A closer look at osteoporosis medication's mechanisms may improve outcomes

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Osteoporosis is the primary cause of bone fractures in the elderly. Bone loss in this disease reflects an imbalance between the activity of bone-degrading cells called osteoclasts and bone-building cells called osteoblasts. Teriparatide, a recombinant form of parathyroid hormone, is currently the only FDA-approved treatment for osteoporosis that targets bone formation. This medication stimulates osteoblast activity and increases the lifespan of osteoblasts, but whether it also enhances the production of these bone-forming cells is unknown.

New findings from Henry Kronenberg's lab at Massachusetts General Hospital and Harvard Medical School suggest that while teriperatide treatment does increase the proliferation of osteoblasts, stopping treatment may have adverse effects. A study published this week in the *JCI* reports that the number of osteoblast precursor cells and their mature descendants increased in mice undergoing teriparatide treatment. When mice were withdrawn from teriparatide treatment, not only did osteoblast proliferation diminish, but the number of newly-formed fat cells, or adipocytes, increased.

These findings suggest that therapies targeting the parathyroid hormone receptor may enhance osteoblast populations by prioritizing osteoblast development over other cell fates, including adipocytes. The findings also offer insights into possible drawbacks of teriparatide therapy, indicating that the long-term consequences of brief treatment periods could outweigh the benefits.

More information: Deepak H. Balani et al, Parathyroid hormone regulates fates of murine osteoblast precursors in vivo, *Journal of Clinical Investigation* (2017). DOI: 10.1172/JCI91699