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Gut Microbiome - Should we treat the gut and not the bones?

Madhan Jeyaraman [a, *], Arulkumar Nallakumarasamy [b], Vijay Kumar Jain [c]

[a] Department of Orthopaedics, ACS Medical College and Hospital, Dr MGR Educational and Research Institute, Chennai, 600056, Tamil Nadu, India
[b] Department of Orthopaedics, All India Institute of Medical Sciences, Bhubaneswar, 751019, Odisha, India
[c] Department of Orthopaedics, Atal Bihari Vajpayee Institute of Medical Sciences, Dr Ram Manohar Lohia Hospital, New Delhi, 110001, India

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ABSTRACT

Gut microbiome (GM) forms an integral part of homeostasis of an individual. Due to the recent development of metagenomics, the plausibility of sequencing GM and its therapeutic ability for various diseases has been explored. Dysbiosis or disequilibrium or pertubations of GM leads to disruption of intercommunication signaling among gut-bone axis, gut-bone-brain axis, and gut-disc axis resulting in the progression of various chronic diseases. The therapeutic interventions to restore the GM like prebiotics and probiotics, bacteriophage therapy, fecal microbiota transplantation, and physical biomodulation have been identified. This review throw the lime light on the effect of gut dysbiosis in musculoskeletal diseases.

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1. Introduction

Gut microbiota (GM) forms an integral part of the gut functioning, host metabolism, and homeostasis, which has a potential role as a novel therapeutic agent. Metagenomics play a vital role in characterising and sequencing the microbes in GM. GI epithelium translates immunological responses in the lamina propria which are recognised by toll-like receptors (TLRs) and NOD-like receptors (NLR). They recognise pathogen-associated molecular patterns such as lipopolysaccharides, lipotechoic acid and peptidoglycans from microorganisms. Current understanding of GM signifies the communication of signaling pathways between GM and human cells to promote the immunological interactions. GM plays a significant role in cardiometabolic disorders, diabetes, autoimmune disordes, musculoskeletal disorders, and neuropsychiatric disorders.

2. Gut microbiome

GM refers to the collective genomes of the micro-organisms in the gut ranging from bacteria, virus, fungi, and protozoa. Human gut caters 100 trillion microbes, which can be quantified by next generation sequencing (NGS). The human GM contains approximately 35,000 bacterial species. In 2007, the National Institutes of Health (NIH) launched the Human Microbiome Project, which has identified microbiota on various parts of the human body, which belong to four major phyla in humans.[1] The composition of healthy human GM is a) Bacteroidetes, b) Firmicutes, c) Actinobacteria, d) Proteobacteria, and e) Fusobacteria (*Fusobacterium)*.[2] The biomedical community throw light on GM due to the potential for mediation of disease casuatuion, prognosticator for disease progression, and a target for therapeutic interventions.

3. Gut-bone axis

Gut-bone axis refers to the intercommunication signals between gut microbiota and the skeletal system. Various researchers postulated that dysbiosis of the gut leads to dysregulation of bone turnover. Mendelian randomization approach demonstrated a significant association between gut microbiota and bone mineral density. Short-term gut microbiota administration enhances bone turnover and downregulates femoral bone mass in germ-free mice. Enhanced radial and longitudinal femoral growth were observed with long-term gut microbiota colonization. The administration of antibiotics interferes with gut microbiota which in turn alters the biomechanical properties and bone mass. Insulin-like growth factor-1 regulates the gut microbiome which forms a bridge in the gut-bone axis.[3] Hydrogen sulfide, a bone regulator, alters the gut microbial composition, which enhances osteogenesis via Wnt signaling.[3] Short-chain fatty acids (SCFAs) [acetate, propionate, and butyrate] are powerful immune cell regulators, which facilitate osteogenesis through Treg cell-mediated regulation of $CD8^+$ T cell

* Corresponding author.
E-mail address: madhanjeyaraman@gmail.com (M. Jeyaraman).

