

# Microplastic Exposure and Bone Health-A Narrative Review

Anusha Rani<sup>1</sup>, Bhagyajyothi M Bhat<sup>2\*</sup>, Rashmi Bhat<sup>3</sup>, Hemalatha Bangera<sup>4</sup>, Edward Lawrence Nazareth<sup>5</sup>

<sup>1</sup>Tutor, Dept of Anatomy, Srinivas Institute of Medical Sciences and Research Centre, Mukka, Karnataka, India

<sup>2</sup>Professor, Dept of Biochemistry, Srinivas Institute of Medical Sciences and Research Centre, Mukka, Karnataka, India

<sup>3</sup> Prof & Head, Dept of Anatomy, Srinivas Institute of Medical Sciences and Research Centre, Mukka, Karnataka, India

<sup>4</sup>Associate Professor, Dept of Anatomy, Srinivas Institute of Medical Sciences and Research Centre, Mukka, Karnataka, India

<sup>5</sup>Prof & Head, Dept of Orthopedics, Srinivas Institute of Medical Sciences and Research Centre, Mukka, Karnataka, India

**\*Address for Correspondence:** Dr. Bhagyajyothi M Bhat, Professor, Department of Biochemistry, Srinivas Institute of Medical Sciences and Research Centre, Mukka-574146, Karnataka, India

**E-mail:** [bhatbhagya48@gmail.com](mailto:bhatbhagya48@gmail.com)

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## ABSTRACT

Microplastics are emerging environmental pollutants with potential adverse effects on human health. Increasing experimental evidence suggests that microplastic exposure may affect skeletal health through oxidative stress, inflammation, mitochondrial dysfunction, and endocrine disruption. This narrative review summarizes current evidence on the relationship between microplastic exposure and bone health, with particular emphasis on bone remodeling, osteoblast and osteoclast activity, and associated biomarkers. Microplastics enter the human body mainly through ingestion and inhalation and have been detected in various human tissues, including skeletal tissues. Experimental studies indicate that microplastics can increase reactive oxygen species, activate inflammatory pathways, impair mitochondrial function, and interfere with estrogen signaling, thereby promoting osteoclastogenesis and reducing osteoblast activity. These changes may contribute to altered bone turnover and osteoporosis-like changes. However, most available evidence is derived from *in vitro* and animal studies, while human studies remain limited. Biomarkers such as superoxide dismutase, malondialdehyde, catalase, interleukin-6, alkaline phosphatase, procollagen type 1 N-terminal propeptide, C-terminal telopeptide, and tartrate-resistant acid phosphatase may help evaluate microplastic-associated skeletal toxicity. Further well-designed human studies and standardized methods for detecting and quantifying microplastics are required to clarify their clinical significance and potential role in osteoporosis.

**Key-words:** Microplastics, Bone health, Osteoporosis, Oxidative stress, Inflammation, Bone turnover markers, Skeletal toxicity

## INTRODUCTION

Microplastics are defined as plastic particles smaller than 5mm in diameter. The widespread use of plastic is largely attributed to their durability, versatility, and cost-effectiveness. Nevertheless, approximately 20% of plastic waste produced globally is recycled, whereas the remaining waste accumulates in environmental compartments such as rivers and soil <sup>[1]</sup>.

The ingestion of contaminated food and water, inhalation of airborne particles, and, to a lesser extent, skin contact with products containing plastic particles are the main ways that microplastics can enter the human body <sup>[2]</sup>. Human blood, placenta, lung, liver, breast milk, reproductive tissue, and even bone tissue have all been found to contain microplastics <sup>[3]</sup>.

Research from animal and experimental studies suggests that exposure to microplastics can interfere with normal bone metabolism by causing oxidative stress, inflammatory responses and mitochondrial dysfunction <sup>[4]</sup>. Scientists have discovered the effects of microplastics on the respiratory, cardiovascular, gastrointestinal systems, but their impacts on the skeletal system remain unexplored <sup>[5]</sup>.

