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REVIEW ARTICLE

Microplastics in Musculoskeletal Disorders: An Emerging Threat

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Abstract

Introduction Microplastics (MPs), defined as plastic particles smaller than 5 mm, are emerging environmental contaminants that pose potential health risks. Although their systemic toxicity is increasingly recognized, the impact of MPs on the musculoskeletal system is underexplored.

Materials and methods A narrative literature review was conducted using PubMed, Scopus, and Web of Science. Studies published from 2010 to 2024 were selected using the keywords 'microplastics,' 'nanoplastics,' 'bone,' 'cartilage,' 'muscle,' and 'musculoskeletal system.' Inclusion criteria encompassed original experimental studies, animal models, and clinical data where available.

Results MPs can enter the body via ingestion, inhalation, and dermal absorption. Once inside, they trigger oxidative stress, inflammation, apoptosis, and cellular dysfunction in musculoskeletal tissues. MPs have been associated with disrupted osteoblast and chondrocyte function, increased osteoclastogenesis, and impaired muscle regeneration. Preclinical models indicate reduced bone density and muscle atrophy following chronic exposure. Nanoplastics may have distinct toxicokinetics with deeper tissue penetration and higher bioavailability.

Conclusion Current evidence suggests that MPs adversely affect musculoskeletal health through inflammation, oxidative damage, and cellular dysfunction. The findings, although predominantly preclinical, raise concerns about long-term skeletal and muscular degeneration. Further clinical studies are urgently needed.

Keywords Microplastics · Osteoclastogenesis · Apoptosis · Degeneration

Introduction

Microplastics (MPs) have emerged as pervasive environmental pollutants, infiltrating ecosystems and human tissues alike. These plastic particles, defined as being smaller than 5 mm in diameter, originate from the fragmentation of larger plastic debris and from industrial products such as microbeads and synthetic fibers [1]. Their widespread presence in water, air, and food sources makes them nearly inescapable. The ubiquity of MPs has turned them into a global environmental hazard, raising concerns about their potential effects on human health [2]. MPs enter the human body through multiple pathways, including ingestion (via contaminated food and water), inhalation (through airborne particles), and dermal contact (from personal care products and synthetic textiles). Once inside the body, these particles can translocate to various tissues and organs, where they may exert harmful effects [3].

The human health consequences of MP exposure have received growing scientific attention over the past decade [4]. Initially, most research focused on the impact of MPs on the gastrointestinal, cardiovascular, and respiratory systems. MPs have been shown to disrupt gut microbiota, promote systemic inflammation, and contribute to metabolic disturbances [5, 6]. Inhaled MPs, on the other hand, may trigger respiratory inflammation and contribute to lung disease [7]. Their role in cardiovascular pathology has also been explored, with evidence suggesting that MPs may promote atherosclerosis and vascular damage

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[8]. However, only recently has attention turned toward the potential effects of MPs on the musculoskeletal system, a vital yet often overlooked component of human health [9].

Several mechanisms have been proposed to explain how MPs may contribute to musculoskeletal damage which is chronic inflammation [9]. MPs can provoke immune responses by activating macrophages and triggering the release of pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). Chronic, low-grade inflammation is a known driver of musculoskeletal conditions, including osteoarthritis and rheumatoid arthritis. The presence of MPs in musculoskeletal tissues may amplify inflammatory processes, accelerating tissue degeneration and joint damage [10].

Another proposed mechanism is oxidative stress, which arises when MPs generate reactive oxygen species (ROS) and deplete antioxidant defenses. Elevated ROS levels can lead to oxidative damage of cellular components, including DNA, proteins, and lipids. In bone and cartilage, oxidative stress impairs cellular function and contributes to the pathogenesis of degenerative musculoskeletal diseases. In muscle tissue, oxidative damage can lead to muscle atrophy and impaired repair mechanisms, further exacerbating musculoskeletal decline [11].

MPs interfere with cellular homeostasis by disrupting autophagy and promoting apoptosis (programmed cell death) [12, 13]. Experimental studies demonstrate that MPs accumulate in bone, cartilage, and muscle tissues, where they impair cell viability and hinder tissue repair processes. In bone, MPs alter osteoblast and osteoclast activity, contributing to bone loss and osteoporosis development [14, 15]. In cartilage, MP exposure accelerates matrix degradation by stimulating matrix metalloproteinase (MMP) release, enzymes associated with cartilage breakdown in osteoarthritis [16]. In muscle, MPs interfere with satellite cell function, impairing muscle regeneration and promoting muscle wasting [13, 17].

The bioaccumulation of MPs in musculoskeletal tissues may also have systemic consequences. MPs have been found to contain or absorb harmful chemical additives, including plasticizers, flame retardants, and heavy metals. These substances may leach into surrounding tissues, further exacerbating musculoskeletal toxicity. The prolonged retention of MPs in musculoskeletal tissues may trigger chronic, low-grade inflammation and fibrosis, contributing to the progressive deterioration of musculoskeletal health [18].

In addition to direct tissue damage, MPs may also influence systemic factors that affect musculoskeletal health. For example, MPs have been shown to alter gut microbiota composition and promote metabolic dysregulation, both of which can indirectly impact musculoskeletal homeostasis [8, 19]. Dysbiosis and metabolic inflammation have been linked to bone loss, muscle wasting, and increased susceptibility to MSDs [20].

Although often discussed interchangeably, microplastics and nanoplastics differ significantly in their physicochemical properties. Nanoplastics, defined as plastic particles < 1 μm, have greater bioavailability, surface area, and tissue penetration potential than MPs [15]. Their small size allows them to cross biological barriers more readily, including the blood–brain and placental barriers. Emerging studies indicate that nanoplastics may exert stronger oxidative and genotoxic effects than MPs, potentially amplifying musculoskeletal toxicity [18].

Despite the growing body of evidence highlighting the potential musculoskeletal toxicity of MPs, this area of research remains relatively nascent. Most existing studies are based on animal models and in vitro experiments, with limited clinical data available. Nevertheless, the emerging evidence underscores the need for further investigation into the effects of MPs on musculoskeletal health, particularly given the rising prevalence of both environmental MP pollution and MSDs. This narrative review explores the current evidence regarding the role of MPs in musculoskeletal disorders (MSDs). It examines the mechanisms by which MPs may contribute to musculoskeletal toxicity, including inflammation, oxidative stress, and cellular dysfunction. Furthermore, it summarizes key experimental findings, highlights potential clinical implications, and underscores the need for more comprehensive research in this field. As MPs continue to accumulate in the environment and human body, understanding their impact on musculoskeletal health is essential for developing effective prevention and mitigation strategies.