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Wnt10 b production and osteoclastogenesis.[3] The cross talks between gut and musculoskeletal system is depicted in Fig. 1.

4. Clinical significance of gut microbiome in orthopaedics

a) Osteoarthritis (OA): A systematic review has demonstrated a strong evidence for role of gut-joint axis in OA.[4] Various literature state that dysbiotic gut is a hidden risk factor for the pathogenesis of OA disease progression due to the production of pro-inflammatory cytokines and bacterial metabolites. Such metabolites create "leaky gut" and enter into systemic circulation causing systemic inflammation.[5] Elevated lipopolysaccharide (LPS) levels induce low-grade inflammation, endotoxemia, activation of macrophages, and joint damage. Gender specific gut dysbiosis were also observed in OA cases. In females with OA, there were higher abundance of *Bacteroides* and *Bilophila* whereas in males with OA, there were higher abundance of *Veillonella* and *Methanobrevibacter*.[4] Bilateral communication signaling between gut-bone axis and central nervou system (CNS) center (gut-brain-bone axis) such as hypothalamic-pituitary axis, nucleus tractus solitarius, and hypothalamic suprachiasmatic nucleus play a significant role in the progression of OA. Pertubations among circadian rhythm, gut microbiota, and redox potentials are involved in the pathogenesis of OA.[5] In humans, a plausible link between OA and gut dysbiosis was observed with serum and synovial fluid LPS levels with activated macrophages in the knee joint capsule, synovium, and joint space narrowing along with worsened WOMAC scores. Abundant *Streptococcus taxa*, a pro-inflammatory microbe, was elevated in hip and knee OA with increased WOMAC scores.[4] For OA treatment, the gut is an exciting and novel target. Dietary changes or fibre, probiotics, or prebiotic supplements may have a favourable effect on the gut joint axis.[5,6]

b) Rheumatoid arthritis (RA): Rheumatoid arthritis pose a web of causatuion for the disease pathogenesis. Literature evidence says that there is a causal association between periodontal disease (PD) and RA. PD is caused by polymicrobial population such as *P. gingivalis, Prevotella intermedia, Tannerella forsythia*, and *Aggregatibacter actinomycetemcomitans.* Various studies elicited the prevalence of 2-fold RA manifestations in PD cases. Higher prevalence of severe periodontitis was observed in patients with RA, suggesting the severity of periodontitis is related to severity of RA.[7] *Porphyromonas gingivalis* correlate serologically with anticitrullinated peptide antibody tittres, an oral anaerobe causinh periodontitis, found to be increased in RA patients.[7] Microbial perturbation *Prevotella copri* is increased in newly-onset untreated RA cases, which induces Th17 cell responses leading to severe arthritis. The cross-talk between Th17 responses and *P. corpi* peptide provides a plausible link for the pathogenesis of RA.[8] Urinary tract infection caused by *Proteus mirabilis* found to be increased in patients with RA, OA, fibromyalgia, and PsA.[9] Abundance of *Eggerthella lenta* and *Collinsella* were observed in patients with RA disease.[9] Downregulation of gram-negative bacteria with upregulation of gram-positive bacteria correlates with the GM dysbiosis in RA individuals. Gut dysbiotic environment in RA must be reversed by the supplementation of prebiotics & probiotics and fecal microbiota transplantation, which can reduced the inflammatory phase leading to eubiosis.[9]

c) Spondyloarthropathies: Inflammation of gut leads to the epithelial dysbarrier axis leading to disruption of gut mucosal homeostasis redulting in gut microbiota dysbiosis. The understanding of temporo-spatial relationship between enhanced intestinal permeability and gut dysbiosis is still under debate and in infancy. Dysequilibrium of GM play a significant role in the pathogenesis of enteropathic arthropathies [spondyloarthropathy (SpA), ankylosing spondtlitis (AS), reactive arthritis (ReA), inflammatory bowel disease (IBD)-mediated arthropathy, juvenile spondyloarthropathy (JSpA), and psoriatic arthritis (PsA)].[10] In AS cases, an increased colonization of *Lachnospiraceae*, *Ruminococcaceae*, and *Prevotellaceae* were observed in terminal ileum.[11] Literature evidences state that decreased abundance of *Streptococcus* and *Actinomyces* were associated with ReA cases. Klebsiella pneumonia is postulated to be involved in