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Bone is living tissue that undergoes continuous remodeling through the coordinated action of osteoblasts, osteoclasts and osteocytes. Skeletal integrity is maintained by a balance between bone formation by osteoblasts and bone resorption by osteoclasts [6]. However, aging and menopause disturb this balance, leading to increased bone loss [7]. The decline in estrogen levels after menopause promotes oxidative stress by increasing reactive oxygen species generation and weakening antioxidant defenses. The resulting inflammatory response, characterized by elevated interleukin-6 (IL-6) and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), activates the RANKL signaling pathway, thereby enhancing osteoclastogenesis and bone loss [8].

Osteoporosis and other metabolic bone diseases are caused by oxidative stress and chronic inflammation. Increased production of reactive oxygen species decreases osteoblast formation and increases osteoclast differentiation. Pro-inflammatory cytokines such as IL-6, interleukin-1 $\beta$  (IL-1 $\beta$ ), and TNF- $\alpha$  enhance osteoclastogenesis by stimulating the RANKL/RANK signaling cascade. This mechanism is seen in postmenopausal women, in whom estrogen deficiency increases oxidative stress, inflammatory responses, and bone turnover, resulting in loss of bone mineral density and increased fracture risk [9].

Recent studies have demonstrated that microplastics may adversely affect skeletal health through mechanisms similar to those involved in postmenopausal osteoporosis. *In vitro* animal studies found that exposure to microplastics leads to oxidative stress and inflammation, which in turn causes osteoclastogenesis and changes bone microarchitecture [1]. Certain chemicals associated with microplastics interfere with estrogen signaling, leading to bone remodeling abnormalities [10].

Although microplastics are increasingly recognized as an emerging environmental threat to human health, their impact on health has not been fully understood. There is currently little research involving human beings, particularly postmenopausal women, and the majority of information comes from *in vitro* and animal studies. Therefore, a thorough analysis of the literature is necessary to gain a deeper understanding of how microplastics may impact bone remodeling and skeletal health. The present research on the effects of microplastic exposure on bone health is summarized in

this narrative review, with a focus on the roles of oxidative stress, inflammation, and changes in bone turnover markers as important processes causing skeletal damage caused by microplastics.

## MICROPLASTIC EXPOSURE AND BONE HEALTH

**Microplastic exposure in humans-** Microplastics are ubiquitous environmental contaminants that enter the human body through a variety of exposure pathways. Human exposure is unavoidable due to their wide distribution in the atmosphere, drinking water, and food sources [11]. The human body is exposed to these microplastics through contaminated food, air and water [12].

The main way that humans are exposed to microplastics is through diet. Numerous food products, including seafood, table salt, bottled water, milk, honey, fruits, vegetables, rice and packaged foods, have been found to contain microplastics [13]. Furthermore, repeated use of plastic containers and food packaging, especially when heating or reusing them, may promote the migration of plastic particles into food, increasing human exposure [14].

The human body is exposed to household dust, tire wear particles, and synthetic textile fibers that enter through breathing. Because fibers are constantly shed from clothing, carpets, and furniture, indoor air often has a higher concentration of microplastics than outdoor air. Smaller particles may translocate into systemic circulation after being breathed and settle in various parts of the respiratory system [15,16].

Dermal contact is considered a relatively minor pathway of human exposure to microplastics. Cosmetics, personal hygiene products, and synthetic textiles that come into direct contact with the skin may contain microplastics [12]. Although skin acts as an excellent barrier to microplastic penetration, prolonged exposure or disruption of the skin barrier, such as through cuts or wounds, may make it easier for microplastics to enter underlying tissue.

Nowadays, scientists have developed many techniques for detection of microplastics in biological samples, including Fourier transform infrared (FTIR) spectroscopy, Raman spectroscopy, and pyrolysis gas chromatography-mass spectrometry (PY-GC-MS) [17]. Because of microplastics' ability to cross biological barriers and enter systemic circulation, they are present throughout

the body. Consequently, they have been detected in human blood, placenta, breast milk, lungs, liver, reproductive tissue and vascular tissue<sup>[18]</sup>. These findings raise concern regarding chronic accumulation and its potential effect on multiple organ systems, particularly the skeletal system.

