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Does Environmental Plastic Exposure Act as an Invisible Opponent in Sport Injury Recovery and Athletic Performance?

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Abstract

The global proliferation of plastic materials has led to the widespread presence of micro- and nanoplastics (MNPs) across environmental and biological systems, raising increasing concerns regarding their potential implications for human health. While a growing body of literature has investigated the toxicological effects of MNP exposure, research has largely focused on general populations, with limited attention to physically active individuals. Athletes represent a distinct physiological subgroup characterized by elevated ventilation rates, increased metabolic demands, and frequent interaction with synthetic environments, all of which may influence exposure pathways and biological responses to environmental contaminants.

This article aims to provide a critical synthesis of current evidence on MNP exposure and its potential relevance to exercise physiology and sports medicine. Drawing on data from environmental science, toxicology, and human physiology, we examine the principal exposure pathways (inhalation, ingestion, and dermal contact) and discuss interactions between MNPs and key biological systems involved in athletic performance and recovery, including the respiratory, gastrointestinal, endocrine, nervous, and musculoskeletal systems. Particular attention is given to processes such as oxidative stress, inflammation, endocrine disruption and alterations in the gut microbiome, which may influence exercise adaptation and post-injury recovery.

This work highlights MNP exposure as a potential environmental factor in sports science; however, the relationships between MNP exposure and performance outcomes remain to be established. Clarifying the relevance and the underlying mechanisms of these effects will be essential to define their potential health and performance implications and to guide evidence-based prevention and/or mitigation strategies within athletic environments.

Keywords: Sport, Environment, Plastic, Recovery, Performance, Injury, Exposure, Prevention

Introduction

Plastic is everywhere. Once celebrated as a symbol of modernity and industrial progress, it has become one of the most pervasive and insidious contaminants of our time, infiltrating every living or non-living material across the surface of the Earth and even the stratosphere above it. From the deepest oceanic trenches to the summit of Mount Everest, from Arctic ice cores to human placental tissue, microplastic particles have been detected in virtually every environment studied to date [1]. The sheer omnipresence of these synthetic fragments, defined as plastic particles smaller than 5 mm in diameter, raises a fundamental and urgent question: what does chronic, low-level microplastic exposure mean for human health?

An increasing number of studies are now actively investigating the connection between plastic pollution and human health outcomes. Emerging evidence points to a wide spectrum of potential biological effects, including oxidative stress, endocrine disruption, systemic inflammation, gut microbiome dysbiosis, and cytotoxicity, largely attributed to microplastics themselves as well as to the chemical additives and environmental contaminants they adsorb and carry [2]. While much of the research has traditionally focused on population groups considered inherently vulnerable (children, pregnant women, occupationally exposed workers), athletes must equally be regarded as a population of heightened biological susceptibility. Driven by extreme physiological demands, chronic training loads, and repeated exposure to synthetic environments, athletes present a distinct exposure profile that makes them particularly sensitive to the effects of microplastic contamination. This consideration assumes an additional layer of complexity when extended to paralympic athletes, for whom the concept of health cannot be confined to conventional biomedical parameters. In paralympic sport, health is a dynamic and multidimensional construct: it encompasses the interaction between pre-existing physical impairment, altered neuromusculoskeletal homeostasis, modified immune and autonomic responses and the adaptive capacities that define physiology of those athletes. Microplastic-related stressors may therefore exert qualitatively different and potentially amplified effects in this cohort, whose physiological baselines and recovery trajectories are distinct from those of able-bodied athletes.

In the world of sport science and high-performance medicine, the overarching goal is to help athletes maintain peak physical condition for as long as possible. Health is not merely a desirable attribute: it is the fundamental prerequisite for effective training and competitive performance [3]. An athlete who is physiologically impaired cannot train effectively, cannot recover adequately between sessions, and will inevitably underperform in competition. The margin between winning and losing at elite level is often measured in fractions of a second or centimeters; even subtle, sub-clinical impairments in metabolic efficiency, muscular function, or immune competence could be decisive. In this context, environmental factors capable of undermining biological homeostasis deserve rigorous scientific scrutiny.

Athletes are, paradoxically, among the individuals most likely to be heavily exposed to environmental microplastics [4]. Additionally, athletes rely heavily on plastic-intensive equipment, apparel, and infrastructure: synthetic textiles shed microfibers during physical activity; sports drinks and nutritional products are delivered in plastic packaging; and training environments, from artificial turf to rubberized tracks, continuously shed polymer particles into the air and onto skin. Dermal absorption, ingestion via hand-to-mouth contact, and direct mucosal exposure further may influence the total body burden.