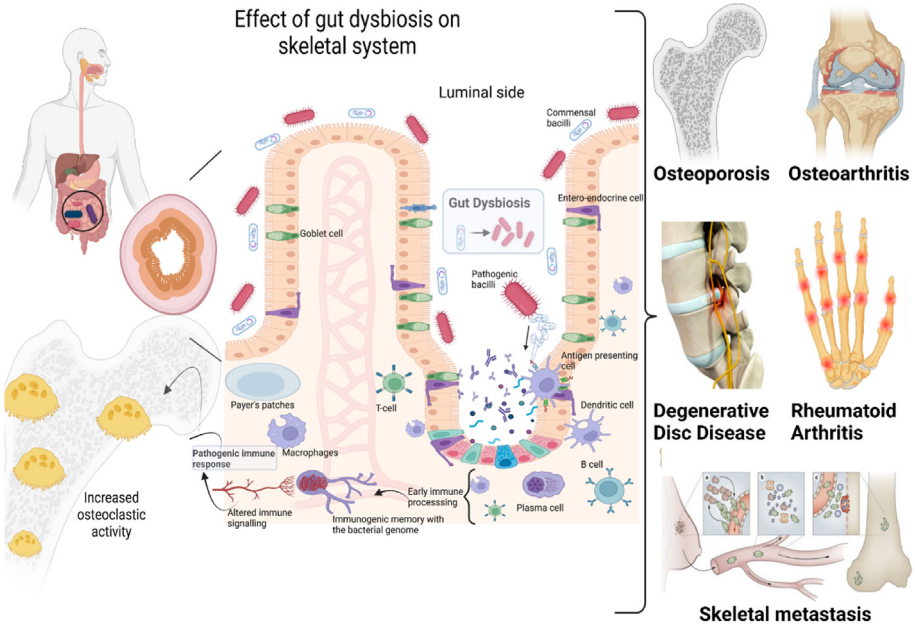


Fig. 1. Cross-talks between gut and musculoskeletal system.