Literature Search Methodology

A comprehensive narrative literature review was undertaken to evaluate the current evidence on the impact of microplastics (MPs) on the musculoskeletal system. The electronic databases PubMed, Scopus, and Web of Science were systematically searched for relevant studies published between January 1, 2010, and April 30, 2024.

The following keywords and their combinations were used for the search strategy: "microplastics", "nanoplastics", "bone", "cartilage", "skeletal muscle", "musculoskeletal system", "osteoblast", "osteoclast", "chondrocyte", "muscle regeneration", and "degeneration". Boolean operators (AND, OR) were employed to enhance the search sensitivity. Searches were further refined using filters for language (English), study type (original research), and publication dates.

The inclusion criteria were original experimental studies evaluating the effects of MPs or nanoplastics on bone, cartilage, or skeletal muscle, in vivo animal models investigating musculoskeletal toxicity of MPs, in vitro studies on relevant

musculoskeletal cells (e.g., osteoblasts, chondrocytes, myocytes), clinical or human observational data, including biomonitoring studies detecting MPs in musculoskeletal tissues, and articles published in peer-reviewed journals. The exclusion criteria included review articles without new data, conference abstracts, editorials, or commentaries, studies not involving musculoskeletal outcomes, studies focused exclusively on other organ systems (e.g., gastrointestinal, cardiovascular, respiratory), and non-English language publications.

Two authors independently screened titles and abstracts to assess eligibility. Full texts were retrieved for potentially relevant studies. Discrepancies were resolved through discussion and consensus. Relevant findings were synthesized to summarize exposure routes, toxicological mechanisms, and structural or functional changes observed in musculoskeletal tissues following MP exposure.

Microplastics (MPs)

MPs are minuscule plastic particles with diameters smaller than 5 mm. They are a growing environmental concern due to their widespread distribution and persistence in the ecosystem. MPs are classified into two main categories based on their origin: primary MPs and secondary MPs [21].

Primary MPs are intentionally manufactured as small particles for use in commercial and industrial products. These include microbeads**,** microcapsules, and plastic powders. Microbeads are frequently used in personal care products such as facial scrubs, exfoliating creams, toothpaste, and body washes. Their abrasive properties make them popular in cleaning agents and industrial applications, including air-blasting technology used to clean machinery and surfaces. Plastic pellets, also known as nurdles, are another form of primary MPs. These tiny beads serve as raw materials in plastic manufacturing and are often unintentionally released into the environment during production, transportation, or storage [22].

Secondary MPs, on the other hand, result from the fragmentation of larger plastic products due to environmental degradation. Over time, plastic waste—such as discarded bottles, bags, fishing nets, and packaging materials—breaks down into smaller particles through physical, chemical, and biological processes. Environmental factors such as UV radiation**,** temperature fluctuations**,** wave action, and mechanical abrasion accelerate this degradation process. For example, plastic litter in oceans and rivers is subjected to continuous friction and photodegradation, gradually forming secondary MPs. Similarly, plastic debris in soil or landfill sites undergoes fragmentation due to microbial activity and physical weathering, contributing to the formation of MPs in terrestrial environments [23].

The distinction between primary and secondary MPs is important because their environmental fate, persistence, and impact on ecosystems and human health can vary significantly. Primary MPs, being smaller and manufactured for specific uses, often have direct and intentional pathways into the environment. In contrast, secondary MPs are formed through degradation, making them more unpredictable and widespread [24]. Age and sex appear to modulate susceptibility to MP toxicity. Juvenile animal models exposed to MPs show greater impairments in skeletal growth and bone mineralization, suggesting developmental vulnerability during critical growth periods when bone formation rates are highest. Similarly, aged models demonstrate exacerbated oxidative stress and musculoskeletal degeneration compared to younger counterparts, potentially due to declining antioxidant defenses with aging. Sex-based differences in inflammatory and endocrine responses to MPs have also been observed, with female models exhibiting distinct patterns of adipose tissue infiltration and immune activation. These differences may be influenced by hormonal factors, particularly estrogen levels, which could modulate inflammatory responses and bone metabolism in response to MP exposure. Postmenopausal conditions, characterized by estrogen deficiency and increased bone resorption, may represent a particularly vulnerable state for MP-induced musculoskeletal effects. These findings underscore the need for age- and gender-stratified research in future human studies [14].

Sources of MPs

MPs originate from a multitude of sources, making them omnipresent in the environment. Their pervasive presence in air, water, soil, and food chains highlights the challenge of mitigating their spread (Fig. 1) [25–28]. The primary sources of MPs are included in Fig. 1.

[This schematic illustrates the major pathways through which microplastics enter the environment, including primary sources (intentionally manufactured particles from cosmetics, healthcare products, and industrial processes) and secondary sources (fragmentation of larger plastic debris). The diagram shows both terrestrial and aquatic contamination routes, highlighting the ubiquitous nature of microplastic pollution. Abbreviations: PVC = polyvinyl chloride. Schematic created by authors synthesizing findings from experimental studies reviewed in this article]

Environmental Pollution

Environmental pollution is one of the primary contributors to microplastic (MP) contamination. Plastic waste discarded in oceans, rivers, and terrestrial ecosystems gradually breaks down into MPs due to various environmental

