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Mini Review

# Osteoporosis Caused by Drugs and Hormones: An Overview of the Molecules and Cells

**Kuo Chen***

*Department of Medicine and Gerontological, Mackay Memorial University, Taipei, Taiwan*

## ABSTRACT

Osteoporosis coming about because of a lopsidedness of bone turnover among resorption and development is a basic medical problem around the world. For postmenopausal women, hormone-related osteoporosis is most commonly caused by estrogenic deficiency, while glucocorticoid-induced osteoporosis remains the most common form of drug-induced osteoporosis. Proton pump inhibitors, hypogonadism, selective serotonin receptor inhibitors, chemotherapies, and medroxyprogesterone acetate are among the other medical conditions and medications associated with secondary osteoporosis. The cellular and molecular mechanisms of bone turnover, the pathophysiology of osteoporosis, and their treatment are summarized in this review. Osteoclast genesis seems to be boosted by the nuclear factor-ligand uncoupling factor. Osteoprotegerin on the other hand, is an antagonist that osteoblast lineage cells secrete. After suppressing and triggering the release of estrogenic promotes apoptosis of osteoclasts and inhibits osteoclast genesis by stimulating the production of OPG and reducing osteoclast differentiation.

**Keywords**: Health; Nursing labour force; Gerontological nursing

## INTRODUCTION

Lack of estrogen causes the separation of bone resorption and formaOsteoporosis coming about because of a lopsidedness of bone turnover among resorption and development is a basic medical problem around the world. For postmenopausal women, hormone-related osteoporosis is most commonly caused by estrogen deficiency, while glucocorticoid-induced osteoporosis remains the most common form of drug-induced osteoporosis. Proton pump inhibitors, hypogonadism, selective serotonin receptor inhibitors, chemotherapies, and medroxyprogesterone acetate are among the other medical conditions and medications associated with secondary osteoporosis. The cellular and molecular mechanisms of bone turnover, the pathophysiology of osteoporosis, and their treatment are summarized in this review. Osteoclast genesis seems to be boosted by the nuclear factor-ligand uncoupling factor.

## DISCUSSION

Osteoprotegerin on the other hand, is antagonist that osteoblast lineage cells secrete. After suppressing and triggering the release of and estrogenic promotes apoptosis of osteoclasts and inhibits osteoclast genesis by stimulating the production of OPG and reducing osteoclast differentiation consequently, leading to more bone loss. Expression rises, and the pathway is inhibited in osteoblasts when glucocorticoids are used excessively. By increasing expression and decreasing expression, they aid in the survival of osteoclasts. The primary treatment for hormone-related and glucocorticoid-induced osteoporosis is a suitable estrogenic supplement and avoiding excessive glucocorticoid use. Bisphosphonates, teriparatide and inhibitors are also part of the current pharmacological treatment. However, a lot of the specific cellular and molecular mechanisms that cause osteoporosis appear to be complicated and unexplored, so more research is needed.

Age, sex, American Society of Anaesthesiologists classification, living circumstances, fracture type, preoperative osteoporosis treatment, anticoagulation, and preoperative walking capacity were all noted at the time of inclusion. Both the surgical process and the interval between admission and operation were discovered. In addition to the actual operation, there was a distinction between closed and open procedures. Mortality during the acute inpatient stay as well as days later and a shift in the patient's location were the outcome criteria; the patient's capacity to walk was noted during follow-up days following admission [1].

Osteoporosis is a skeletal disorder that has a significant impact on socioeconomic systems. It is characterized by a decrease in bone mass caused by an imbalance between bone resorption and bone formation, which alters the bone's microstructure .The

**Correspondence to:** Kuo Chen, Department of Medicine and Gerontological, Mackay Memorial University, Taipei, Taiwan; E-mail: kuochen332@helath.edu

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deterioration of the microstructure of the bone, increases the likelihood of fracture, which can lead to impairment and disability. After a fragility fracture, significant functional decline is typically inevitable because osteoporosis is frequently asymptomatic and tends to be diagnosed clinically, meaning in the advanced stage As a result, early detection and prevention of osteoporosis are crucial to bone health Notable gamble elements of osteoporosis incorporate advanced age, postmenopausal status, maternal history of osteoporosis, smoking propensities, lacking calcium admission, inordinate liquor utilization, low active work, and extreme glucocorticoid use Osteoporotic fractures can lead to limb deformity, chronic pain, decreased quality of life, disability, additional complications, and even death [2].