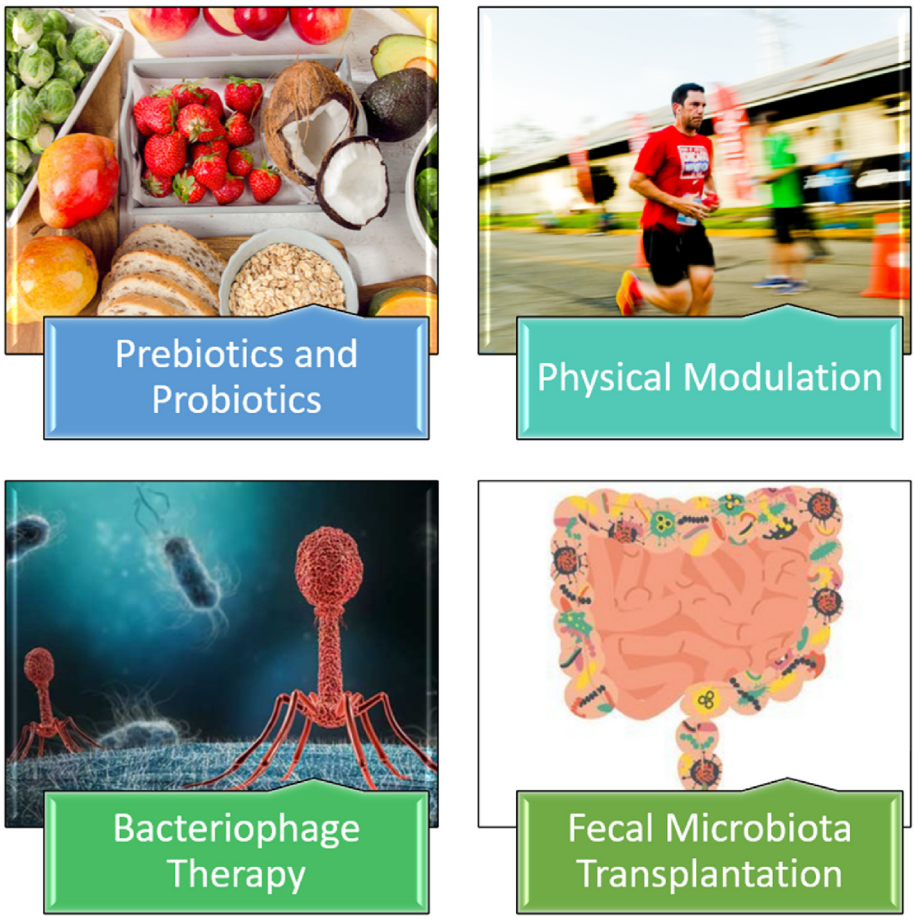


Fig. 2. Forms of gut microbiota restoration.

the AS pathogenesis with higher level of IgA antibodies in the serum.[11] The usage of prebiotics and probiotics in HLA-B27 transgenic rats with SpA demonstrated sutle benefits in both colitis and gut-mediated arthritis.[12] An increased levels of *Clostridiacece* group of bacteria in gut is associated with IBD-related arthropathy and rheumatoid arthritis.[13] Fecal microbiota analysis revealed the enhanced *Ruminococcus gnavus* levels are correlated with the evidence of SpA and IBD.[13] The subclinical gut inflammation leads to disturbed microbial flora namely *Ruminococcus, Akkermansia,* and *Coprococcus* leading to the development of PsA susceptible individuals.[13] The common gut commensal *Alistipes* is found decreased in both PsA and IBD individuals. In AS and JSpA, the microbes such as *Clostridium leptum* and *Fecalibacterium prausnitzii* were found decreased in fecal microbial analysis.[11] In JSpA, there are some evidence of enhance cellular immune response against outer membrane protein of *Salmonella typhimurium* and dysbiosis of *Bacteroides* genus and *Akkermansia muciniphila.*[14]

d) Osteoporosis: Researchers have postulated the temporal association between gut dysbiosis and the quality of bone formation and destruction. Osteoporosis is mediated by nitric oxide (eNOS and iNOS), endocrine (calcitonin, insulin-like growth factor-1, glucagon-like peptide-1, thyroxine, and estrogen), gut-brain-bone axis (5-HT), homocysteine, and immune-mediated (Tregs and T17 cells) signaling pathways. Dysbiotic GM leads to the release of inflammatory cytokines which activates osteoclastogenic pathway and results in osteoporosis. In ovariectomized mice, supplementation of Lactobacillis and Bifidobacteria facilitated the absorption of Vit D and enhanced the differentiated of osteoblast by reducing the differentiation of osteoclast and prevting the bone loss.[15] SCFAs supplementation restores GM, induces anti-inflammatory responses, and promotes the calcium absorption in the intestine and hence increasing the bone mineral density. Intestinal flora transplantation resulted in reducing the morbidity of gut-mediated bone loss.[15] GM transplantation (as shown in Fig. 2) maintains the equilibrium between the microbes and the signaling patwhays of bone formation and resportion, upregulate the proliferation of osteoblasts, downregulate the osteoclastic resoprtion, inhibit apoptosis of osteoblasts, and hence minimising the degree of osteoporosis.[15]

e) Bone Metastasis: GM regulate and enhance skeletal remodeling and bone metastasis in breast cancer, small cell and non small cell lung cancer.[16] In brain metastasis of non small cell lung carcinoma, *P. aeruginosa* was found in abundance.[17] A recent study found that *Streptococcus, Campylobacter*, and *Moraxellaceae* were more prevalent in bone metastasis in breast cancer than in the control group.[18] Downregulation of *Megamonas* and *Akkermansia* are associated with berast cancer with bone metastasis.[18] These studies suggest that the gut microbiota may be a desirable target for the creation of cancer-fighting therapeutics and diagnostic procedures.

