperature) conditions, various ion substitutions are known to inhibit the growth of apatite crystallites. There is strong evidence that the body takes full advantage of its ability to control the grain size of bioapatite through its composition, particularly the carbonate content. Bone, which--in order to be healthy--must undergo frequent resorption and reprecipitation, has a higher carbonate concentration and smaller grain size (by a factor of ~10X) than does tooth enamel, which should not resorb. We infer that biochemical feedback allows the body to use carbonate and available trace ions (*e.g.*,  $\text{Mg}^{2+}$  and  $\text{Na}^+$ ) to optimize not only the size, but also the defect structure of the bioapatite nanocrystals, both of which control the strength of the mineral-collagen interface.

Supporting evidence for the above hypothesis comes from our Raman microprobe spectroscopic comparison of untreated bone with bone that had undergone *in vitro* fluoridation. Our Raman data showed that the fluoridated bioapatite's "degree of crystallinity" had increased significantly, whereas the bone's mechanical strength had decreased [6]. A likely explanation for the latter is that the bioapatite-collagen interface was compromised by fluoridation either through an increase in mineral grain size (thus, decreased surface area) or a reduction in the mineral's defect density (thus, perhaps, decreased interfacial bond strength).

#### References:

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