TERIPARATIDE – EFFECTS ON MICROSTRUCTURE OF BONE

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Anabolic drugs are aimed to promote the formation of new bone to restore bone structure that has previously been lost. PTH promotes differentiation of committed osteoblast precursors in the bone marrow and possibly also in lining cells, and increases the lifespan of mature osteoblasts and osteocytes by preventing apoptosis. PTH rapidly reduces sclerostin mRNA and protein production by osteoblasts. A transient reduction of sclerostin production by osteocytes in response to intermittent PTH could reduce osteoblast apoptosis. The anabolic effectiveness of PTH requires that it be administered intermittently to achieve a rapid increase in PTH and a rapid decline to preexisting levels. This intermittent mode results in activation of genes responding specifically to a transiently activated signaling system. PTH deposits new bone, increasing thickness of the trabeculae and

cortex. Most of the increase in cortical and trabecular thickness induced by PTH is due to modeling and remodeling on the endocortical surface of the cortex and on either side of the trabeculae. The effect of teriparatide on bone microarchitecture was investigated in sixty-six postmenopausal women with osteoporosis (mean age of 68.0 years and mean BMD T-score of -1.7 at total hip and -2.8 at lumbar spine; 62% with prevalent fractures) has been treated