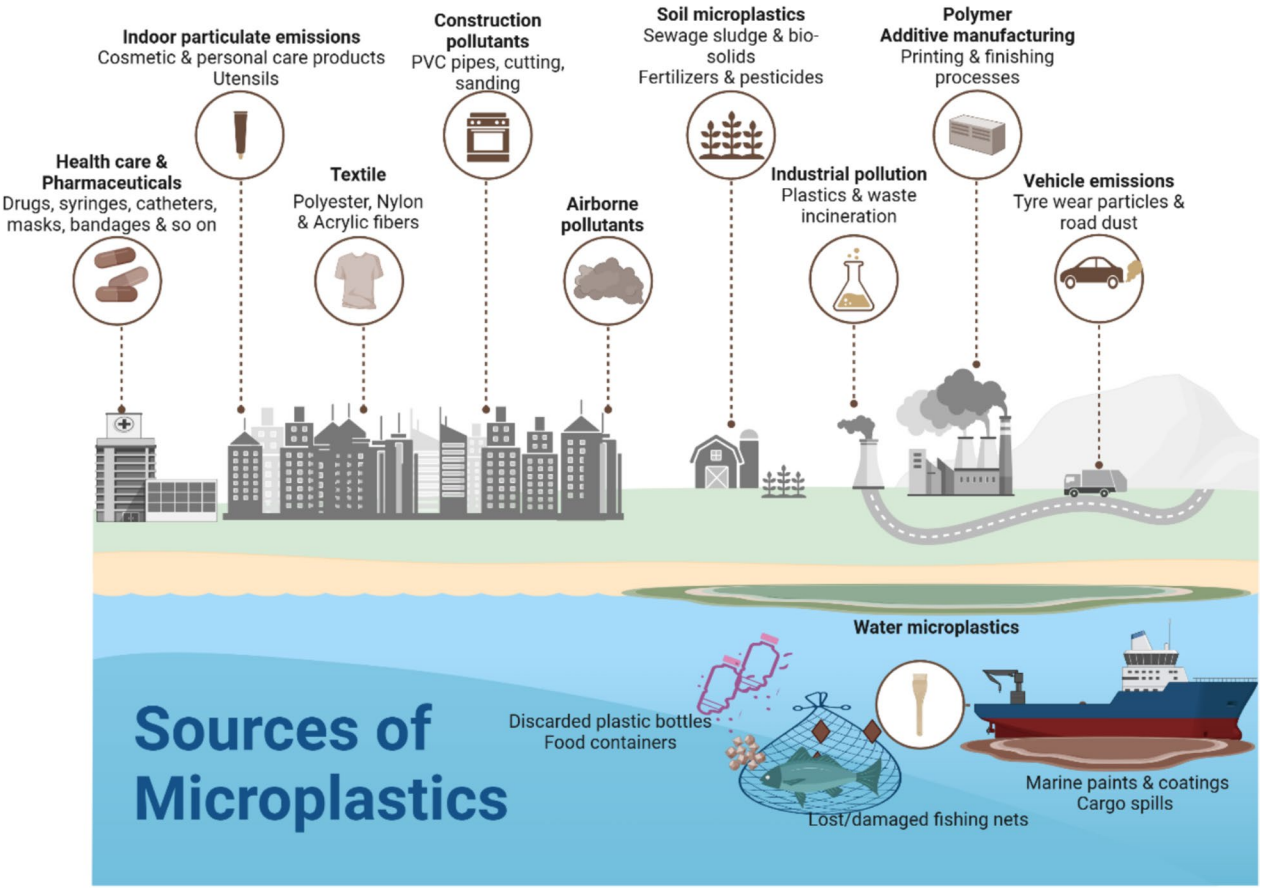


Fig. 1 Sources of microplastics in the environment. (Created with Biorender.com)

factors. Ultraviolet (UV) radiation from sunlight triggers photodegradation, breaking chemical bonds in plastic polymers and causing them to fragment into smaller particles. In aquatic environments, the continuous impact of wave action on plastic debris leads to physical fragmentation, further contributing to MP formation. On land, mechanical stress from wind, rain, and temperature fluctuations accelerates the gradual degradation of plastics into MPs. Additionally, biological degradation plays a role, as certain microorganisms, such as fungi and bacteria, colonize plastic surfaces and release enzymes that promote plastic breakdown, aiding in the formation of MPs.

Aquatic ecosystems, particularly oceans, are considered major reservoirs for MPs. Large plastic debris, including discarded fishing gear, packaging materials, and general waste, undergoes continuous fragmentation over time, adding to the MP load. Rivers also serve as transport channels, carrying MPs from terrestrial sources to larger water bodies. These particles eventually accumulate in marine ecosystems, further increasing their distribution and persistence in the environment [29].

Industrial and Domestic Waste

Industrial activities and domestic waste are major contributors to MP pollution. In industrial settings, plastic manufacturing, processing, and waste disposal generate MPs that enter the environment through wastewater, runoff, and emissions. During plastic production and processing, factories release microplastic particles into the air and water systems. Plastic pellets, also known as nurdles, frequently spill or are improperly handled, leading to environmental contamination. Waste management and recycling plants also contribute to MP pollution. Inefficient filtration systems in recycling facilities result in the discharge of MPs into nearby water bodies. Additionally, wastewater treatment plants are often unable to fully capture MPs, allowing them to flow into rivers and lakes. Synthetic textile fibers are another significant source of MPs. Washing synthetic garments, such as those made from polyester, nylon, and acrylic, releases microfibers—a form of secondary MP—into wastewater. These fibers are too small to be effectively filtered out by conventional treatment systems, contributing to aquatic MP

contamination. Domestic products also play a role in MP pollution. Personal care items containing microbeads, such as facial scrubs and toothpaste, are washed down drains, entering wastewater systems and eventually reaching the environment [30].

Food and Water Contamination

MPs have been detected in a wide range of food and water sources, posing a direct route of human exposure. Seafood is one of the primary sources of MP ingestion. Marine organisms such as fish, shellfish, and crustaceans frequently consume MPs from polluted waters. When these seafood items are harvested and consumed, humans inadvertently ingest the MPs accumulated in their tissues. Common food items like table salt and sugar have also been found to contain MPs. During processing and packaging, MPs can be introduced into the final product, contributing to dietary exposure. Bottled and tap water are additional sources of MP contamination. Research has shown that bottled water often contains higher MP concentrations due to plastic packaging and production processes. Tap water, on the other hand, can become contaminated through poorly filtered water treatment systems, allowing MPs to enter household supplies. Agricultural products, including fruits and vegetables, have also been found to contain MPs. The use of contaminated irrigation water and plastic mulch films in farming practices introduces MPs into the soil. These particles can then be absorbed by plants or deposited on their surfaces, further contributing to human exposure through food consumption [31].

Airborne Exposure

MPs are also present in the air, particularly in urban and industrialized areas, posing a potential risk of respiratory exposure. Several sources contribute to airborne MP contamination. Vehicle tire wear is a major contributor, as tire abrasion from road friction releases tire wear particles (TWPs) containing plastic polymers. These particles are dispersed into the air and can be inhaled by humans. Industrial emissions also play a significant role, with factories and manufacturing plants releasing airborne MPs through exhaust systems. Industrial processes involving plastic cutting, melting, or grinding generate fine MP dust that becomes suspended in the air. Textile fibers are another common source of airborne MPs. Synthetic clothing and upholstery release microfibers during use, particularly in indoor environments. Everyday activities, such as wearing or brushing synthetic fabrics, contribute to indoor MP exposure. Additionally, construction and urban activities generate MPs through the breakdown of plastic materials used in building processes. Road dust, which carries tire particles and plastic debris, further adds to airborne MP contamination. Airborne MPs are particularly concerning due to their potential for respiratory exposure. Inhalation of MP-laden air may lead to the deposition of particles in the lungs, potentially triggering inflammation and respiratory issues, posing a health risk to humans [32].

