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THE RELATIONSHIP BETWEEN OSTEOPOROSIS AND HORMONAL CHANGES IN THE POSTMENOPAUSAL PERIOD

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Annotation

This thesis explores the link between postmenopausal osteoporosis and hormonal changes, highlighting estrogen deficiency as the main cause of accelerated bone loss. Reduced estrogen increases osteoclast activity and decreases osteoblast function, leading to lower bone mineral density. Postmenopausal women may lose 2-3% of bone mass annually in the first five years after menopause. The study reviews hormonal mechanisms, diagnostic methods, and treatment strategies for managing postmenopausal osteoporosis.

Keywords: osteoporosis, menopause, estrogen deficiency, bone mineral density, hormone replacement therapy, bisphosphonates, osteoclast activity, osteoblast function, calcium metabolism, fracture prevention

Today, osteoporosis is a major global health concern for postmenopausal women, affecting around 200 million women and causing over 8.9 million fractures annually. As women now live longer, spending nearly one-third of their lives postmenopausal, the link between menopause and bone health has gained growing importance. After menopause, fracture risk rises sharply—vertebral fractures affect 25% of women over 65, and hip fractures occur in 15% of women by age 80. In the U.S. alone, related healthcare costs exceed 17 billion dollars annually. The postmenopausal transition brings a 90% drop in estrogen levels, along with declines in testosterone, growth hormone, and insulin-like growth factor-1. These hormonal shifts disrupt bone metabolism, accelerating bone resorption, reducing formation, and increasing fracture risk.

Estrogen withdrawal after menopause upsets skeletal balance. Estrogen receptors in bone-forming osteoblasts, bone-resorbing osteoclasts, and osteocytes normally sustain bone homeostasis; when hormone levels fall, remodeling accelerates and resorption outweighs formation. Lack of estrogen increases production of receptor activator of nuclear factor kappa-B ligand while lowering its natural inhibitor, osteoprotegerin, thereby expanding osteoclast number and life span. At the same time osteoblast activity declines because bone morphogenetic proteins and collagen synthesis are suppressed. Observational work with dual-energy X-ray absorptiometry shows that trabecular-rich spine and hip lose three to five percent of mineral content each year during the first five postmenopausal years, whereas cortical regions such as the forearm lose one to two percent. High-resolution peripheral quantitative computed tomography adds detail, revealing trabecular thinning, wider spaces, and disrupted connectivity-changes that make bones weaker than density measurements alone suggest. Diagnostic assessment blends mineral density testing, the Fracture Risk Assessment Tool for individual probability, and biochemical markers such as the C-terminal telopeptide of type I collagen and the amino-terminal propeptide of type I procollagen. The World Health Organization defines osteoporosis as bone mineral density at least two point five standard deviations below the young-adult mean at the spine, hip, or forearm. Treatment begins with hormone replacement therapy started early after menopause, which can cut vertebral and hip fractures by up to seventy percent but must be weighed against cardiovascular and breast-cancer hazards. Anti-resorptive bisphosphonates-including alendronate, risedronate, and zoledronic acid-lower fracture risk by forty to seventy percent, though rare events such as atypical femoral fracture and jaw osteonecrosis require vigilance. New choices include the monoclonal antibody denosumab, which neutralizes receptor activator of nuclear factor kappa-B ligand, and anabolic peptides such as teriparatide and abaloparatide that actively build bone for women with severe or unresponsive disease.

In conclusion, the relationship between osteoporosis and hormonal changes after menopause involves complex biological mechanisms that lead to bone weakening and higher fracture risk. Estrogen deficiency is the main cause of this process, affecting bone cells, structure, and strength. These changes greatly increase

the risk of serious fractures in postmenopausal women. Modern diagnostics help assess bone health, while new treatments effectively reduce bone loss and fractures. Future research should focus on personalized approaches based on individual genetic, hormonal, and lifestyle factors.

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