

MATHEMATICAL MODELING OF MINERAL AND BONE METABOLISM IN PATIENTS
WITH KIDNEY FAILURE

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According to the International Osteoporosis Foundation (IOF, Annual Report 2016) 1 in 2 women and 1 in 5 men aged 50+ will have an osteoporotic fracture. Every year there are approximately 9 mln such fractures worldwide. Fractures are the cause of long-term disability of individuals and have negative socio-economic consequences. The risk of bone fractures in patients with renal failure is twice higher than in general population of comparable age. Kidney disease leads to abnormalities in mineral and bone metabolism: 1) biochemical disorders (increase of phosphate and parathyroid hormone in serum, vitamin D deficiency, decrease in calcium concentration, etc.), 2) abnormalities in mineralization, growth and bone strength, and 3) calcification of blood vessels and other soft tissues. Understanding the interaction between kidneys and bones may contribute to the insight into complex and interrelated mechanisms that occur between these organs. The aim of the proposed PhD thesis is to develop a mathematical model describing the mineral and bone metabolism in conditions of homeostasis, osteoporosis and in patients with renal failure in whom mineral and bone metabolism is impaired due to renal dysfunction. The model should take into account organs as bones, kidneys, parathyroid glands and intestines and describe their 'crosstalk'. The flow of calcium and phosphate will be regulated by parathyroid hormone, vitamin D and other regulatory mechanisms. The model should predict short- and long-term changes in bones. Computer codes should be developed to simulate in silico clinical trials. The project will be carried out in the international cooperation with the Karolinska Institute in Stockholm. The development and use of the planned mathematical model will contribute to a better understanding of regulatory mechanisms related to mineral metabolism and its disorders caused by kidney disease.