Pathways of Exposure and Absorption

As illustrated in Fig. 1, humans are continuously exposed to MPs through multiple pathways, including ingestion, inhalation, and dermal contact. These exposure routes enable MPs to enter the body, where they can translocate to various tissues, including musculoskeletal structures. Once inside the body, MPs may accumulate in bones, joints, cartilage, and muscle tissues, where they exert cytotoxic, inflammatory, and degenerative effects. The ability of MPs to infiltrate musculoskeletal tissues has raised significant concerns about their potential role in the pathogenesis of MSDs [33–37]. Recent advancements have enabled the detection of MPs in human biological samples, including blood, placental tissue, stool, and even lung and synovial tissue. Techniques such as Fourier-transform infrared spectroscopy (FTIR), Raman microspectroscopy, and pyrolysis-gas chromatography-mass spectrometry (Py-GC/MS) are commonly used for identification and quantification. However, these methods require further refinement to improve sensitivity and standardization. Despite emerging evidence of MP accumulation in human tissues, biomonitoring efforts remain limited and are not yet incorporated into routine environmental health surveillance [24–26].

Ingestion

Ingestion is one of the most significant and frequent routes of microplastic (MP) exposure for humans. MPs are present in a wide range of food products, drinking water, and processed consumables, making their entry into the human food chain inevitable. Drinking water is a common source of MP ingestion, with both bottled and tap water found to contain MPs. Bottled water often carries a higher concentration due to contamination from plastic packaging and production processes. Seafood and fish are also major contributors, as marine animals such as fish, mussels, and shrimp frequently ingest MPs from polluted waters. When humans consume these seafood products, they indirectly ingest the MPs accumulated in the tissues. Additionally, studies have revealed that common food items like table salt and refined sugar contain MPs, likely due to contamination during processing and packaging. Processed and packaged foods further contribute to MP ingestion, especially when stored in plastic containers. Prolonged contact with packaging material or

heat exposure can cause MPs to leach into the food. Upon ingestion, MPs travel through the gastrointestinal (GI) tract and interact with the intestinal lining. Larger MPs, typically those over 150 µm, remain in the gut lumen, while smaller particles may penetrate the intestinal barrier. Experimental studies indicate that MPs can cross the intestinal epithelium through two primary mechanisms. First, they can be internalized by intestinal epithelial cells via endocytosis, specifically through phagocytosis or pinocytosis. This allows the particles to bypass the gut lining and enter systemic circulation. Second, MPs may pass through the spaces between epithelial cells via paracellular transport, particularly when the intestinal barrier is compromised due to inflammation or disease. Once MPs cross the intestinal barrier, they can enter the lymphatic system and bloodstream, enabling them to circulate throughout the body. Research demonstrates that MPs smaller than 150 µm can infiltrate intestinal tissue and access both the lymphatic and circulatory systems. Through systemic circulation, MPs can reach distant organs and tissues, including musculoskeletal structures such as bones, cartilage, and muscles. Over time, the accumulation of ingested MPs in these tissues may induce oxidative stress, inflammation, and cellular damage, potentially contributing to chronic health issues [38].

Inhalation

Inhalation is another significant pathway of microplastic (MP) exposure, particularly in urban and industrial settings. MPs are present in airborne dust, vehicular emissions, and industrial pollutants, making human inhalation of these particles a daily occurrence. This exposure is especially prevalent in densely populated areas with high traffic pollution and industrial activities. Tire wear particles (TWPs) are a major source of airborne MPs. Friction between tires and road surfaces generates MPs, which are dispersed into the atmosphere and frequently inhaled by individuals in urban environments. Synthetic textile fibers also contribute to airborne MP contamination. Microfibers released from synthetic clothing during wear or washing can become suspended in the air. Indoor environments, particularly homes with carpets, upholstery, and synthetic textiles, often contain significant levels of airborne MPs. Industrial emissions are another key source of inhaled MPs. Factories and processing plants release MPs during plastic cutting, grinding, and melting processes, contributing to atmospheric pollution. Additionally, road dust and urban pollution carry MPs from plastic debris, construction materials, and tire particles, further increasing airborne exposure. Once inhaled, MPs travel through the respiratory tract, and their size determines their depth of penetration. Larger MPs are typically trapped in the upper respiratory tract and expelled through mucociliary clearance. However, smaller MPs—particularly those under 10 µm—can bypass these defenses and reach the alveoli, the small air sacs in the lungs responsible for gas exchange. In the alveoli, MPs can penetrate the alveolar-capillary barrier and enter the systemic circulation, spreading throughout the body. Inhaled MPs can also trigger local inflammation by activating macrophages and promoting the release of pro-inflammatory cytokines, contributing to pulmonary damage. Chronic inhalation of MPs, particularly in industrial settings, may result in systemic inflammation. This inflammation can exacerbate pre-existing MSDs by promoting inflammatory responses in musculoskeletal tissues. Furthermore, airborne MPs that enter the systemic circulation can translocate to distant sites, including bones, cartilage, and muscles, where they accumulate over time, potentially causing tissue damage and contributing to chronic health conditions [39].

Dermal Absorption

Although less common than ingestion and inhalation, dermal absorption of MPs is another potential exposure route, particularly for individuals frequently in contact with plastic-containing products. MPs can be absorbed through the skin, especially when they are small enough to penetrate the stratum corneum, the outermost layer of the epidermis. MPs smaller than 100 nanometers (nanoplastics) have been shown to infiltrate deeper dermal layers, reaching the dermis and potentially entering the circulatory and lymphatic systems.

Dermal exposure to MPs can occur through various sources. Personal care products, such as skincare items containing microbeads or plastic particles, can lead to dermal absorption. Frequent use of these products may increase MP penetration over time. Synthetic textiles are another source of exposure. Prolonged skin contact with synthetic fabrics can facilitate the transfer of MPs through friction and heat exposure, particularly during physical activity. Occupational exposure further increases the risk of dermal MP absorption. Industrial workers handling plastic materials or powders are at greater risk, as skin contact during manufacturing, processing, or cleaning activities can result in MP penetration. Although dermal absorption is less efficient compared to ingestion and inhalation, it still represents a potential route of systemic MP exposure, particularly in occupational settings. Once absorbed through the skin, MPs may reach deeper tissues and eventually enter circulation, contributing to musculoskeletal accumulation. Over time, this accumulation could lead to tissue inflammation and damage, highlighting the need for protective measures in high-risk environments [40].