Microplastics are of 2 types: primary and secondary microplastics. Primary microplastics are those formed intentionally, found in cosmetics, face washes, etc. Secondary microplastics are produced due to the degradation of larger particles because of the action of air, heat, water and radiation. Absorption and toxicity of microplastics depend on various factors like their size, shape, polymer composition, surface charge and presence of environmental contaminants. Nanoplastics, because of their smaller size, have greater cellular uptake and tissue penetration than microplastics<sup>[1]</sup>. Experimental studies suggest that these particles induce oxidative stress, inflammation, mitochondrial dysfunction, and endocrine disruption, thereby leading to cellular damage. Long-term effects on humans, particularly on the skeletal system, remain unexplored.

**Bone biology and bone remodeling-** Bone is specialized connective tissue that provides structural support, protects vital organs, helps in locomotion, stores minerals, and serves as a site of RBC production. Bone is metabolically active throughout life; it undergoes continuous remodeling to maintain bone strength, to repair minor bone damage, and to maintain calcium and phosphate homeostasis<sup>[19]</sup>.

There are three types of bones in the human skeleton: compact bone and spongy bone. Compact bones are dense in structure, whereas spongy bones are porous in nature<sup>[19]</sup>.

Osteoprogenitor cells, osteoblasts, osteocytes and osteoclasts are four major types of bone cells. Osteoblasts are bone-forming cells, whereas osteoclasts are bone-resorbing cells. Osteocytes are the most numerous of bone cells, responsible for regulating bone remodeling by coordinating the activities of osteoblasts and osteoclasts. Osteoprogenitor cells are precursors that give rise to osteoblasts<sup>[19,20]</sup>.

Bone remodeling is a lifelong process involving the coordinated removal of old or damaged bone and the formation of new bone, which is essential for maintaining bone strength and skeletal homeostasis<sup>[6]</sup>. It

involves coordinated action of osteoblasts, osteocytes and osteoclasts. The process involves an activation phase followed by bone resorption, reversal bone formation and mineralization. Osteoclasts first remove damaged bone, followed by bone formation and mineralization by osteoblasts<sup>[21]</sup>. A balanced interplay between bone formation and bone resorption is crucial for preserving skeletal integrity and preventing metabolic bone disease<sup>[6]</sup>.

The RANKL/RANK/OPG pathway is a critical regulator of bone remodeling by controlling osteoclastogenesis. Binding of RANKL to the RANK receptor on osteoclast precursor cells triggers their maturation and enhances bone resorptive activity. Osteoprotegerin (OPG) inhibits this process by binding to RANKL and prevents formation of osteoclasts<sup>[22]</sup>. Bone formation is promoted by wnt/ $\beta$  catenin signaling pathway<sup>[23]</sup>. Bone remodeling is regulated by hormones like parathyroid hormone, estrogen, calcitonin and vitamin D. Estrogen suppresses bone resorption and its deficiency after menopause accelerates bone loss<sup>[7,24]</sup>. Bone resorption is stimulated by IL-6 and TNF- $\alpha$ , but oxidative stress disrupts bone remodeling by reducing osteoblast function and accelerating osteoclast function<sup>[25]</sup>.

### **Mechanisms linking microplastic exposure with bone health-**

Following ingestion or inhalation, microplastics can translocate across biological barriers and accumulate in various tissues, where they trigger cellular stress that may interfere with normal bone metabolism<sup>[17]</sup>. Experimental studies showed that microplastics disrupt bone metabolism through several interconnected mechanisms like oxidative stress, chronic inflammation, endocrine and mitochondrial dysfunction<sup>[1]</sup>.