Against this backdrop, the central question concerns how microplastic exposure may influence athletes' health, performance, and injury recovery. The potential accumulation of synthetic particles within lung parenchyma, gastrointestinal mucosa, and systemic circulation may represent an unrecognized environmental variable capable of interacting with key physiological systems involved in athletic function. In this context, microplastic-induced inflammatory responses could theoretically interfere with anabolic adaptations to training. In addition, endocrine-disrupting compounds adsorbed onto plastic particles may affect hormonal signaling pathways involved in musculoskeletal health and recovery processes, while alterations in gut microbiota composition induced by ingestion of microplastics could influence nutrient absorption and energy availability. These considerations are supported primarily by experimental and epidemiological evidence that is still emerging and not yet directly established in athletic populations.

Environmental prevalence and distribution

Plastic pollution has emerged as a major global environmental challenge, with production increasing dramatically over the past decades. It has recently been estimated that more than 400 million tons of plastic are produced worldwide each year, a substantial proportion of which eventually accumulates in terrestrial and aquatic ecosystems [5]

Once released into the environment, plastic debris undergoes relentless degradation through several mechanisms, such as mechanical abrasion, photo-oxidation, ultraviolet radiation and microbial action, leading to the ubiquitous distribution of microplastics across all global strata [5].

Those degradation processes generate smaller particles, commonly classified as microplastics and nanoplastics. Microplastics (MP) are generally defined as plastic particles smaller than 5 mm, while nanoplastics (NP) are typically considered particles with dimensions below 1 μm, although definitions may vary slightly across studies [6-7]. Both originate either from the fragmentation of larger plastic debris or from primary sources intentionally manufactured at small sizes, such as industrial pellets or cosmetic additives. As fragmentation continues, microplastics can further degrade into additional nanoplastics, increasing their mobility in environmental compartments and potentially their biological reactivity. MP and NP (MNPs) can derive from a wide range of polymeric materials, including polyethylene (PE), polypropylene (PP), polystyrene (PS), polyethylene terephthalate (PET), and polyvinyl chloride (PVC), which differ in density, chemical composition, and environmental persistence [8].

Due to their persistence, to their continuous fragmentation processes and to their high resistance to biodegradation, these polymers may persist in the environment for decades or even centuries, progressively fragmenting into smaller particles. Hence, MNPs can disperse widely across environmental compartments, including oceans, freshwater systems, soil, and the atmosphere [9].

In the marine environment, an estimated five trillion plastic particles are floating at the ocean surface, with a total mass of approximately 250,000 tons, while additional large quantities accumulate in coastal sediments and deep-sea environments [10]. Rivers play a critical role in transporting plastic debris from land to the oceans, with global estimates suggesting that between 0.8 and 2.7 million tons of plastic enter marine systems annually through riverine inputs [11]. However, recent evidence indicates that terrestrial environments may represent an even larger reservoir of MP, particularly in agricultural soils receiving wastewater sludge and other plastic-

containing amendments [12]. Furthermore, microplastics have been detected in atmospheric fallout, including in remote mountainous regions, demonstrating their capacity for long-range transport and global dispersal [13].

While airborne microplastics have been detected in both urban and remote environments, higher concentrations are generally reported in densely populated and anthropogenically influenced areas, suggesting that urban outdoor athletes may experience increased inhalation exposure [13-14]. This dispersion cannot be considered static; atmospheric currents and hydrological cycles act as primary vectors, transporting synthetic fibers and fragments far from their industrial sources into remote training environments, including high-altitude regions and open-water circuits (United Nations Environment Programme, UNEP 2021) [15].

Indoor environments have also been identified as significant sources of airborne microfibres, primarily originating from synthetic textiles and materials such as carpets and furnishings; however, specific data on sports facilities and training environments remain limited [16-17]. The potential contribution of synthetic sportswear to direct microplastic exposure during physical activity has been suggested based on known fibre shedding from textiles, although in vivo evidence in athletic settings is currently lacking [18]. Furthermore, individuals engaged in water-based sports, such as swimming or open-water activities, may also be exposed through incidental ingestion of contaminated water; however, direct evidence in those athletes remains scarce. Dermal absorption is currently considered a negligible pathway for MP uptake due to the barrier function of intact skin, while the potential for NP to penetrate biological barriers remains an area of ongoing investigation [19-20]. Prolonged contact with fabrics, equipment, mats, and tools in gyms and on sports fields (including synthetic turf) is a potential source of contamination for athletes.

Consequently, plastic pollution is increasingly recognized not only as an environmental issue but also as an emerging challenge for global public health.