Systemic Distribution

Once MPs enter the bloodstream, they are capable of systemic translocation to various organs and tissues. Their small size, particularly in the case of nanoplastics, allows them to cross biological barriers, including the blood-brain barrier and the placental barrier. Similarly, MPs can infiltrate musculoskeletal tissues through systemic circulation. Experimental studies have demonstrated the presence of MPs in bone marrow, where they may disrupt hematopoiesis (blood cell formation) and contribute to bone-related disorders. MPs have also been detected in cartilage and joints, where they can accumulate in joint spaces, promoting local inflammation and cartilage degradation. This inflammatory response may accelerate the development of degenerative joint diseases, such as osteoarthritis. Muscle tissues are also susceptible to MP infiltration. Animal studies have revealed the presence of MPs in skeletal muscle, where they induce oxidative stress and disrupt muscle cell homeostasis. MPs have further been shown to impair muscle regeneration and contribute to muscle atrophy. The persistent accumulation of MPs in musculoskeletal tissues may lead to chronic inflammation, oxidative damage, and tissue degeneration. Over time, this exposure could exacerbate or even initiate the development ofMSDs, including osteoporosis, arthritis, and muscle wasting conditions [41].

Cellular and Molecular Mechanisms of MP Toxicity

MPs exposure has been shown to disrupt cellular homeostasis and induce pathological changes at the molecular level through several interconnected mechanisms (Fig. 2). While these mechanisms have been primarily demonstrated in laboratory and animal studies, human evidence remains limited. MPs trigger oxidative stress, chronic inflammation, DNA damage, and mitochondrial dysfunction, all of which contribute to tissue degeneration and impaired musculoskeletal function. These cellular and molecular mechanisms play a crucial role in the progression of MSDs, such as osteoporosis, osteoarthritis, and muscle atrophy (Fig. 2) [42]. Understanding these mechanisms is essential for elucidating the potential long-term health impacts of MPs on the musculoskeletal system.

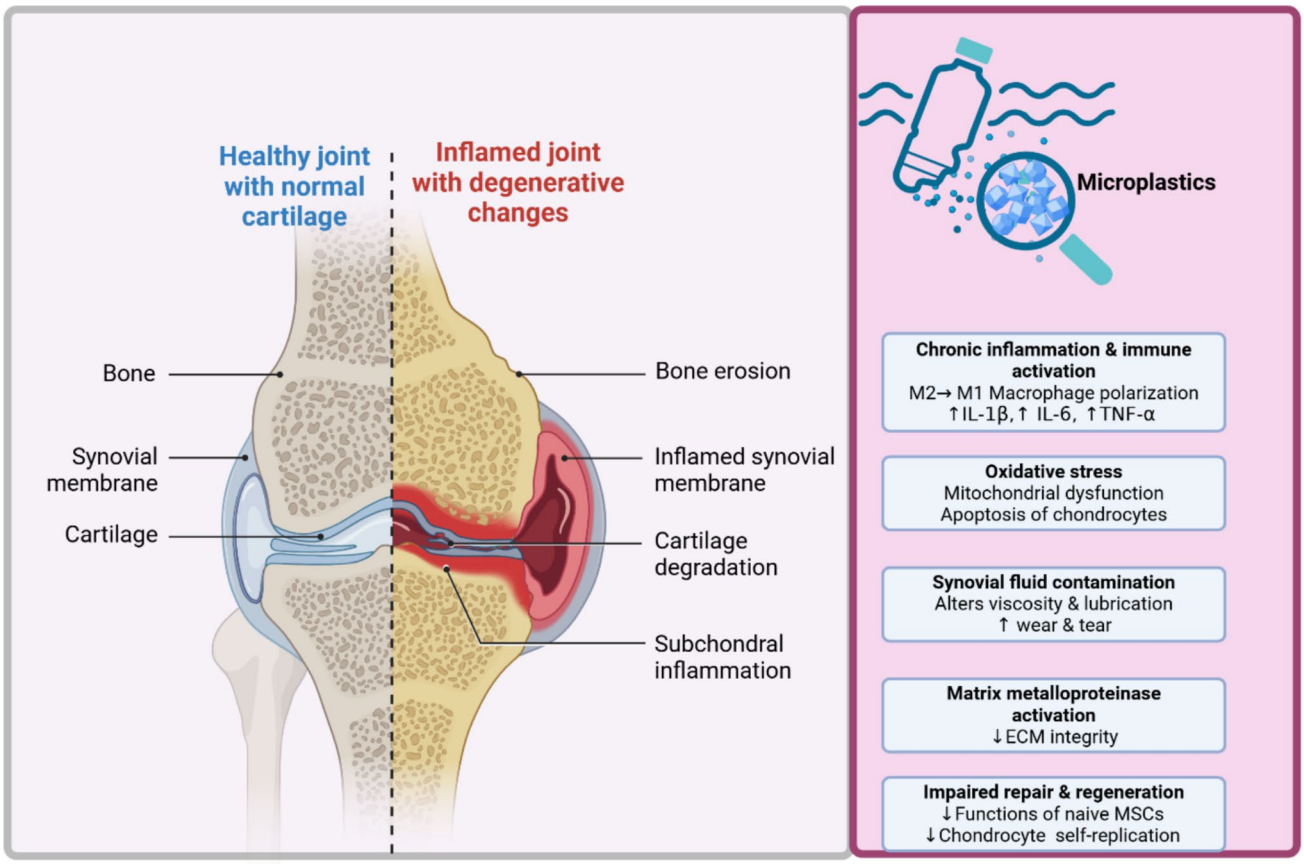


Fig. 2 Mechanisms of microplastic-induced effects in joint pathology. (Created with Biorender.com)

[The diagram compares a healthy joint (left) with an inflamed joint showing degenerative changes (right) following microplastic exposure. The right panel details the molecular mechanisms by which microplastics contribute to joint inflammation and degeneration, including chronic inflammation and immune activation, oxidative stress leading to mitochondrial dysfunction and chondrocyte apoptosis, synovial fluid contamination affecting lubrication, matrix metalloproteinase activation reducing extracellular matrix integrity, and impaired repair and regeneration. Abbreviations: M2→M1 = macrophage polarization from anti-inflammatory to pro-inflammatory phenotype; IL-1β = interleukin-1 beta; IL-6 = interleukin-6; TNF-α = tumor necrosis factor-alpha; ECM = extracellular matrix; MSCs = mesenchymal stem cells. Schematic created by authors based on mechanistic evidence from reviewed literature]