Oxidative stress is considered one of the major mechanisms underlying microplastic-induced skeletal toxicity. Exposure to microplastics promotes the excessive generation of reactive oxygen species (ROS), overwhelming the cellular antioxidant defense system and leading to oxidative damage of lipids, proteins, and DNA<sup>[25]</sup>. Oxidative stress promotes bone resorption and suppresses bone formation.

Microplastics trigger inflammatory responses by activating signaling pathways such as nuclear factor kappa B (NF- $\kappa$ B), which enhances the release of pro-inflammatory cytokines including IL-6, IL-1 $\beta$ , and TNF- $\alpha$ <sup>[26,27]</sup>. These cytokines promote osteoclast differentiation



by upregulating receptor activator of nuclear factor kappa B ligand (RANKL), thereby increasing bone resorption and contributing to bone loss <sup>[22]</sup>.

Another mechanism is endocrine disruption. Microplastics, along with the additives such as bisphenol A and phthalates, can interfere with endocrine hormone signaling pathways involved in bone metabolism <sup>[28]</sup>. Disrupting estrogen signaling matters because it supports bone formation by osteoblasts and suppresses osteoclast activity <sup>[24]</sup>.

Experimental studies show that microplastics impair mitochondrial function, alter calcium homeostasis, and cause osteoblast and osteocyte death, thereby reducing bone formation <sup>[1]</sup>.

**Experimental and clinical studies-** Growing evidence from experimental studies shows that microplastic exposure adversely affects bone health by disrupting bone remodeling and skeletal homeostasis. Although most available studies are in vitro and animal studies, studies in humans are limited <sup>[1,2]</sup>.

**In vitro studies-** Giannandrea *et al.* <sup>[29]</sup> exposed bone cells to nanoplastics and found reduced cell viability, mitochondrial dysfunction, reduced autophagy, and increased oxidative stress, suggesting that nanoplastics directly impair bone-forming cells and contribute to skeletal toxicity.

Yang *et al.* <sup>[30]</sup> found that exposure to polystyrene microplastics in bone marrow mesenchymal stem cells of mice. The study demonstrated that PS-MP increased intracellular rROS- induced mitochondrial dysfunction, suppressed osteogenic differentiation, and reduced expression of osteogenic markers like RUNX2, ALP, and osteocalcin, indicating impaired bone formation.

Pan *et al.* <sup>[31]</sup> exposed osteoblasts to polystyrene nanoplastics and found reduced cell viability, increased apoptosis, activation of the NF-KB inflammatory pathway, and decreased bone formation. These findings suggest that microplastic exposure impairs osteoblast activity through oxidative stress and inflammation.

Mahamud *et al.* <sup>[11]</sup> reviewed molecular and cellular studies and concluded that microplastics induce oxidative stress, inflammation, mitochondrial and DNA damage, and cellular senescence in bone cells, thereby altering bone homeostasis.

Li *et al.* <sup>[32]</sup> reported that 5 µm polystyrene microplastics induced osteocyte cytotoxicity, oxidative stress, DNA damage, cell-cycle arrest, and osteocyte senescence through COX-2 activation. Aspirin treatment significantly reduced microplastic-induced senescence.

**In vivo animal studies-** Pan *et al.* <sup>[31]</sup> demonstrated in mice that chronic exposure to polystyrene microplastics induced osteoporosis-like trabecular bone loss, increased osteoclast formation, and accelerated bone marrow mesenchymal stem cell senescence through activation of the NF-KB pathway. Blocking NF-KB significantly reduced bone loss, confirming the role of inflammation in microplastic-induced skeletal damage.

Liu *et al.* <sup>[33]</sup> investigated polystyrene microplastic exposure in zebrafish and found that it impaired skeletal development, reduced bone mineralization, caused vertebral deformity, and delayed ossification. It was found that oxidative stress and disrupted bone metabolism were the major mechanisms underlying skeletal toxicity.

**Human studies-** Leslie *et al.* <sup>[17]</sup> provided first evidence of microplastics in human blood using pyrolysis-GC-MS. Polyethylene terephthalate, polystyrene, and polyethylene were detected in the blood of healthy adults. This shows that microplastics can enter systemic circulation and potentially reach distant organs, including bone.