Microplastics exposure pathways and sport-related pattern

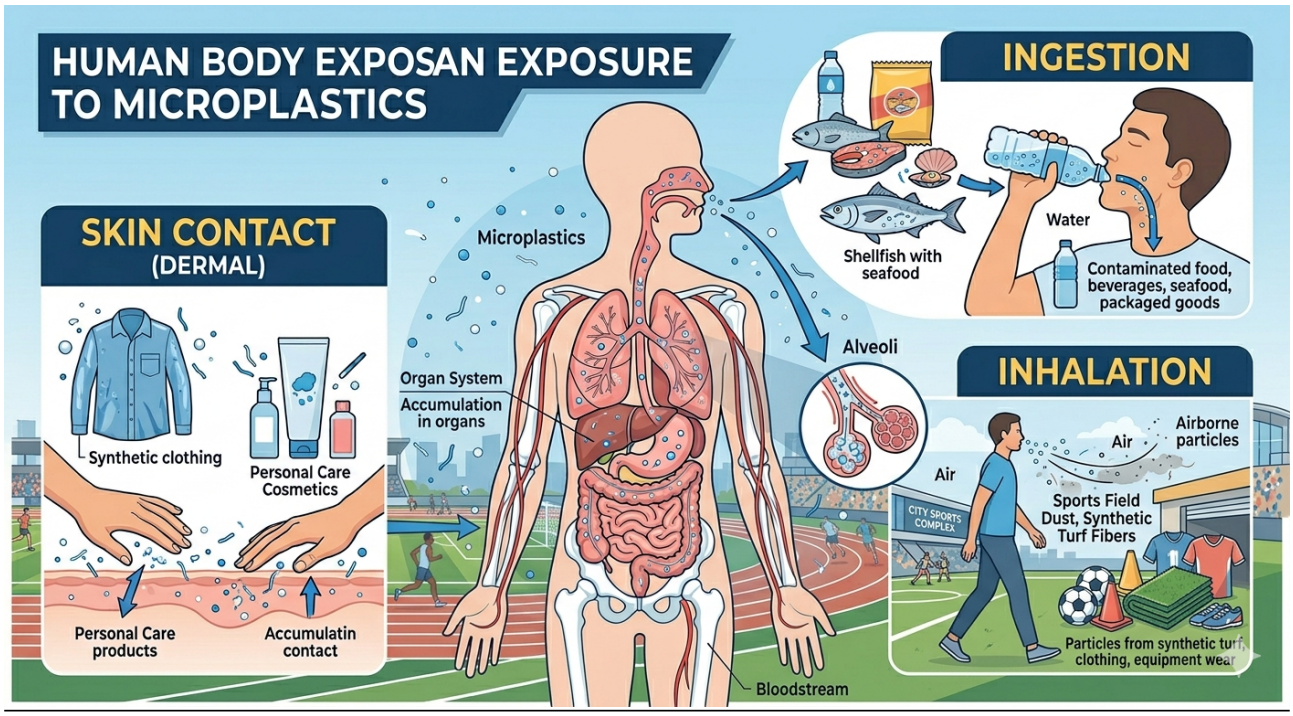

Figure 1: The ways in which microplastics can enter the human body. Mainly include inhalation, ingestion and dermal contact.

Due to the extensive environmental detection of plastic debris, human exposure to microplastics is now considered widespread and ostensibly unavoidable, occurring through multiple pathways including ingestion, inhalation, and, to a lesser extent, dermal contact. MPs have been detected in a wide range of food and beverage items, including seafood, drinking water, and salt [21], suggesting that dietary intake represents a major pathway of exposure, although these estimates are subject to large amounts of variation [22]. A balanced nutrition strategy focusing on plant-based diets, anti-inflammatory foods, and adequate hydration can positively impact athletes' recovery and health both during activity and post-retirement [23].

An additional well-established exposure pathway is ingestion, with microplastics detected in drinking water, bottled beverages, and various food products [24]. Given the increased fluid and caloric intake typical of athletes, this route may be quantitatively relevant, although it has not yet been specifically characterised in physically active populations.

As with all sources of contamination, the risk depends on where athletes train. Training in open water, on sports fields, in swimming pools [25], or in a gym exposes individuals to different risk profiles. Another determinant is whether athletes train indoors or outdoors. In parallel, airborne microplastics have been identified in both indoor and outdoor environments, raising concerns about chronic inhalation, particularly in urban and highly polluted areas. Recent studies have also reported the presence of microplastic particles in human biological samples such as blood, lung tissue, and placenta, indicating their ability to enter and potentially translocate within the human body [26-27-28].