Oxidative Stress and Inflammation

One of the primary mechanisms by which MPs cause cellular damage is by inducing oxidative stress. Upon entering musculoskeletal tissues, MPs stimulate the production of reactive oxygen species (ROS) in bone, cartilage, and muscle cells. ROS, including superoxide radicals, hydrogen peroxide, and hydroxyl radicals, are highly reactive molecules. While low ROS levels play a role in normal cell signaling, excessive production leads to oxidative damage. In bone tissue, elevated ROS levels disrupt the balance between osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells). ROS activate osteoclast differentiation and bone resorption via the RANKL (receptor activator of nuclear factor kappa-B ligand, a key protein regulating bone breakdown) pathway, increasing bone turnover and promoting osteoporosis. Simultaneously, oxidative stress impairs osteoblast proliferation and mineralization, reducing bone integrity [15]. In cartilage, ROS accumulation triggers chondrocyte apoptosis and extracellular matrix (ECM) degradation by activating matrix metalloproteinases (MMPs), which degrade cartilage components such as collagen and aggrecan. This contributes to osteoarthritis progression. In muscle tissue, MPs induce lipid peroxidation and mitochondrial damage, promoting muscle fiber degeneration. ROS trigger myocyte apoptosis and impair muscle function by reducing mitochondrial energy production, causing fatigue and weakness. Furthermore, MPs activate chronic inflammatory pathways, including nuclear factor kappa B (NF-κB) and mitogen-activated protein kinase (MAPK), resulting in the release of pro-inflammatory cytokines (TNF-α, IL-6), exacerbating tissue damage and delaying healing [43].

DNA Damage and Genetic Instability

MPs also promote DNA damage and genetic instability through direct and indirect mechanisms. MPs physically interact with cellular nuclei, causing mechanical stress that results in DNA fragmentation and chromosomal abnormalities. Additionally, ROS-mediated oxidative stress induces DNA strand breaks, base modifications, and chromosomal aberrations, promoting genomic instability [44]. This damage reduces musculoskeletal cell viability by triggering cell cycle arrest and apoptosis, impairing tissue repair capacity. Prolonged MP exposure may also increase the risk of musculoskeletal malignancies, such as bone sarcomas and soft tissue tumors, due to the accumulation of DNA mutations [45]. In bone cells, DNA damage reduces osteoblast proliferation and differentiation, weakening bone formation. In cartilage, it impairs chondrocyte survival, contributing to joint degeneration and osteoarthritis progression. In muscle cells, DNA damage reduces myogenic capacity by impairing satellite cell function, limiting muscle regeneration and promoting muscle atrophy [46].

Mitochondrial Dysfunction

MP exposure disrupts mitochondrial function, impairing cellular energy production. MPs damage the electron transport chain (ETC), reducing adenosine triphosphate (ATP) synthesis, which limits the energy supply necessary for musculoskeletal cell function. Dysfunctional mitochondria also generate excess ROS, creating a self-perpetuating cycle of oxidative stress and cellular damage [47]. MPs trigger mitochondrial apoptotic pathways, promoting cell death. In bone tissue, mitochondrial dysfunction reduces osteoblast differentiation and bone mineralization, contributing to bone loss. In cartilage, mitochondrial impairment decreases chondrocyte metabolic activity, hampering ECM synthesis and repair, accelerating cartilage degeneration. In muscle, MP-induced mitochondrial dysfunction reduces muscle contractility and endurance, promoting atrophy [48].

Cellular Apoptosis and Necrosis

The combined effects of oxidative stress, DNA damage, and mitochondrial dysfunction promote cell death through apoptotic and necrotic pathways. MPs activate caspase-dependent apoptosis, causing musculoskeletal cell death, which contributes to tissue degeneration and impaired healing. In severe cases, chronic MP exposure triggers necrosis, characterized by uncontrolled cell death due to inflammation and oxidative stress, further aggravating tissue damage [49].

Impaired Cellular Signaling and Homeostasis

MPs disrupt essential cellular signaling pathways, affecting musculoskeletal tissue homeostasis. They interfere with the Wnt/β-catenin signaling pathway (a cellular communication system essential for bone cell development), which is critical for bone formation. By inhibiting Wnt signaling, MPs reduce osteoblast differentiation and impair bone regeneration. Additionally, MPs dysregulate autophagy, a cellular process necessary for clearing damaged organelles and maintaining homeostasis [50]. Autophagy impairment contributes to cartilage and muscle cell degeneration, further accelerating tissue damage [51].

Impact of MPs on the Musculoskeletal System

Emerging evidence highlights the concerning effects of MPs on multiple components of the musculoskeletal system, including bone, cartilage, muscle, and associated tissues (Fig. 3). Studies have demonstrated that MPs can enter the bloodstream and infiltrate bone tissue, leading to detrimental changes. For instance, prolonged exposure to MPs in animal models has been shown to alter gene expression in bone marrow cells, potentially impairing hematological function. The accumulation of MPs in bone has also been linked to the disruption of normal bone remodeling processes [52]. Animal experiments have revealed that chronic exposure to MPs slows bone growth and weakens trabecular architecture. Juvenile rats subjected to MP ingestion for 28 days

