Impact of parathyroid hormone on osteoporosis; a short look at the current knowledge

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Abstract
Osteoporosis is a disease characterized by losing bone tissue, resulting in increased bone fragility and an increased risk of fractures. Parathyroid hormone (PTH) plays an essential role in the development of osteoporosis. High levels of PTH for prolonged periods lead to a decrease in bone density. However, intermittent administration of PTH is an effective treatment for osteoporosis, suggesting that careful dosing of PTH can have a therapeutic benefit in the treatment of osteoporosis.

Keywords: Osteoporosis, Bone density, PTH, Calcium, Parathyroid hormone, osteoblasts, Parathyroid hormone, Parathormone

Introduction
Calcium metabolism is regulated by the parathyroid hormone (PTH, parathormone), secreted by the parathyroid glands. PTH increases blood calcium levels by increasing calcium release from bone and calcium reabsorption in the kidneys (1,2). However, when the level of parathormone remains high for a prolonged period, it can lead to osteoporosis. Osteoporosis is characterized by low bone density and an increased risk of fractures. In this condition, bones become weak and brittle, making them more prone to fractures (3,4). Parathormone plays a dual role in the development of osteoporosis. In the short term, parathormone stimulates bone formation by promoting the activity of osteoblasts, bone-forming cells, leading to increased bone density. However, high levels of parathormone induce bone resorption for a long time, as it stimulates osteoclasts and bone-removing cells and decreases bone density (5,6). Intermittent administration of parathormone, called teriparatide, is an effective treatment for osteoporosis (5,7).

Effect of PTH on bone function and structure
Studies have shown that PTH can stimulate bone formation at low doses, but at higher doses, it can activate bone resorption and lead to net bone loss (6). This is due to the dual effects of parathormone on osteoblasts and osteoclasts, the two main types of bone cells responsible for bone formation and resorption, respectively (6,8). Moreover, intermittent administration of parathormone, also known as teriparatide, can stimulate bone formation and increase bone mineral density in patients with osteoporosis. This is due to the anabolic effects of parathormone, meaning it promotes bone formation. However, extended exposure to high parathormone levels can lead to bone resorption, worsening osteoporosis (9).

In addition to its direct effects on bone cells, parathormone can indirectly affect bone metabolism by regulating the production of other hormones, such as vitamin D and estrogen. Vitamin D is essential for calcium absorption and bone mineralization, while estrogen has a protective effect on bone density in women (10,11).

It is essential to closely monitor the use of parathormone for therapy to minimize its benefits and any adverse effects. Recent research has also suggested a potential link between changes in parathormone levels and increased fracture risk in older adults with osteoporosis. This highlights the importance of monitoring parathormone levels as a possible indicator of bone health and fracture risk (12,13). The PTH is a crucial regulator of calcium and phosphate homeostasis in the body. The parathyroid
gland produces it and increases blood calcium levels by stimulating calcium release from bones, increasing calcium absorption from the gut, and decreasing calcium excretion by the kidneys. PTH has been studied as a potential therapeutic target in patients with osteoporosis due to its ability to stimulate bone formation (2,14). Daily injections of synthetic PTH, also known as teriparatide, have been shown to increase bone mineral density and reduce fracture risk in postmenopausal women with osteoporosis. The anabolic effects of PTH on bone are due to its ability to stimulate the activity of osteoblasts, cells responsible for bone formation (7,15). This results in increased collagen production, mineralization of bone tissue, and overall bone strength. However, prolonged exposure to high levels of PTH can lead to bone resorption, which is the breakdown of bone tissue. This can worsen osteoporosis over time and increase the risk of fractures.

High levels of PTH have also been linked to other health issues, such as kidney stones and cardiovascular disease (16,17). Therefore, using PTH as a therapeutic target for osteoporosis must be carefully monitored to ensure optimal dosage and duration of exposure. Ongoing research is needed to understand further the complex mechanisms involved and to develop more effective and safer treatments for osteoporosis (18).

**Treatment of osteoporosis**
Current treatment options for osteoporosis include bisphosphonates, hormone replacement therapy, and PTH analogs. PTH analogs such as teriparatide have increased bone density and reduced fracture risk in patients with severe osteoporosis. However, long-term use of PTH analogs may increase the risk of bone cancer and should be carefully monitored (7,19).

**Conclusion**
Parathormone plays a complex role in regulating bone metabolism, and its impact on osteoporosis depends on the dose and duration of exposure. Further research is needed to understand the mechanisms underlying PTH-mediated bone loss better and to develop safer and more effective treatments for osteoporosis.

**Conflicts of interest**
The author declares that he has no competing interests.

**Ethical issues**
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**References**