Regarding physically active individuals and athletes, exposure patterns may differ from the general population due to sport-specific behaviours and environmental conditions. Intense physical exertion drives substantially elevated ventilation rates, which significantly increases inhalation of airborne microplastic fibres and particles, particularly in indoor environments such as synthetic-surface training halls, gymnasiums, and swimming pools [29]. During intense exercise, increased ventilation rates can significantly enhance the inhalation of airborne microplastics, particularly in endurance sports such as long-distance running, cycling, and triathlon, where athletes are exposed to large volumes of air over prolonged periods [30]. Consequently, the athlete's environment, whether a metropolitan stadium or a coastal marathon route, becomes a site of significant encounter with these persistent contaminants, facilitating their entry into human systemic circulation.

This widespread diffusion creates a multi-pathway exposure matrix: particles are not only ingested through the contaminated trophic chain [28] but also inhaled as airborne pollutants during periods of high ventilatory demand [27], and acquired through direct skin contact with synthetic clothing, as well as through cosmetics and personal care products, which may include creams, soaps, shampoos, and sunscreens that could contain microplastic particles or plastic-derived chemical additives. Those elements can penetrate the skin barrier, particularly when applied to warm, perspiring skin during or after exercise.

Exposure as a multisystemic stressor in athletes

The combination of massive plastic production, limited recycling, and inherent resistance to biodegradation has fueled widespread environmental contamination by nano- and microplastics. Although the pathophysiological consequences of acute and chronic exposure are not yet fully understood, experimental and toxicological studies suggest that those particles may interact with biological systems through several and interconnected physiological mechanisms.

Both in vitro and in vivo studies have shown that exposure to microplastic particles can increase the production of reactive oxygen species (ROS), potentially leading to mitochondrial dysfunction, lipid peroxidation, and activation of cellular stress pathways [31]. Oxidative stress is associated with activation of inflammatory signaling cascades, including pathways mediated by NF-κB, which can stimulate the release of pro-inflammatory cytokines such as IL-6, IL-1β and TNF-α [32]. These responses suggest also that microplastics may contribute to persistent low-grade inflammatory processes when exposure is chronic.

RESPIRATORY SYSTEM

MNPs can enter the body through inhalation and deposit in the respiratory tract, where they may trigger local inflammatory responses and accumulate in lung tissue. Due to their small size, these particles can reach the lower airways, where they may face limited clearance mechanisms, promoting persistent pulmonary exposure and chronic inflammation [33].

At the cellular level, MNP-induced pulmonary toxicity is largely mediated by oxidative stress and redox imbalance. Experimental studies show that microplastics increase reactive oxygen species (ROS) production, deplete antioxidant defenses, and reduce the activity of key antioxidant enzymes such as GSH-Px, CAT, and SOD in bronchial epithelial cells [34]. In addition, microplastics interact with lung macrophages, epithelial, and stromal cells, inducing pro-inflammatory cytokine release. Those mechanisms are linked to DNA damage, cellular senescence, and fibrotic remodeling and respiratory diseases including asthma, COPD, pulmonary fibrosis, and lung cancer [35-36].

Furthermore, respiratory impairment associated with MNPs exposure may also affect physical performance. A recent study shows that microplastic exposure may reduce the efficiency of oxygen uptake and utilization during exercise, directly affecting exercise endurance. Animal models showed

significant decreases in maximum speeds and endurance levels in exposed organisms.

Endurance athletes exposed to airborne microplastics may experience reduced lung capacity and increasing fatigue-related injuries. Contact sport athletes face elevated microplastic exposure from turf infill materials, compounding injury risks (e.g., ACL injuries). A compromised muscle function and respiratory efficiency may exacerbate injury susceptibility, athletic performance, and potentially adverse effects on cognitive performance through the pathway between the lungs and the brain (lung-brain axis).

NERVOUS SYSTEM

Plastic debris, particularly nanoplastics, can reach the central nervous system (CNS) after crossing biological barriers such as the blood–brain barrier (BBB), the nasal olfactory epithelium, or via the gut–brain axis [37]. Once in the brain, these particles can accumulate in neural tissue and induce multiple neurotoxic processes including BBB disruption, oxidative stress, mitochondrial dysfunction, and neuroinflammation [38].

NP are poorly degraded by lysosomal enzymes: they can accumulate intracellularly and impair autophagic flux, leading to lysosomal damage, endoplasmic reticulum stress, and increased oxidative stress [39]. Emerging evidence suggests that inhaled microplastics may influence cognitive performance through the lung–brain axis, promoting neuroinflammation and cognitive impairment in experimental models [40]. These combined respiratory and neurological effects may ultimately compromise endurance, reaction time, and decision-making in physically demanding activities.

