



Oral Abstract

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Anabolic effect of PTH in young rats is attenuated by calcitonin

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Parathyroid hormone (PTH), at continuously high levels is a catabolic agent in bone, yet when administered intermittently, PTH has an anabolic effect, and is used for treating osteoporosis. To investigate whether active osteoclasts contribute to the anabolic effect of PTH, rats were treated with anabolic doses of PTH (3µg/kg/day or 30µg/kg/day hPTH(1-34) for three weeks) in the presence and absence of salmon calcitonin (sCT), an acute blocker of osteoclast activation. Treatment with hPTH(1-34) at 3µg/kg/day significantly increasing femoral trabecular BMD (Tb.BMD) by 32%. This was prevented when sCT was administered concurrently with PTH. In a separate experiment, treatment with hPTH(1-34) at 30µg/kg/day elevated Tb.BMD by 108%, and raised tibial trabecular bone volume (BV/TV) by 255%. When sCT was administered concurrently with hPTH(1-34) this anabolic effect was significantly attenuated; there was no significant elevation in Tb.BMD, and the anabolic effect of PTH on BV/TV was halved. Administration of sCT alone had no effect on Tb.BMD or BV/TV. Administration of sCT 5 hours after PTH, or 1 hour before PTH administration did not block the anabolic effect of PTH on Tb.BMD, indicating that active osteoclasts are required within one hour of PTH administration for the full anabolic effect of PTH.

In conclusion, this data suggest that the anabolic effect of PTH is dependent on acute activation of osteoclasts and this can be blocked by co-administration of CT. Thus in young female rats osteoclast-osteoblast communication is critical for PTH anabolic effects on bone.