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Mineralogy and chemistry of osteoporosis – mechanism of mineralization (calcification) of human tissues

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Human bone mineralization studies were conducted using classic mineralogical techniques (SEM, XRD, IR, PLM, EDX, and AAS). Samples were taken from human vertebrae, healing broken bones, stages of bone elongation, bone tumors, and demineralization of osteoporetic bones. These investigations were performed in parallel with examination of mineralized (calcified) bone cartilage, blood vessels, coronary vessels, and heart valves and muscle.

Results indicate that dissolution of bone hydroxylapatite [$Ca_5(PO_4)_3(OH)$] due to ageing of an organism releases mainly Ca^{2+} and PO_4^{3+}, weakening the bone structure (osteoporosis). These elements are deposited in various tissues causing their mineralization (calcification).

Calcification starts and develops at centres of crystallization present in tissues; centres are initiated by fragments of tissues, usually at the atomic level. Destruction may be of autogenic or allogenic origin. Autogenic centres of crystallization are genetically induced. Allogenic centres of calcification form mechanically at loci of high physical activity of tissues (mechanical destruction of tissues) or irritation by stable mineral phases (such as, asbestos or quartz dust particles) introduced into the organism. Centres of crystallization may be formed chemically, created by aggressive external pollutants, including various micropollutants or acids. Moreover, centres may be induced by toxic metabolites of microorganisms (bacteria, viruses and others) that are active during infectious disease stages.

The dissolution and removal of elements from bones and subsequent recrystallization in various human tissues may cause serious diseases, e.g., hypertension from crystallization of Ca and P phases in blood vessels or coronary vessels, leading to heart failure.

Scientific interactions of physicians, biochemists, and mineralogists could lead to significant understanding of the biology and prevention of calcification-induced disease.

References

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[2] Skinner H.C.W. (2003) op.cit.