Indirect neurotoxicity may also occur through the microbiota–gut–brain axis: microplastic-induced intestinal barrier disruption can increase systemic inflammatory mediators (e.g., LPS, IL-1β, TNF-α), further amplifying neuroinflammation.

GASTROINTESTINAL SYSTEM - MICROBIOTA-GUT-BRAIN AXIS

The gastrointestinal tract represents one of the primary interfaces of exposure, as ingestion through contaminated food, water, and food-contact materials constitutes a major pathway of human exposure. Data from experimental models have shown that ingested MP may interact with intestinal epithelial cells, potentially affecting gut barrier integrity and promoting alterations in gut microbiota composition [41], increasing intestinal permeability, inducing oxidative stress and inflammation, and altering metabolic processes with downstream systemic effects [42].

At structural level, MP can damage intestinal villi, reduce mucus secretion, and impair epithelial integrity. Reduced expression of tight junction proteins such as ZO-1, occludin, and claudin-3 compromises the intestinal barrier, facilitating inflammation and microbial imbalance [43].

One of the most significant consequences of MNPs exposure is dysbiosis. Studies report reductions in beneficial genera such as Lactobacillus and Bifidobacterium and increases in potentially pathogenic species including Escherichia coli and Clostridium difficile. In addition, MP can host microbial communities which may act as reservoirs for pathogens and antimicrobial resistance genes [44]. The dysbiosis can alter metabolic pathways involved in lipid metabolism and immune regulation. Increased intestinal permeability associated with microbiota disruption may elevate circulating lipopolysaccharide (LPS), promoting systemic inflammatory responses that affect multiple organs [45].

These alterations are particularly relevant for athletes, as the gut microbiota plays an important role in exercise metabolism and performance. Physically active individuals typically show greater microbial diversity and enrichment of taxa such as Akkermansia muciniphila and Veillonella, which are associated with improved metabolic efficiency and endurance capacity [46]. Gut bacteria also produce short-chain fatty acids (SCFAs), including butyrate and propionate, which support intestinal health, energy metabolism, and aerobic performance [47].

INTEGUMENTARY SYSTEM

Skin represents an effective barrier against the penetration of external agents into the human body. Recent toxicological evidence indicates that the penetration of these particles is a complex process governed by their physicochemical properties, including size, morphology, and surface chemistry [48]. While the stratum corneum remains largely impermeable to larger fragments, nanoparticles at the lower end of the scale can bypass this architectural defense, potentially acting as vectors for adsorbed hazardous molecules or environmental contaminants [48].

Dermal exposure to MNPs is ubiquitous, occurring through contact with indoor dust, synthetic textile microfibers, and polymer-based personal care products [49]. The efficiency of dermal absorption is highly dependent on barrier integrity. While healthy skin offers significant resistance, conditions characterized by barrier dysregulation, such as inflammatory disorders, significantly lower the threshold for particle entry [49]. Furthermore, the chronic presence of MNPs may directly impair skin homeostasis, inducing a localized state of oxidative stress and systemic signaling disruption [50].

For athletes, dermal exposure is intensified by sport-specific physiological stressors. The combination of prolonged mechanical friction, hyperhydration of the stratum corneum due to sweat, and minor epidermal micro-lesions can transiently increase skin permeability [50]. Once internalized, MNPs interact with the skin's immune-competent cells, including keratinocytes and dendritic cells, triggering pro-inflammatory cascades, lipid peroxidation, and alterations in autophagic flux [49-50]. These mechanisms not only predispose the athlete to premature skin aging but also represent an overlooked contributor to the cumulative systemic inflammatory burden [51]. Prolonged physical

activity may enhance the condition of dermal uptake of microplastics.

ENDOCRINE SYSTEM

Chronic plastics exposure may disrupt hormonal homeostasis across multiple endocrine axes, potentially impairing metabolism, recovery capacity, and anabolic signaling essential for athletic adaptation and performance. The endocrine system is a major physiological target of MNPs exposure: these particles can contain endocrine-disrupting chemicals (EDCs) capable of binding to hormone receptors and interfering with hormonal signaling across multiple organs, including the hypothalamus, pituitary, thyroid, and gonads. Evidence indicates that MNPs exposure can disrupt

different key regulatory axes, with consequences for reproductive, metabolic, and immune function: the hypothalamic–pituitary–gonadal (HPG), hypothalamic–pituitary–thyroid (HPT), and hypothalamic–pituitary–adrenal (HPA) system [52].

