Geophysical Research Abstracts
Vol. 16, EGU2014-16113, 2014
EGU General Assembly 2014

Apatite mineralization in elasmobranch skeletons via a polyphosphate intermediate

Sidney Omelon (1), Nicolas Lacroix (1), Levannia Lildhar (1), Fabio Variola (1), and Mason Dean (2)

(1) Department of Chemical and Biological Engineering, University of Ottawa, Ottawa, Canada (somelon@uottawa.ca), (2) Department of Biomaterials, Max Planck Institute of Colloids and Intefraces, Potsdam-Golm, Germany (mason.dean@mpikg.mpg.de)

All vertebrate skeletons are stiffened with apatite, a calcium phosphate mineral. Control of apatite mineralization is essential to the growth and repair of the biology of these skeletons, ensuring that apatite is deposited in the correct tissue location at the desired time. The mechanism of this biochemical control remains debated, but must involve increasing the localized apatite saturation state.

It was theorized in 1923 that alkaline phosphatase (ALP) activity provides this control mechanism by increasing the inorganic phosphate (Pi) concentration via dephosphorylation of phosphorylated molecules. The ALP substrate for biological apatite is not known. We propose that polyphosphates (polyPs) produced by mitochondria may be the substrate for biological apatite formation by ALP activity. PolyPs $(PO_3^-)_n$, also known as condensed phosphates, represent a concentrated, bioavailable Pi-storage strategy. Mitochondria import Pi and synthesize phosphate polymers through an unknown biochemical mechanism. When chelated with calcium and/or other cations, the effective P-concentration of these neutrally charged, amorphous, polyP species can be very high ($\sim$ 0.5 M), without inducing phosphate mineral crystallization. This P-concentration in the low Pi-concentration biological environment offers a method of concentrating P well above an apatite supersaturation required for nucleation.

Bone is the most studied mineralized skeletal tissue. However, locating and analyzing active mineralizing areas is challenging. We studied calcified cartilage skeletons of elasmobranch fishes (sharks, stingrays and relatives) to analyse the phosphate chemistry in this continually mineralizing skeleton. Although the majority of the elasmobranch skeleton is unmineralized cartilage, it is wrapped in an outer layer of mineralized tissue comprised of small tiles called tesserae. These calcified tesserae continually grow through the formation of new mineral on their borders.

Co-localization of ALP and polyPs were identified at the mineralizing tessera borders using Raman spectroscopy, fluorescence microscopy and histological methods. Application of exogenous ALP to skeletal tissue cross-sections resulted in polyP disappearance, and Pi production. It is proposed that elasmobranch skeletal cells produce polyP-containing granules as a concentrated P-source, while ALP activity controls when and where Pi is cleaved from polyP, increasing the apatite supersaturation to nucleate apatite minerals in the skeleton. These data support not only interaction of polyP and ALP as a cell-mediated apatite mineralization control mechanism, but also suggest that this mechanism arose millions of years ago and is common to both bony and cartilaginous skeletal systems.