Yang *et al.* <sup>[30]</sup> detected microplastics in human skeletal tissue, including bone, cartilage, and intervertebral disc. They found that microplastics reach skeletal tissue through the bloodstream and may interfere with inflammatory cytokines, raising concerns regarding their potential role in bone remodeling and osteoporosis.

Lee *et al.* <sup>[34]</sup> conducted a cross-sectional study involving 365 healthy adults to investigate the association between microplastics in human blood and coagulation markers. Microplastics were detected in 88.9% of blood samples with a mean concentration of 4.2 particles/ ml. Individuals with a higher microplastic burden showed increased activated partial thromboplastin time, C-reactive protein, and fibrinogen levels.

**Table 1:** Biomarkers for microplastic-induced bone toxicity

| Biomarker                                      | Category           | Change after microplastic exposure | Role in bone toxicity           | References                         |
|------------------------------------------------|--------------------|------------------------------------|---------------------------------|------------------------------------|
| SOD (superoxide dismutase)                     | oxidative stress   | Decreased                          | Reduced antioxidant defense     | Pan <i>et al.</i> <sup>[31]</sup>  |
| MDA (melondialdehyde)                          | Lipid peroxidation | Increased                          | Indicates oxidative damage      | Pan <i>et al.</i> <sup>[31]</sup>  |
| Catalase                                       | oxidative stress   | Decreased                          | Impaired ROS scavenging         | Pan <i>et al.</i> <sup>[31]</sup>  |
| IL-6 (interleukin-6)                           | Inflammation       | Increased                          | Promotes osteoclastogenesis     | Malik <i>et al.</i> <sup>[5]</sup> |
| CRP(C-reactive protein)                        | Inflammation       | Increased                          | Marker of systemic inflammation | Wright & Kelly <sup>[2]</sup>      |
| ALP-Alkaline phosphatase                       | Bone formation     | Decreased                          | Reduced osteoblast activity     | Pan <i>et al.</i> <sup>[31]</sup>  |
| PINP (procollagen type 1N-terminal propeptide) | Bone formation     | Decreased                          | Reduced collagen synthesis      | Malik <i>et al.</i> <sup>[5]</sup> |
| CTX (C-terminal telopeptide)                   | Bone resorption    | Increased                          | Increased bone resorption       | Malik <i>et al.</i> <sup>[5]</sup> |
| TRAP (Tartarate Resistant Acid Phosphatase)    | Osteoclast marker  | Increased                          | Increased osteoclast activity   | Pan <i>et al.</i> <sup>[31]</sup>  |

### CURRENT EVIDENCE AND RESEARCH PERSPECTIVES

According to available data, exposure to microplastics may be a new environmental risk factor for poor bone health. According to experimental research, microplastics disrupt normal bone remodeling and skeletal homeostasis by causing oxidative stress, chronic inflammation, mitochondrial dysfunction and endocrine disruption. These biological processes may contribute to osteoporosis, especially in postmenopausal women, by promoting osteoblast malfunction, improving osteoclast differentiation and shifting the balance toward greater bone resorption <sup>[11]</sup>.

One of the mechanisms behind the skeletal toxicity caused by microplastics seems to be OS. After being exposed to microplastics, the body produces more ROS than it can handle, which damages DNA, causes lipid peroxidation, and impairs cellular function. By activating signaling pathways like RANK/RANKL and NF-KB, this promotes osteoclast activity while suppressing osteoblast proliferation and differentiation <sup>[25]</sup>.

Another process that connects microplastic and bone health is inflammation. Increased production of pro-inflammatory cytokines such as IL-6, TNF- $\alpha$ , and IL-1 $\beta$ , which accelerate bone resorption and promote osteoclastogenesis, has been linked to microplastic exposure <sup>[30,31]</sup>.