At the molecular level, microplastics can act as carriers for chemical additives such as bisphenols, phthalates, polybrominated diphenyl ethers, polychlorinated biphenyls, and perfluorinated compounds. These substances may leach from the plastic matrix after entering the body and interfere with endocrine receptors and hormone regulation [53]. The pituitary gland is particularly susceptible, as EDC-mediated hypothalamic disruption may alter the release of key hormones including GnRH, LH, and FSH, ultimately affecting gonadal function [52].

Experimental studies report that chronic microplastic exposure can impair the HPG axis and reduce testosterone production. In animal models, polystyrene and polyethylene microplastics have been associated with testicular damage, altered spermatogenesis, and decreased circulating levels of testosterone, LH, and FSH [54-55]. Such hormonal disruption is particularly relevant for athletes, as testosterone plays a central role in muscle growth, bone strength, metabolism, and recovery [56].

MNPs exposure may also affect thyroid and stress-response regulation. Disruption of the HPT axis can alter thyroid hormone levels and metabolic rate, while alterations in the HPA axis, including changes in cortisol and ACTH levels, may influence stress adaptation and recovery processes [57]. Since the cortisol-to-testosterone ratio is commonly used as a biomarker of anabolic–catabolic balance in sport, endocrine disruption could negatively influence training adaptation and performance.

Athletes may experience additional exposure through synthetic environments. Tight synthetic sportswear and artificial turf can increase contact with microplastics and associated EDCs, potentially facilitating dermal absorption through sweat glands, hair follicles, or minor skin lesions during prolonged physical activity.

Beyond the systemic impacts of MP on multisystemic homeostasis, an additional and potentially underexplored dimension in exercise physiology concerns the role of plastic-associated chemical additives in modulating endocrine pathways, relevant to post-exercise recovery. In particular, growing toxicological evidence suggests that exposure to certain phthalates may interfere with steroidogenesis by downregulating the expression of steroidogenic acute regulatory (StAR) protein and key cytochrome P450 enzymes, processes that have been associated with reduced circulating testosterone levels in experimental and epidemiological studies [58].

These findings are highly relevant in the context of MPs as vectors and reservoirs of endocrine-disrupting chemicals. Such endocrine-disrupting activity may extend beyond reproductive physiology, potentially influencing anabolic signaling pathways involved in skeletal muscle adaptation and repair. Androgens play a well-established role in modulating muscle protein synthesis and satellite cell activation, both of which are critical processes in the recovery from exercise-induced muscle damage.

Accordingly, disruption of steroidogenic pathways may impair the hormonal environment required for optimal musculoskeletal recovery. Rather than representing a direct causal effect, this mechanism may contribute to a broader endocrine–metabolic perturbation that could influence the kinetics of tissue repair, adaptation to training loads, and resilience to repetitive mechanical stress.

Within this framework, microplastic exposure, and its associated chemical payload, may be considered a potential environmental modifier of recovery capacity and injury susceptibility in athletes, although direct evidence in athletic populations remains currently to investigate.

MUSCULOSKELETAL SYSTEM

At the muscular level, limited data impedes a full understanding of the internal exposure to microplastics, especially concerning the musculoskeletal system.

Microplastics have been shown to cause abnormal endochondral ossification and disrupt the normal function of pre-osteoblasts, osteocyte-like cells, and pre-osteoclasts through gene mutations, endoplasmic reticulum stress induction, and reduced autophagosome formation in bone growth areas. Although there are few reports on their effects on muscle, it has been noted that microplastics inhibit energy and lipid metabolism, decrease type I muscle fiber density, impair muscle angiogenesis, cause muscle atrophy, and increase lipid deposition. [59]

Within the musculoskeletal system, MNPs can trigger the chronic production of reactive oxygen species (ROS), fostering a persistent low-grade inflammatory state that overlaps with and amplifies the acute post-exercise inflammatory response. This toxicological synergy may saturate cellular

antioxidant defences and disrupt androgen-dependent signalling pathways critical for muscle protein synthesis and ultrastructural repair. The disruption of testosterone signalling is particularly relevant in this context: MNP-induced suppression of the HPG axis reduces the anabolic hormonal environment on which skeletal muscle adaptation fundamentally depends, compounding the direct oxidative and inflammatory damage at the tissue level. [60]

Over time, this condition could manifest as symptoms commonly associated with overtraining syndrome: persistent fatigue, decreased performance, prolonged soreness, and heightened injury risk. Thus, the musculoskeletal system could suffer a progressive vulnerability to strains, tears, and delayed healing processes, undermining both the athlete's resilience and their longevity in sport.