f) Fracture-related Infections (FRI): There is a plausible link between GM and FRIs. FRI cases exhibit deranged bacterial

load with upregulated gut dysbiotic index than fracture healed cases. *Escherichia, Staphylociccus, Streptococcus*, and *Enterococcus* were found abundant in FRI group when compared to fracture healed group. FRI cases show down-regulation in the class Clostridia. Various researchers have documented in animal models that the increased intestinal permeability after fracture remodeling. Subsequent supplementation of *Akkermansia* repair the intestinal mucosa and enhance fracture healing. The local serum inflammatory markers correlate positively with *E. coli* and hegatively with *Roseburia* in FRI. PPAR-γ pathway inhibition in intestinal epithelial cells leads to an increase of *E. coli* in GM and a decrease of butyrate producing bacteria, as proven by the GM sequencing in FRI patients. However, the intercommunication signal between PPAR-γ pathway and GM warrants further study on FRI.[19]

g) Degenerative disc diseases and low back ache: The concept of gut-disc and gut-bone marrow axes explain the causal relationship of GM in degenerative disc disease and low back ache. GM translocation to intervertebral disc (IVD) by disrupting gut epithelial barrier induces and enhances the pathogenesis of degenerative disc diseases and low back ache. In normal IVD, *Firmicutes, Actinobacteria*, and *Saccharopolyspora* are found to be abundant. *Bacillus coagulans* and *Bacillus clausii* were found to be upregulated in degenerated and herniated IVDs. In sub clinical and inflammatory responses inducing degeneration of discs, *Pseudomonas veronii, Pseudomonas stutzeri,* and *Streptococcus anginosus* were found to be increased.[20] In normal IVD, *Lactobacillus mucosae*, *Blautia producta, Propionibacterium granulosum*, *Sphingomonas yabuuchiae,* and *Staphylococcus epidermidis* were found to be abundant whereas in degenerated disc and disc herniation, *Prevotella tannerae*, *Halomonas nitritophilus,* and *Streptococcus alactolyticus* were present. But *Streptococcus anginosus*, *Prevotella pallens*, *Avibacterium gallinarum,* and *Enterobacter cowanii,* were found only in degenerated disc.[21] By regulating and transplanting the dysbiotic gut with either prebiotics and probiotics or fecal microbiota transplantation, the disc degeneration and low back ache can be prevented.

5. Future directives

Gut microbiota (GM) forms the next generation treatment modalities in orthopaedic disorders. The key focus must be on the alterations of gut microbiota composition at taxonomic levels and the functioning of each microbe. Metagenomics serve a major tool in tracing the signaling pathways of gut-bone axis. The interventional targets of gut barrier function and the production of SCFAs enact to explore the wide range of gut-bone signaling pathways to implement the treatment on various musculoskeletal disorders. The transplantation of GM to maintain bone homeostasis is still debatable. More preclinical and clinical studies have to be performed to interrogate and validate the key inflammatory mediators that influence gut-bone axis to reduce the morbidity of gut-mediated musculoskeletal disorders.

6. Conclusions

The musculoskeletal disorders are chronic and non-communicable in nature. Various postulates stated gut dysbiosis leads to various musculoskeletal ailemnts. GM-based therapeutics are potentially inexpensive, which address the molecular target in the chronic musculoskeletal disorders. Though our understanding on GM is in infancy, variousclinical studies must be taken up on the role and benefical clinical effect of GM in musculoskeletal disorders to validate the results on the larger scale.

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Informed consent (patient/guardian), mandatory only for case reports/clinical images

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Institutional ethical committee approval (for all human studies)

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Authors contributions

Conceptualisation and design – VKJ; Administrative support – VKJ; Provision of study materials or patients – MJ; Collection and assembly of data – MJ and AKN; Figure preparation – MJ and AKN; Manuscript writing – MJ and AKN; Proof reading – VKJ. All authors have read and agreed to the published version of the manuscript.