Osteoporosis has become a major cause of morbidity and mortality among the elderly, putting a strain on public healthcare systems worldwide as life expectancy rises. It is considered as essential osteoporosis in post-menopausal ladies and in the older without a conspicuous sickness, while optional osteoporosis is a result of drugs or illnesses. Osteoporosis is linked to advanced age, postmenopausal status, and secondary causes that contribute to osteoporosis, such as chronic diseases and lifestyle issues, according to doctors. However, they may not be aware that a number of commonly prescribed medications have been linked to a lower and an increased risk of fracture, thereby negating the advantages of treatment efficacy and posing physical, economic, and psychosocial challenges for affected individuals, their families, and communities. In addition, patients may not be aware of osteoporosis until symptomatic fragility fractures occur because they lack relevant information about the risk. As a result, osteoporosis management and prevention measures are rarely implemented promptly, increasing the risk of osteoporotic fractures in patients [3].

A significant decline in their freedom is caused by the addition of 26 more patients to nursing homes. Due to the fact that some chores can't be completed with a rollator, one's freedom in terms of everyday activities also declines. Our findings agree with Peters et AL thorough.'s literature review they demonstrated that had a detrimental effect on the general health status and health-related quality of life of older patients with a particular focus on the substantial impairment of the physical, psychological, and social facets of health. They demonstrated that there is evidence in the literature that the pre-fracture state appears to have a deleterious influence on physical functioning or nutritional status. Accessing techniques: either averaging normalizing the signal or applying filters to eliminate unwanted signals [4].

The most common cause of hormone-related osteoporosis in postmenopausal women is oestrogen deficiency, which occurs as a result of the ovaries' natural aging process However, the most common form of drug-induced osteoporosis remains glucocorticoid-induced osteoporosis Other medications, in addition to glucocorticoids, increase the likelihood of developing osteoporosis. Proton pump inhibitors hypogonadism and agents that induce hypogonadism, selective serotonin receptor chemotherapies, medroxyprogesterone acetate antidepressants, anticonvulsants, inflammatory bowel disease, and thyroid hormone replacement or suppressive agents are all common medications and medical conditions that are associated with secondary osteoporosis This review aims to investigate the underlying mechanisms of bone remodeling, the overall effects of estrogenic deficiency on bone turnover, the pathophysiology of common osteoporosis-related medications, and the medical conditions that lead to secondary osteoporosis mentioned above. Additionally, as cancer treatment has advanced in recent years, the population of patients undergoing surgeries or chemotherapies that may result in secondary osteoporosis has also increased [5,6].

## CONCLUSION

Oestrogen promotes the apoptosis of osteoclasts and inhibits osteoclast genesis via several pathways. Estrogen not only stimulates the production of but also reduces the differentiation of osteoclasts by suppressing IL-1 and TNF, therefore inhibiting the release Oestrogen promotes the apoptosis of osteoclasts via the effect of .Estrogen deficiency leads to the uncoupling of bone resorption and formation, which means an increased osteoclastic resorption without a corresponding osteoblastic activity. The osteoblastic activity fails to catch up with increased osteoclastic resorption, therefore resulting in greater bone loss. Ligand appears to be the critical uncoupling factor that enhances osteoclast genesis. During estrogenic deficiency, both the production of and the sensitivity to IL-1 of stromal cells increase, stimulating stromal cells to release. The cascade leads to the secretion of RANKL from osteoblasts, binding to on osteoclasts, and promoting osteoclast development. On the other hand, is an antagonist against secreted by osteoblast lineage cells, and it contributes to the anti-restorative actions of estrogen.

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## CONFLICT OF INTEREST

None.

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