Discussion

Plastics exposure may represent an emerging environmental factor with potential relevance for injury and recovery in athletes. Current evidence indicates that plastic particles can enter the human body through multiple pathways, primarily inhalation and ingestion. Recent studies suggest that a fraction of these particles may subsequently translocate across biological barriers and reach systemic circulation. Once bioavailable, these particles sequester within diverse organ systems, triggering effects that may operate through synergistic or cumulative mechanisms, potentially contributing to compromise homeostatic balance. Rather than targeting a single organ system, MNP-related effects appear to involve multiple interconnected pathways, although the extent to which such mechanisms translate into clinically meaningful outcomes in humans is still unclear.

Athletes represent a distinct group of population characterized by high physiological demands and specific environmental exposure patterns. Higher ventilatory rates during exercise may increase the inhalation of airborne particles; prolonged training sessions in synthetic-turf environments, indoor facilities, and contaminated water sources may influence ingestion and dermal exposure. The widespread use of synthetic materials in sportswear and equipment may further increase environmental contact with microplastics and associated chemicals.

Furthermore, the biological impact of MNP exposure may not be uniform across athletic populations and could vary depending on sport-specific demands, environmental conditions, and dominant physiological systems involved. Experimental studies in animal and model systems have reported alterations in endurance capacity and post-exercise metabolic responses following microplastic exposure, although the applicability of these findings to humans remains uncertain. Different sport disciplines may be differentially affected. For example, in endurance-based activities, where performance is tightly coupled to respiratory efficiency and mitochondrial function, MNP-related pulmonary inflammation or disruptions in oxidative metabolism could increase the energetic cost of exercise and impair aerobic capacity. Similarly, in sports requiring high levels of cognitive function and sensorimotor integration, potential neuroinflammatory or neurotoxic effects observed in experimental models may represent a relevant pathway of interaction.

Athletes engaged in aquatic environments may present a particularly complex exposure profile. Microplastics have been detected in surface waters used for recreational and competitive activities, and incidental ingestion during swimming or open-water exercise has been documented. Indoor swimming environments may introduce additional factors, including airborne by-products and the presence of microplastic-associated microbial communities, which could interact with respiratory exposure pathways. However, although current evidence is insufficient, the physically active population may accordingly exhibit a heightened susceptibility to bioaccumulation compared to sedentary individuals.

From a physiological perspective, it has been hypothesized that MNP exposure could interact with processes central to performance, recovery and tissue repair. Recent studies indicate potential effects on mitochondrial function, inflammatory signaling, endocrine regulation, and gut microbiota composition. Such alterations could influence aerobic efficiency, muscle adaptation, immune function, and recovery dynamics. However, these relationships remain to be investigated. Given these uncertainties, MNP exposure should be considered a plausible, but as yet insufficiently characterized, contributor to environmental load in athletes rather than an established determinant of performance or injury risk.

From a preventive perspective, precautionary strategies may be considered. At the individual level, reducing avoidable sources of plastic exposure, such as minimizing the use of plastic food containers, avoiding heating food in plastic, and favoring alternative materials when feasible, may represent pragmatic measures. At the institutional level, improving indoor air quality, optimizing ventilation, and evaluating materials used in sports infrastructure could contribute to reducing exposure. Such measures should be proportionate to the current level of evidence and integrated within broader environmental and public health strategies.

At the regulatory level, recent developments suggest increasing awareness of material-related exposures in sports environments. Updated infrastructure frameworks from UEFA acknowledge the coexistence of natural, artificial, and hybrid playing surfaces, potentially opening the way to future evidence-based decisions that incorporate both performance and health considerations.

Abbreviations

MP : microplastics NP : nanoplastics
MNPs : micro and nano plastics

COPD : Chronic obstructive pulmonary disease HPA : Hypothalamic-Pituitary-Adrenal axis GSH-Px : Glutathione Peroxidase
CAT : catalasi
SOD : superoxide dismutase SCFA : short chain fatty acids

Conflicts of interest

The authors declare that they have no conflicts of interest.