Declaration of competing interest

Nil.

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Update

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Erratum regarding missing peer review statement in previously published articles

Owing to a Publisher error, peer review statement was not included in the published versions of the following articles, that appeared in previous issues of Journal of Clinical Orthopaedics and Trauma.

The appropriate peer review statements, for each article are provided below.

1. "Correlation between height and semitendinosus tendon length, prediction of minimum semitendinosus tendon length based on height-an easy and accurate method" (Journal of Clinical Orthopaedics and Trauma., 31 (2022) 101918) https://doi.org/10.1016/j.jcot.2022.101918

Peer review statement: The author list of this article includes an editorial board members, namely Ankit Goyal and Hitesh Lal. We would like to note that they were not involved in the peer review process or any of the decision-making steps of the manuscript.

2. "Cystic bone Lesions: Diagnostic pitfalls and therapeutic considerations" (Journal of Clinical Orthopaedics and Trauma., 67 (2025) 103046) https://doi.org/10.1016/j.jcot.2025.103046

Peer review statement: The author list of this article includes an editorial board member, namely Ashish Gulia. We would like to note that they were not involved in the peer review process or any of the decision-making steps of the manuscript.

3. "Artificial intelligence-based orthopaedic perpetual design" (Journal of Clinical Orthopaedics and Trauma., 49 (2024) 102356) https://doi.org/10.1016/j.jcot.2024.102356

Peer review statement: The author list of this article includes an editorial board members, namely Abid Haleem, Abhishek Vaish and Raju Vaishya. We would like to note that they were not involved in the peer review process or any of the decision-making steps of the manuscript.

4. "Gut Microbiome - Should we treat the gut and not the bones?" (Journal of Clinical Orthopaedics and Trauma., 39 (2023) 102149) https://doi.org/10.1016/j.jcot.2023.102149

Peer review statement: The author list of this article includes an editorial board member, namely Vijay Kumar Jain. We would like to note that they were not involved in the peer review process or any of the decision-making steps of the manuscript.

5. "Decoding the hidden realm: Molecular pioneering unravelling osteoarticular tuberculosis diagnosis" (Journal of Clinical Orthopaedics and Trauma., 56 (2024) 102538) https://doi.org/10.1016/j.jcot.2024.102538

Peer review statement: The author list of this article includes an editorial board members, namely Vijay Kumar Jain and Kartikeyan Iyengar. We would like to note that they were not involved in the peer review process or any of the decision-making steps of the manuscript.

6. "Trends of publications on primary sarcomas of bone: A bibliometric analysis" (Journal of Clinical Orthopaedics and Trauma., 63 (2025) 102917) https://doi.org/10.1016/j.jcot.2025.102917

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7. "Bacteriophage therapy in infection after fracture fixation (IAFF) in orthopaedic surgery" (Journal of Clinical Orthopaedics and Trauma., 35 (2022) 102067) https://doi.org/10.1016/j.jcot.2022.102067

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8. "Combating antimicrobial resistance in osteoarticular infections: Current strategies and future directions" (Journal of Clinical Orthopaedics and Trauma., 58 (2024) 102791) https://doi.org/10.1016/j.jcot.2024.102791

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9. "*Mycobacterium Tuberculosis* infection of the wrist joint: A current concepts review" (Journal of Clinical Orthopaedics and Trauma., 44 (2023) 102257) https://doi.org/10.1016/j.jcot.2023.102257

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10. "Bone graft tuberculosis outbreak in USA: Is it a concern in India?" (Journal of Clinical Orthopaedics and Trauma., 39 (2023) 102145) https://doi.org/10.1016/j.jcot.2023.102145

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11. "Letter to editor: Sathyendra et al. Comparative study of local injections of autologous platelet rich plasma versus corticosteroid in management of chronic plantar fasciitis" (Journal of Clinical Orthopaedics and Trauma., 62 (2025) 102903) https://doi.org/10.1016/j.jcot.2024.102903

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