

GUT DYSBIOSIS AS A MODULATOR OF BONE REMODELING: IMPLICATIONS FOR OSTEOPOROSIS

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RESUMO

Introduction: Osteoporosis (OP) is a chronic metabolic disease characterized by reduced bone mass and deterioration of microarchitecture, resulting in an increased risk of fractures. Recent evidence points to the gut microbiota (GM) as a central regulator of bone homeostasis through immunological, endocrine, and metabolic pathways. Dysbiosis promotes systemic inflammation, alters estrogen metabolism, impairs calcium absorption, and increases osteoclastic activity, thereby accelerating bone loss. Understanding how bone homeostasis between osteoclasts and osteoblasts is influenced by the gut microbiota through metabolic, endocrine, and immunological pathways is vital for the prospect of new clinical strategies for the treatment of osteoporosis. **Objective:** The objective of this study is to understand the mechanisms involved in the relationship between the gut microbiota and bone remodeling, as well as therapeutic perspectives arising from this interaction. **Materials and methods:** This study was conducted in the form of a narrative literature review, aiming to synthesize current evidence on the relationship between gut microbiota and osteoporosis. The bibliographic search was performed in PubMed, using combinations of descriptors such as "gut microbiota", "osteoporosis", "bone remodeling", "probiotics", and "short-chain fatty acids". Original articles, systematic reviews, meta-analyses, and experimental reports published between 2024 and 2026 were included. Inclusion criteria considered studies addressing immunological, endocrine, and metabolic mechanisms of the gut microbiota related to bone remodeling, as well as therapeutic strategies involving probiotics, prebiotics, synbiotics, and fecal microbiota transplantation.

Results: Literature analysis indicates that the gut microbiota exerts a direct influence on bone remodeling through immunological, endocrine, and metabolic mechanisms. Protective effects were identified associated with the production of short-chain fatty acids (SCFAs), which favor the expansion of regulatory T cells and osteoblast differentiation, in addition to improving calcium absorption. Conversely, metabolites such as trimethylamine N-oxide (TMAO) were shown to be related to increased bone resorption. Therapeutic strategies involving probiotics, prebiotics, and synbiotics showed positive results in clinical and experimental studies, with improvements in bone formation markers and a reduction in osteoclastic activity. The findings reinforce that the gut microbiota is a promising target for the prevention and treatment of osteoporosis. Modulating microbial composition-whether through probiotics, prebiotics, or fecal microbiota transplantation-can contribute to restoring eubiosis, reducing systemic inflammation, and preserving bone health. **Conclusion:** The integration of microbiota-based therapeutic approaches represents an innovative and complementary strategy to conventional osteoporosis therapies. The manipulation of the gut microbiota, through specific probiotics and prebiotics, promotes an increase in bone mineral density and improves microarchitecture, reducing the risk of fractures in individuals with osteoporosis.

Palavras-chave: Gut Microbiota Osteoporosis Bone Remodeling Dysbiosis.