EDITORIAL

## New Insights into the Mechanisms of Biomineralization

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The ubiquitous process of biological mineralization has preoccupied scientists for centuries.

In 1849 Quekett [1] wrote a paper entitled "On the Intimate Structure of Bone, as Composing the Skeleton, in the Four Great Classes of Animals, viz., Mammals, Birds, Reptiles and Fishes. . . ." The vertebrate skeletal mineral was identified in 1894 by Levy [2] to be a calcium phosphate carbonate. In 1926, de Jong [3] in his paper "La substance minerale dans les os" identified the structure of bone mineral as a poorly crystalline carbonated apatite mineral.

In this issue of *Calcified Tissue International*, we highlight some of the controversies and challenges about the mechanisms of biological mineralization. This issue will focus mainly on the mammalian system of physiological and pathological mineralization, where the mineral deposited is an apatitic structure.

As bone and teeth are composites of an organic matrix in which a mineral phase is deposited, it is the interplay between these two components that is investigated by Veis and Dorvee, who propose a new paradigm for crystal nucleation in organic matrices. De Yoreo and colleagues focus their review on the dynamics and energetics of matrix assembly and mineralization. Landis and Jacquet show how the collagen polypeptide stereochemistry underlies the binding of calcium and phosphate ions at sites within collagen holes and overlap regions and leads to nucleation of apatite crystals. Using a different approach

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In order to understand how disruption in the collagen assembly can affect bone mineralization in diseases such as osteogenesis imperfecta, Eyre and Weis review recent discoveries showing the potential for a common pathogenic mechanism of misassembled collagen fibrils leading to abnormal bone mineralization. Brylka and Jahnen-Dechent investigate how fetuin-A, a systemic protein inhibitor of mineralization, is important in the stabilization and clearance of the amorphous mineral precursor phase and thus prevents the local buildup of mineral in soft tissue. Some noncollagenous proteins such as osteopontin are present at high levels both in bone and in pathological calcification. Hunter in his review asks whether osteopontin is a promoter or an inhibitor of biological apatite formation. It is well known that many soft tissues in our body can calcify under pathological conditions. To understand how pathological calcification occurs in blood vessels, Giachelli and colleagues investigate the mechanisms of vascular calcification and its potential treatment. Another form of pathological calcification is the formation of stones in extracellular fluids; Krieger and Bushinsky investigate the relation between bone and kidney stones.

Finally, Omelon and colleagues review the role of polyphosphates in the inhibition of calcium and phosphate precipitation as well as their role as a source of calcium and phosphate to promote mineralization in both bacteria and mammals.

We hope that this issue of *Calcified Tissue International* will help readers understand the controversies in this field and spur further research toward fully understanding the mechanisms leading to this most important biological phenomenon.

Marc Grynpas, Ph.D. Guest Editor

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