Changes in osteocyte lacunar properties are due to new bone formation in hypoparathyroidism patients treated by recombinant parathyroid hormone

Maja Østergaard,[1] Tanja T. Sikjær,[2] Samera Siddiqui,[1] Morten B. Nielsen,[1] Lars Rejnmark,[2,3] and Henrik Birkedal[1]

[1]iNANO and Department of Chemistry, Aarhus University, Denmark, [2]Department of Endocrinology and Internal Medicine, Aarhus University Hospital, Denmark, [3]Institute of Clinical Medicine, Aarhus University, Denmark

Background: Bone is formed and regulated under a great amount of biological control. Disorders in this control can have serious consequences for bone health. Patients suffering from hypoparathyroidism (hypoPT) have reduced parathyroid gland capacity and hence impaired ability to make parathyroid hormone (PTH). PTH regulates the calcium and phosphate levels in the blood, thus it is essential for maintaining bone. Patients lacking the hormone therefore suffer from low blood calcium and a very low bone turnover, causing a lack of bone renewal.[1,2] By investigating the effect of this lack of control on the biomineralization process, we can gain insight into the normal-case mineralization mechanism. Recent studies have shown promise for treating with recombinant human parathyroid hormone (rhPTH), since this treatment leads to renewed bone formation.[1-6]

Aim: To investigate the rhPTH induced mineralization process in hypoPT patients on the micro- and nanoscale.

Methods: Iliac crest biopsies from hypoPT patients treated with rhPTH or placebo for 6 and/or 30 months were measured with high resolution synchrotron radiation micro computed tomography (SR-μCT) at the TOMCAT beamline at the Swiss Light Source (SLS) with an isotropic voxel size of 0.325 μm. From this, bone volume (BV) ratios, grey level variations, and lacunar morphometric parameters were assessed with *N*-way ANOVA tests.

Results: Lower grey levels are observed in patients treated with rhPTH corresponding to the expected promotion of bone renewal, which happens more quickly in trabecular bone, as the grey levels there are lower. In addition, it is noted that longer-term treatment promotes the growth of more new bone. The lacunae show no signs of treatment-induced alterations. However, it is seen that lacunae are larger, less stretched, and more abundant in the newly formed bone, indicating that bone cells are actively laying down new bone. This suggests a typical mineralization process despite the remodeling being brought on by the treatment.

Conclusion: Statistical tests showed that at the scale probed in this study, treatment with rhPTH induces bone formation in patients suffering from hypoPT. Most likely, normal bone mineralization is induced with the treatment, as the lacunae do not change in the bones treated with rhPTH.

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