

“Celastrol” for Osteoporosis



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DEAR EDITOR,

Osteoporosis causes more than 8.9 million fractures annually worldwide (1). It is a disease leading to low bone mass, deterioration of bone tissue, and disruption of bone microarchitecture. This is a common public health problem that existed in women and older people (2). What is the treatment of osteoporosis? In western, some anti-resorptive agents, such as estrogen, bisphosphonates, and selective estrogen receptor modulators (SERMs) are the drugs of choice for osteoporosis, other treatments include parathyroid hormone (PTH), nutritional support of calcium, and vitamin D (3). However, it tends to have various potential side effects, e.g., influence on the end of a metabolic cycle of bone physiology, and structural changes of bones. It induces osteonecrosis of a jaw bone in the long-term doses treatment of bisphosphonates (4).

Therefore, there are many Chinese herbal medicines studied in China over the past 10 years, such as Herba Epimedii, Cortex Eucommiae, Radix Astragali, and Fructus Psoraleae (5).

“Celastrol” is another traditional Chinese herbal for investigating osteoporosis recently years. It contains a pentacyclic triterpenoid isolated from the root extracts of *Tripterygium wilfordii*, which possess anti-tumor, anti-fertile, and anti-inflammatory biological activities (6). According to the traditional Chinese theory, this is bitter, pungent, and cool in taste. Its functions are to dispel pathogenic wind and remove dampness, promote blood circulation and free meridians, detumescence for suppressing pains, destroying parasites, and detoxifying (7).

In the clinical study, it is useful for skin pruritus and rheumatic arthritis. Growing evidence has

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Table 1. Published papers of Celastrol for osteoporosis.

	Xi J et al. (2018) [8]	Cascão R et al. (2017) [9]	Li XD et al. (2016) [10]
Objective	The effects of Celastrol on glucocorticoid-induced osteoporosis (GIOP)	Celastrol preserves articular structures decreases the number of osteoclasts and osteoblasts present in arthritic joints	Celastrol is an NF- κ B inhibitor that ameliorates hypercalciuria and articular cartilage lesions in osteoporosis
Results	Reduce bodyweight Decrease the levels of urine calcium/creatinine, tartrate-resistant acid phosphatase 5b, C-terminal telopeptide of type I collagen Induce osteocalcin in GIOP	Reduce tartrate-resistant acid phosphatase 5b, procollagen type 1 amino-terminal propeptide, and C-terminal crosslinked telopeptide of type II collagen serum levels	Exhibit a significant decrease in urinary calcium excretion such as the concentrations of parathyroid hormone, tartrate-resistant acid phosphatase 5b, C-terminal telopeptide, and deoxyypyridinoline
Significance	Inhibit prostaglandin E2 and caspase 3 protein expression levels PI3K/AKT signaling pathways induce phosphoinositol 3 kinase (PI3K), phosphorylated protein kinase B (AKT), and glycogen synthase kinase 3 phosphorylation	Minimize bone loss and bone microarchitecture degradation	Improve the structure of articular cartilage and cancellous bone, significant decline in levels of NF- κ B (P65), MMP-1, and MMP-9 for preventing osteoporosis

shown that celastrol is also effective for osteoporosis (Table 1).

The above-updated information demonstrates that celastrol is an alternative Chinese herbal for the treatment of osteoporosis. However, discrepancies exist in celastrol dosing and toxicity. Much more works need to be done including the dosage and toxicity safety assessments of celastrol in the human clinical study.

AUTHOR CONTRIBUTIONS

All authors contributed to the concept, acquisition, and analysis of data, drafting of the article, and critical revision for important intellectual content.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to disclose.

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REFERENCES

1. Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporos Int* 2006;17(12):1726-33.
2. Sözen T, Özışık L, Başaran NÇ. An overview and management of osteoporosis. *Eur J Rheumatol* 2017;4(1):46-56.
3. Chen LR, Ko NY, Chen KH. Medical Treatment for Osteoporosis: From Molecular to Clinical Opinions. *Int J Mol Sci* 2019;20(9):2213.
4. Wang ZQ, Li JL, Sun YL, Yao M, Gao J, Yang Z, Shi Q, Cui XJ, Wang YJ. Chinese herbal medicine for osteoporosis: a systematic review of randomized controlled trials. *Evid Based Complement Alternat Med* 2013;2013:356260.
5. Yang F, Xu MR, Yang MF, Lam CH, Lau MT, Yuan ZH. Patterns of the use of traditional Chinese medicines in the treatment of osteoporosis. *Chin. J. Rehabil. Med* 2005;9(31):203-205.
6. Venkatesha SH, Moudgil KD. Celastrol and its role in controlling chronic diseases. *Antiinflammatory nutraceuticals and chronic diseases*. USA: Springer;2016:267-289.
7. Law S, Leung AW, Xu C. Is the traditional Chinese herb, "Celastrol" effective to combat COVID-19? *J. Mater. Environ. Sci* 2020;11(8):1205-1208.

8. Xi J, Li Q, Luo X, Wang Y, Li J, Guo L, Wu G. Celastrol inhibits glucocorticoid induced osteoporosis in rat via the PI3K/AKT and Wnt signaling pathways. *Mol Med Rep* 2018;18(5):4753-4759.
9. Cascão R, Vidal B, Jalmari Finnilä MA, Lopes IP, Teixeira RL, Saarakkala S, Moita LF, Fonseca JE. Effect of celastrol on bone structure and mechanics in arthritic rats. *RMD Open* 2017;3(2):e000438.
10. Liu XD, Cai F, Zhang Y, Yang A, Liu L. Celastrol, an NF- κ B inhibitor, ameliorates hypercalciuria and articular cartilage lesions in a mouse model of secondary osteoporosis. *J. Pharmacol. Sci* 2016;130(4):204-211.