Human studies demonstrating the connection between microplastic exposure and bone health are still uncommon, despite the abundance of experimental research. The majority of recent results are based on in vitro research, which could not accurately reflect exposure situations in humans. Furthermore, the variation in microplastic size, polymer type, concentration, exposure duration, and detection methods prevents direct comparisons and adds to study variability. Therefore, to gain better knowledge, standardized techniques for microplastic detection and carefully planned human studies are required <sup>[31]</sup>.

Because estrogen deficiency already puts the population at risk for increased bone resorption and osteoporosis, the possible consequences for postmenopausal women. This imbalance is further exaggerated by oxidative stress and inflammation brought on by microplastics, which hasten bone loss and raise the risk of fractures. However, to ascertain if these experimental findings translate into clinically meaningful effects in humans, carefully planned experimental investigations are necessary <sup>[8]</sup>.

The majority of data on microplastic-induced bone toxicity comes from in vitro and animal studies, with very few human investigations <sup>[11]</sup>. The lack of established methods for microplastic detection and quantification in human biological samples limits the repeatability of

present research <sup>[17]</sup>. It is unclear how long-term microplastic exposure affects postmenopausal women's bone turnover, bone mineral density, and fracture risk. Future research should concentrate on carefully designed long-term studies to demonstrate the connection between exposure to microplastics and bone health. Standardized analytical methods for quantifying microplastics in biological samples and through evaluation of oxidative stress, inflammatory, and bone turnover markers are needed to better understand underlying mechanisms and identify reliable biomarkers of skeletal toxicity <sup>[11]</sup>.

### LIMITATIONS

The review is narrative in nature and does not follow the methodology of systematic review or meta-analysis, which may introduce selection bias. Most of the available evidence is based on animal and in vitro studies, with limited data from human studies, especially in post-menopausal women.

### CONCLUSIONS

Exposure to microplastics is becoming more widely acknowledged as a possible environmental risk that could negatively impact bone health. Microplastics can disrupt normal bone homeostasis by inducing oxidative stress, inflammation, reduced osteoblast function, increased osteoclast activity, and dysregulated bone remodeling according to in vitro research, animal models, and a small number of human studies. When taken as a whole, these changes may decrease bone formation, speed up bone resorption, and raise the risk of osteoporosis, especially in susceptible groups like postmenopausal women. However, because of the scarcity of human research and the lack of standardized methods for identifying and measuring microplastics in biological samples, the available data is insufficient to support the direct causative relationship. Therefore, to close the gap in current knowledge, carefully planned human research and the creation of standardized analytical techniques are crucial.

### CONTRIBUTION OF AUTHORS

**Research concept-** Anusha Rani, Bhagyajyothi M Bhat, Rashmi Bhat, Hemalatha Bangera,

**Research design-** Anusha Rani, Rashmi Bhat, Hemalatha Bangera, Edward Lawrence Nazareth

**Supervision-** Anusha Rani, Bhagyajyothi M Bhat, Hemalatha Bangera, Edward Lawrence Nazareth

**Materials-** Anusha Rani, Bhagyajyothi M Bhat, Rashmi Bhat, Edward Lawrence Nazareth

**Data collection-** Bhagyajyothi M Bhat, Rashmi Bhat, Hemalatha Bangera, Edward Lawrence Nazareth

**Data analysis and interpretation-** Bhagyajyothi M Bhat, Rashmi Bhat, Edward Lawrence Nazareth

**Literature search-** Anusha Rani, Bhagyajyothi M Bhat, Rashmi Bhat, Hemalatha Bangera

**Writing article-** Anusha Rani, Bhagyajyothi M Bhat, RaHemalatha Bangera, Edward Lawrence Nazareth

**Critical review-** Bhagyajyothi M Bhat, Rashmi Bhat, Hemalatha Bangera

**Article editing-** Anusha Rani, Hemalatha Bangera, Edward Lawrence Nazareth

**Final approval-** Anusha Rani, Bhagyajyothi M Bhat, Rashmi Bhat, Hemalatha Bangera, Edward Lawrence Nazareth

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