

# Gut Bacteria Linked to Osteoporosis Risk in Primary Hyperparathyroidism

*Researchers identify Bifidobacterium longum as a potential microbial driver of bone loss through immune-mediated mechanisms*

CHENGDU, CHINA, June 12, 2026

/EINPresswire.com/ -- Researchers

from Emory University and Columbia University have uncovered a link between the gut microbiome and bone loss in patients with primary hyperparathyroidism (PHPT). The study demonstrates that specific gut bacteria, particularly *Bifidobacterium longum*, may influence bone density by stimulating immune cells that promote bone resorption. The findings suggest that gut microbiome composition could help predict osteoporosis risk in PHPT patients and may open new avenues for preventive interventions.



Researchers identified *Bifidobacterium longum* as a gut bacterial species associated with immune activation and reduced bone density in patients with primary hyperparathyroidism.

Primary hyperparathyroidism (PHPT) is a common endocrine disorder characterized by excessive secretion of parathyroid hormone (PTH). While some patients experience substantial bone loss and an increased risk of fractures, others with similar hormone levels maintain relatively healthy bones. Until now, the reason for this variability has remained unclear.

Now, in a new study led by Professor Roberto Pacifici, published on 25 May 2026 in [Bone Research](#), researchers investigated whether the gut microbiome could explain differences in skeletal outcomes among PHPT patients. The team analyzed stool samples, bone density measurements, and immune-cell profiles from 50 individuals with PHPT. Their findings revealed that gut microbial composition was closely associated with bone health and immune activity.

To test whether the microbiome directly influences bone loss, the researchers performed fecal microbiota transfer (FMT) experiments, transplanting gut microbiota from PHPT patients with osteoporosis, osteopenia, or normal bone density into germ-free mice. Remarkably, mice receiving microbiota from osteoporotic patients developed greater bone loss and exhibited

increased levels of inflammatory immune cells compared with mice receiving microbiota from patients with healthier bones. “We show that the extent to which primary hyperparathyroidism impacts the human skeleton correlated with the abundance of *Bifidobacterium longum*, a taxon that we show to induce the expansion of both intestinal and BM TNF+ T cells and Th17 cells,” explained Prof. Pacifici.

The study identified two key immune cell populations, tumor necrosis factor (TNF)-producing T cells and T helper 17 (Th17) cells, as critical mediators linking the gut microbiome to bone deterioration. Researchers found that higher levels of these immune cells were consistently associated with lower bone density in both patients with PHPT and recipient mice that received microbiota transplants from these patients. These findings suggest that immune activation may serve as an important biological bridge between gut bacteria and skeletal health.

To further investigate this connection, the researchers analyzed the relationship between specific bacterial species and immune-cell activity. Among the bacterial species identified, *Bifidobacterium longum* emerged as a particularly influential microbe. Statistical analyses revealed that increased abundance of *Bifidobacterium longum* was associated with elevated expression of the inflammatory molecules TNF and IL-17, both of which are known to promote bone resorption. These inflammatory signals were, in turn, associated with lower bone mineral density and poorer bone structure in patients with PHPT in experimental models.

The team then conducted a series of mechanistic experiments to determine whether *Bifidobacterium longum* could directly influence bone health. Using germ-free mouse models, they demonstrated that colonization with *Bifidobacterium longum* stimulated the expansion of TNF-producing T cells and Th17 cells in the intestine and bone marrow. The bacterium also enhanced the migration of these immune cells from the gut to the bone marrow, where they released inflammatory factors capable of accelerating bone breakdown. When exposed to elevated parathyroid hormone levels, mice harboring *Bifidobacterium longum* experienced significantly greater bone loss than control animals, providing direct evidence that this bacterial species can amplify the skeletal effects of PHPT.

Interestingly, the researchers found no significant differences in overall microbiome composition among patients with osteoporosis, osteopenia, or normal bone density. Instead, susceptibility to bone loss appeared to depend on the abundance of specific bacterial species rather than broad changes in the microbial community. This finding highlights the importance of identifying functionally relevant microbes that may influence disease outcomes. Together, the results suggest that the gut microbiome is not merely associated with skeletal health but may actively determine the severity of bone loss in patients with PHPT. The identification of *Bifidobacterium longum* and immune-cell signatures linked to osteoporosis risk raises the possibility of developing microbiome-based biomarkers to identify patients who are most vulnerable to skeletal complications. “These findings confirmed that the presence of *Bifidobacterium longum* in the gut microbiome allows PTH to cause the expansion and migration of TNF+ T cells and Th17 cells and to induce bone loss,” concluded Prof. Pacifici.

Beyond improving risk prediction, the findings also point toward new therapeutic opportunities. Targeted microbiome interventions, including selective microbial modulation, antibiotics, precision probiotics, or other microbiota-directed strategies, may eventually help prevent or reduce bone loss in patients with PHPT. Such approaches could complement existing treatments and pave the way for more personalized management of metabolic bone disease.

## Reference

Title of original paper: Bacterial specificity of the gut microbiome predicts bone density in primary hyperparathyroidism

Journal: Bone Research

DOI: <https://doi.org/10.1038/s41413-026-00529-1>

## About Emory University

Emory University is a leading research university based in Atlanta, Georgia, dedicated to advancing knowledge and improving human health through innovative research, education, and clinical care. Its multidisciplinary programs bring together experts across biomedical sciences, medicine, public health, and microbiome research to address complex health challenges.

Website: <https://www.emory.edu/home/index.html>

## About Professor Roberto Pacifici from Emory University

Dr. Roberto Pacifici, MD, is the Garland Herndon Distinguished Professor of Medicine and Director of the Division of Endocrinology, Metabolism and Lipids. He completed fellowship training in Endocrinology and Metabolism at Washington University School of Medicine. With nearly four decades of research and clinical experience since 1988, Prof. Pacifici is widely recognized as a pioneer of osteoimmunology and osteomicrobiology. His research focuses on the interactions between the immune system, gut microbiome, and bone health. He has authored more than 100 peer-reviewed publications and book chapters, and has made seminal contributions to understanding how immune cells and gut microbes regulate bone metabolism.

## Funding information

This study was supported by grants from the National Institutes of Health (DK124821 and RR028009 to R.P. and DK09839 to R.M.J.). This study was also supported by the Emory University Emory Integrated Computational Core Facility (RRID:SCR\_023525) and by the Emory University gnotobiotic core. M.N.W. was supported in part by Medical Health Research Career Scientist Award Number IK6RD001819 from the United States Department of Veterans Affairs Medical

Health Portfolio.

Yini Bao

Sichuan University

+86 2885546461

br@scu.edu.cn

Visit us on social media:

[X](#)

---

This press release can be viewed online at: <https://www.einpresswire.com/article/919191459>

EIN Presswire's priority is source transparency. We do not allow opaque clients, and our editors try to be careful about weeding out false and misleading content. As a user, if you see something we have missed, please do bring it to our attention. Your help is welcome. EIN Presswire, Everyone's Internet News Presswire™, tries to define some of the boundaries that are reasonable in today's world. Please see our Editorial Guidelines for more information.

© 1995-2026 Newsmatics Inc. All Right Reserved.