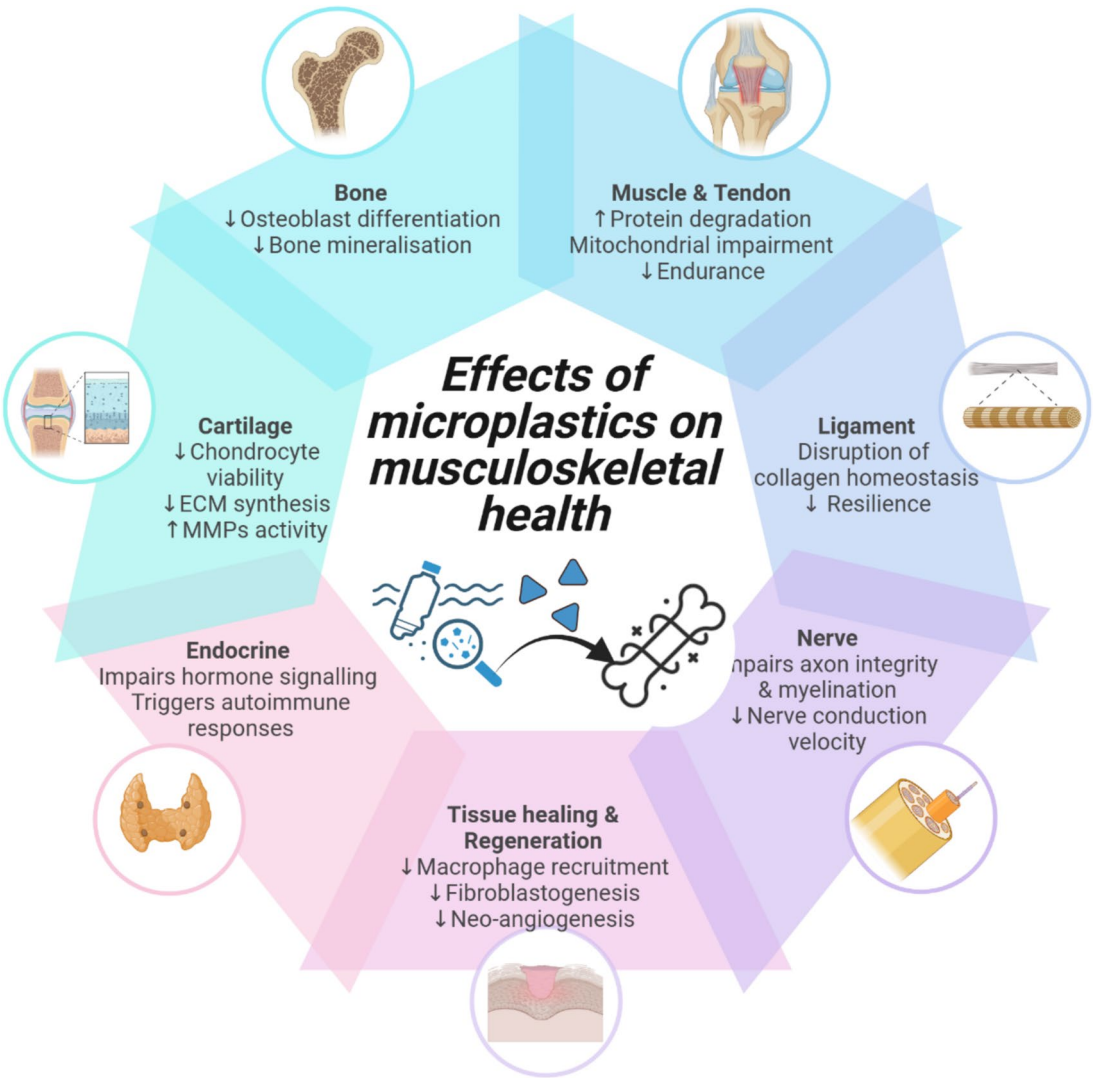


Fig. 3 Comprehensive effects of microplastics on musculoskeletal health. (Created with Biorender.com)

exhibited reduced tibial length and significant thinning of the trabecular bone [53]. Additionally, cartilage damage was observed in the form of a reduced proliferative zone and an increased hypertrophic zone, indicating that MPs may disturb the endochondral ossification process (the normal mechanism by which cartilage is replaced by bone during growth). Such effects could potentially predispose individuals to bone fragility and developmental abnormalities [54].

On a cellular level, in-vitro studies show that MPs and nanoparticles (NPs) induce oxidative stress and inflammation in bone cells. Exposure triggers the production of reactive oxygen species (ROS) and promotes apoptosis in pre-osteoblasts, osteocytes, and pre-osteoclasts. MPs also impair osteoblast migration and disrupt the balance between bone-forming osteoblasts and bone-resorbing osteoclasts. This imbalance could accelerate bone loss and contribute to the pathogenesis of conditions like osteoporosis. Additionally, MPs have been shown to reduce calcium-to-phosphorus (Ca/P) ratios in serum, which is crucial for bone mineralization. The resulting bone resorption and impaired reconstruction may further weaken skeletal integrity [55]. In bone tissue, MP exposure has been associated with increased osteoclastogenesis via RANKL upregulation while concurrently being linked to reduced osteoblast function and mineralization, suggesting disrupted bone remodeling that may contribute to osteoporosis development. Cartilage exposure to MPs has been associated with activation of matrix metalloproteinases (MMPs) and TNF-α-mediated pathways, potentially accelerating extracellular matrix degradation and chondrocyte apoptosis—processes characteristic of osteoarthritis [52, 53].

[This diagram illustrates the multi-system impact of microplastic exposure on various components of the musculoskeletal system. Effects are shown for bone (decreased osteoblast differentiation and bone mineralization), cartilage (reduced chondrocyte viability, decreased ECM synthesis, increased MMP activity), muscle and tendon (increased protein degradation, mitochondrial impairment, decreased endurance), ligament (disruption of collagen homeostasis, reduced resilience), nerve (impaired axon integrity and myelination, decreased nerve conduction velocity), endocrine system (impaired hormone signaling, triggered autoimmune responses), and tissue healing and regeneration (decreased macrophage recruitment, fibroblastogenesis, and neo-angiogenesis). Abbreviations: ECM = extracellular matrix; MMP = matrix metalloproteinase. Schematic created by authors synthesizing findings from experimental studies reviewed in this article]

Muscle tissue is also vulnerable to MP exposure. Although research in this area is still limited and human studies are virtually absent, animal studies suggest that MPs reduce muscle fiber density, impair muscle angiogenesis, and hinder myoblast differentiation. In piglet models, MPs were shown to decrease the redness index and reduce type I muscle fiber density, indicating impaired muscle quality and potential functional decline[56]. Furthermore, MPs have been linked to increased lipid deposition in muscle tissues, which may negatively affect muscle performance and overall strength. In-vitro studies have also demonstrated that MPs reduce myoblast differentiation while promoting apoptosis, suggesting that prolonged exposure could potentially contribute to muscle degeneration and weakness [56]. In muscle tissue, MPs interfere with satellite cell differentiation and mitochondrial function, leading to impaired regeneration and increased atrophy. These mechanisms collectively compromise musculoskeletal integrity [52].

