The role of parathyroid hormone in bone health and metabolism.

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Introduction

Parathyroid Hormone (PTH), a crucial regulator of calcium and phosphate balance, plays a fundamental role in bone health and metabolism. Secreted by the parathyroid glands, which are small endocrine glands located behind the thyroid gland in the neck, PTH influences several physiological processes that impact bone density, strength, and overall skeletal health. Understanding how PTH operates can shed light on its critical role in maintaining bone health and its implications for various metabolic disorders. The primary function of PTH is to regulate serum calcium levels, which are essential for numerous bodily functions, including muscle contraction, nerve signaling, and blood clotting. When blood calcium levels drop below a certain threshold, the parathyroid glands secrete PTH into the bloodstream. This hormone then acts on several target tissues to restore calcium levels to their normal range [1, 2].

One of the key actions of PTH is its effect on bone. PTH influences bone health through a process known as bone remodeling, which is the continuous cycle of bone resorption and formation. This process involves the coordinated activity of two types of bone cells: osteoclasts, which break down bone tissue, and osteoblasts, which build new bone tissue. PTH primarily stimulates osteoclast activity, leading to increased bone resorption. This action releases calcium from the bone matrix into the bloodstream, raising serum calcium levels. Although this might initially seem detrimental, it is important for maintaining calcium homeostasis, especially in the face of fluctuating calcium intake or changes in calcium demand [3, 4].

In addition to stimulating osteoclast activity, PTH indirectly supports bone formation through its effects on osteoblasts. PTH stimulates osteoblasts to produce new bone matrix, although this effect is more complex and dependent on the pattern and duration of PTH exposure. Intermittent, physiological levels of PTH can promote bone formation and increase bone density, whereas continuous high levels of PTH, as seen in some pathological conditions, primarily lead to bone resorption and loss. The relationship between PTH and bone health is particularly evident in conditions such as osteoporosis and hyperparathyroidism. Osteoporosis, a condition characterized by reduced bone density and increased fracture risk, can result from an imbalance between bone resorption and formation. Elevated levels of PTH, often due to primary hyperparathyroidism, can exacerbate bone loss by increasing osteoclast activity and bone resorption. Managing osteoporosis often involves treatments that counteract excessive PTH activity, such as bisphosphonates or calcimimetics, which help to reduce bone resorption and promote bone health [5, 6].

On the other hand, the therapeutic use of PTH in certain forms of osteoporosis illustrates its potential benefits. Intermittent administration of recombinant PTH (teriparatide) can stimulate bone formation and increase bone density. This treatment approach harnesses the bone-forming potential of PTH when given in controlled, intermittent doses, offering a valuable option for individuals with severe osteoporosis who have not responded adequately to other treatments. PTH's role in calcium metabolism extends beyond bone health. It also influences renal function, contributing to calcium homeostasis by affecting the kidneys' ability to excrete or reabsorb calcium. PTH promotes the reabsorption of calcium in the renal tubules, reducing calcium loss in the urine. Additionally, PTH stimulates the conversion of inactive vitamin D to its active form, calcitriol, in the kidneys. Calcitriol enhances intestinal absorption of calcium and phosphate, further supporting the maintenance of adequate serum calcium levels [7, 8].

The interplay between PTH, calcium, and vitamin D is vital for overall metabolic health. Insufficient vitamin D levels can impair calcium absorption and lead to secondary hyperparathyroidism, where the parathyroid glands increase PTH production to compensate for low calcium levels. This condition can result in increased bone resorption and, over time, contribute to bone loss and other health issues. In contrast, conditions that lead to excessively high levels of PTH, such as primary hyperparathyroidism, can have significant effects on bone health. This condition, often caused by a benign tumor of the parathyroid gland, leads to elevated PTH levels and excessive bone resorption. Over time, this can result in weakened bones, increased fracture risk, and symptoms such as bone pain and kidney stones. Treatment for primary hyperparathyroidism typically involves surgical removal of the affected parathyroid tissue to normalize PTH levels and alleviate associated symptoms [9, 10].

Conclusion

In summary, parathyroid hormone plays a critical role in bone health and metabolism by regulating calcium levels through its effects on bone resorption, renal function, and vitamin D

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metabolism. Its actions are essential for maintaining calcium homeostasis and supporting various physiological functions. However, imbalances in PTH levels can lead to significant health issues, including osteoporosis and hyperparathyroidism. A comprehensive understanding of PTH's functions and its implications for bone health is essential for diagnosing and managing conditions associated with abnormal PTH levels, ultimately contributing to better overall health and well-being.

References

1. Makey IA, Geldmaker LE, Casler JD, et al. Localization and surgical approach to mediastinal parathyroid glands. J Cardiothorac Surg. 2022;17(1):299.
2. Kim Y, Kim SW, Lee KD, et al. Phase-sensitive fluorescence detector for parathyroid glands during thyroidectomy: a preliminary report. J Biophotonics. 2020;13(2):e201960078.
3. Chen T, Wang Y, Hao Z, et al. Parathyroid hormone and its related peptides in bone metabolism. Biochem Pharmacol. 2021;192:114669.
4. Goltzman D, Mannstadt M, Marcocci C. Physiology of the calcium-parathyroid hormone-vitamin D axis. Front Horm Res. 2018;50:1-3.
5. Kužma M, Jackuliak P, Killinger Z, et al. Parathyroid hormone-related changes of bone structure. Physiol Res. 2021;70(1):S3.
6. Bollerslev J, Pretorius M, Heck A. Parathyroid hormone independent hypercalcemia in adults. Best Pract Res Clin Endocrinol Metab. 2018;32(5):621-38.
7. Leder BZ. Parathyroid hormone and parathyroid hormone-related protein analogs in osteoporosis therapy. Current osteoporosis reports. 2017;15:110-9.
8. Zhang L, Liu N, Shao J, et al. Bidirectional control of parathyroid hormone and bone mass by subfornical organ. Neuron. 2023;111(12):1914-32.
9. Wojda SJ, Donahue SW. Parathyroid hormone for bone regeneration. J Orthop Res 2018;36(10):2586-94.
10. Alexander RT, Dimke H. Effects of parathyroid hormone on renal tubular calcium and phosphate handling. Acta Physiol. 2023;238(1):e13959.

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