Despite mounting evidence from preclinical models, the effects of MPs on human musculoskeletal tissues remain largely unexplored. In contrast to limited human studies, animal and in vitro research consistently demonstrates potential harmful effects. While some animal studies have demonstrated a link between MP exposure and reduced bone and muscle function, clinical studies on humans are still lacking. Additionally, although MPs have been associated with tumor promotion in other tissues, their potential role in musculoskeletal cancers remains poorly understood[57]. Given the growing evidence of MPs' harmful effects on bone and muscle health, further research is essential to assess their long-term impact on human musculoskeletal function, especially in vulnerable populations such as children. The summary of the clinical studies published in the literature are tabulated in Table 1.

Future Directions

As microplastic contamination becomes increasingly pervasive in the environment, understanding its long-term impact on musculoskeletal health is vital. Future research should prioritize large-scale, human-based studies to confirm the effects observed in animal models. Longitudinal studies investigating chronic MP exposure and its association with bone density loss, muscle degeneration, and cartilage damage are necessary to identify potential clinical implications. Furthermore, specific attention should be given to vulnerable populations, particularly children, whose developing musculoskeletal systems may be more susceptible to MP-induced damage. Since MPs have been detected in human bone marrow and synovium, orthopedic researchers should explore whether chronic MP accumulation contributes to joint disorders, such as osteoarthritis or inflammatory arthritis. Additionally, investigating the potential link between MPs and tendon or ligament degeneration could offer new insights into chronic musculoskeletal conditions, including tendinitis and tendon rupture. In orthopedic surgery, more studies are needed to evaluate the presence of MPs in tissues

Table 1 Summary of the clinical studies of microplastics in musculoskeletal disorders

Author (year)	Model/species	Plastic type and nominal size	Exposure route and duration	Main musculoskeletal end-points	Key limitations
Shengchen et al. [58] (2021)	C2C12 cells + murine TA muscle	PS-MPs 1–10 µm	In-vitro and intramuscular, 14 d	ROS-mediated blockade of myogenesis; myoblast-to-adipocyte shift	High dose; partial in-vivo validation
Najahi et al. [59] (2022)	Human BM-MSCs	PET-MPs < 1–2.6 µm	In-vitro 72 h	↓osteogenic, ↑adipogenic differentiation; senescence shift	In-vitro; PET only
Tarasco et al. [60] (2022)	Zebrafish 30 dpf–90 dpf	PE-MPs 50–100 µm	Contaminated diet, 60 d	Skeletal deformities, ↓bone mineral quality; inter-generational effects	Aquatic model; large particles
Chen et al. [61] (2023)	Broiler chickens	Mixed MPs (0.1–5 µm)	Dietary, 8 wk	Paradoxical muscle hypertrophy but ↓quality and mitochondrial function	Poultry physiology differs from human
Xie et al. [62] (2024)	MRL/lpr mice (auto-immune synovitis)	PS-MPs 0.5–5 mg kg^{-1}	Oral, 4 wk	Synovial pyroptosis (NLRP3/CASP-1/GSDMD); joint fibrosis	SLE background may confound
Zhang et al. [35] (2024)	Juvenile rats	PS-MPs ≈5 µm	Oral gavage, 28 d	Growth-plate disorganisation via ER-stress (↑PERK/ATF4/CHOP)	Short term; female only
Giannandrea et al. [37] (2024)	Murine bone-cell panel (MC3T3-E1, MLO-Y4, RAW264.7)	PS-NPs < 100 nm	In-vitro 24–72 h	ROS, apoptosis, impaired osteoblast migration, ↑osteoclastogenesis	Cell culture model
Yang et al. [56] (2024)	Piglets	PS-MPs 75/150 mg kg^{-1} feed	Dietary, 42 d	↓type I fibre density; impaired angiogenesis (THBS1); poorer meat redness	Livestock model; growth stage specific
Yang et al. [56] (2025)	Human skeletal tissues (bone, cartilage, intervertebral disc; $n = 24$ surgical samples + rat IV validation)	Mixed environmental MPs 25–407 µm (PP 35 %, EVA 30 %, PS 20 %)	Cross-sectional biopsy; rat systemic exposure 4 wk	Highest MP burden in discs > cartilage ≈ bone; elevated serum TNF-α, PINP, TRACP-5b indicating inflammatory bone turnover	Small cohort; observational design limits causality; heterogenous polymers
Pan et al. [14] (2025)	C57BL/6 mice	PS-MPs 1–5 µm	Drinking water, 6 months	Trabecular bone loss; BMSC senescence; NF-κB-driven ↑osteoclastogenesis	Single polymer; supra-environmental dose

collected during procedures, such as bone biopsies or tendon repair surgeries. Assessing the impact of plastic-based implants and surgical materials on musculoskeletal tissues is also crucial, as they may serve as potential MP sources. Moreover, exploring the relationship between MP exposure and musculoskeletal malignancies, including bone and soft tissue sarcomas, could reveal new carcinogenic pathways. Developing strategies to reduce MP exposure through environmental regulations and consumer safety measures is essential, building upon existing policy frameworks for environmental contaminants and following successful models of chemical regulation in consumer products. Public health initiatives aimed at raising awareness about the potential musculoskeletal risks of MPs and promoting policies to limit their presence in consumer products could help mitigate long-term health effects.

The current evidence base is primarily derived from in vitro and animal studies, which, while informative, limit the generalizability to human health. Many experimental models used high concentrations of MPs that may not reflect real-world exposures. Furthermore, methodological inconsistencies in particle type, size, exposure duration, and outcome measures make cross-study comparison challenging. Few studies assessed dose-response relationships or long-term effects. The lack of standardized toxicity assessment frameworks further complicates interpretation. These limitations highlights the need for harmonized protocols and better translational models.

Risk of bias is a key concern in the current literature. Many preclinical studies lacked randomization, blinding, or standardized outcome assessment. Sample sizes were often small, and long-term outcomes were rarely evaluated. Additionally, publication bias toward reporting harmful effects may skew the overall understanding of MP toxicity.

Conclusion

MPs represent an emerging environmental threat with concerning implications for musculoskeletal health. Through interconnected mechanisms of oxidative stress, inflammation, mitochondrial dysfunction, and cellular apoptosis, MP exposure disrupts the delicate balance of bone remodeling, cartilage maintenance, and muscle regeneration—ultimately contributing to conditions such as osteoporosis, osteoarthritis, and muscle atrophy. While current evidence derives primarily from preclinical models, the consistency of findings across multiple tissue types and exposure pathways suggests an urgent need for human health investigation. The global increase in environmental MP contamination, combined with the growing burden of musculoskeletal disorders, creates a compelling case for immediate research prioritization, validated biomonitoring development, and proactive regulatory intervention to protect public health.

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Declarations

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