References

1. Mercola J. From Bakelite to Biohazard: The Century-Long Rise of Microplastics. Cureus. 2025 Dec 19;17(12):e99627. doi: 10.7759/cureus.99627. PMID: 41555975; PMCID: PMC12812305.
2. Chulkov V, Gasanov M, Isakov V, Denisenko A, Nwosu C, Rodkin S. Molecular and Cellular Effects of Microplastics and Nanoplastics in the Pathogenesis of Cardiovascular, Nervous, Urinary, Digestive, and Reproductive System Diseases: A Global Systematic Review. Int J Mol Sci. 2025 Nov 19;26(22):11194. doi: 10.3390/ijms262211194. PMID: 41303677; PMCID: PMC12653346.
3. Drew MK, Toohey LA, Smith M, Baugh CM, Carter H, McPhail SM, Jacobsson J, Timpka T, Appaneal R. Health Systems in High-Performance Sport: Key Functions to Protect Health and Optimize Performance in Elite Athletes. Sports Med. 2023 Aug;53(8):1479-1489. doi: 10.1007/s40279-023-01855-8. Epub 2023 Jun 7. PMID: 37285068; PMCID: PMC10356621
4. Jiao X, Cao Q, Deng Z. Microplastics and exercise: impacts on performance and physiological health. Front Sports Act Living. 2025 Aug 19;7:1611255. doi: 10.3389/fspor.2025.1611255. PMID: 40904807; PMCID: PMC12403993.
5. Geyer R, Jambeck JR, Law KL. Production, use, and fate of all plastics ever made. Sci Adv. 2017 Jul 19;3(7):e1700782. doi: 10.1126/sciadv.1700782. PMID: 28776036; PMCID: PMC5517107.
6. Gigault J., Halle A.T., Baudrimont M., et al. (2018). Current opinion: What is a nanoplastic? Environmental Pollution, 235:1030–1034
7. Hartmann N.B., Hüffer T., Thompson R.C., et al. (2019). Are we speaking the same language? Recommendations for a definition and categorization framework for plastic debris. Environmental Science & Technology, 53(3):1039–1047.
8. Andrady A.L. (2011). Microplastics in the marine environment. Marine Pollution Bulletin, 62(8):1596–1605.
9. Wright S.L., Kelly F.J. (2017). Plastic and human health: A micro issue? Environmental Science & Technology, 51(12):6634-6647.
10. Eriksen M., Lebreton L.C.M., Carson H.S., et al. (2014). Plastic pollution in the world's oceans: More than 5 trillion plastic pieces weighing over 250,000 tons afloat at sea. PLoS ONE, 9:e111913.
11. Lebreton L.C.M., van der Zwet J., Damsteeg J.W., Slat B., Andrady A., Reisser J. (2017). River plastic emissions to the world's oceans. Nature Communications, 8:15611.
12. Nizzetto L., Futter M., Langaas S. (2016). Are agricultural soils dumps for microplastics of urban origin? Environmental Science & Technology, 50:10777–10779.
13. Allen S., Allen D., Moss K., Le Roux G., Phoenix V.R., Sonke J.E. (2019). Atmospheric transport and deposition of microplastics in a remote mountain catchment. Nature Geoscience, 12:339–344.
14. Dris R., Gasperi J., Saad M., Mirande C., Tassin B. (2016). Synthetic fibers in atmospheric fallout: A source of microplastics in the environment? Environmental Science & Technology, 50(12):654–660.
15. UNEP (2021). From Pollution to Solution: A Global Assessment of Marine Litter and Plastic Pollution. United Nations Environment Programme.
16. Dris R., Gasperi J., Mirande C., Mandin C., Guerrouache M., Langlois V., Tassin B. (2017). A first overview of textile fibers, including microplastics, in indoor and outdoor environments. Environmental Pollution, 221:453–458.
17. Prata J.C. (2018). Airborne microplastics: Consequences to human health? Environmental Pollution, 234:115–126.
18. Hernandez E., Nowack B., Mitrano D.M. (2017). Polyester textiles as a source of microplastics from households: A mechanistic study to understand microfiber release during washing. Environmental Science & Technology, 51:7036–7046.
19. EFSA Panel on Contaminants in the Food Chain (CONTAM) (2021). Presence of microplastics and nanoplastics in food, with particular focus on seafood. EFSA Journal, 19(6):e06479.
20. SAPEA (Science Advice for Policy by European Academies) (2019). A Scientific Perspective on Microplastics in Nature and Society.
21. Walker-Franklin I, Villalobos Santeli A, Neal-Walthall N. Tracking Microplastics From Source to Impact: A Review of Environmental Presence, Exposure, Remediation, and Health Risks. Curr Environ Health Rep. 2026 Mar 18;13(1):12. doi: 10.1007/s40572-026-00531-z. PMID: 41844949; PMCID: PMC12995963.
22. Cox K.D., Covernton G.A., Davies H.L., et al. (2019). Human consumption of microplastics. Environmental Science & Technology, 53:7068–7074.
23. Del Grosso F, Turco D. Nutrition in sport: an opportunity to balance performance, sustainability and Preventive Medicine. Res Sports Med. 2026 Jan-Feb;34(1):61-70. doi: 10.1080/15438627.2025.2525854. Epub 2025 Jul 1. PMID: 40590526.
24. World Health Organization (2019). Microplastics in drinking-water. Geneva ISBN: 978-92-4-151619-8
25. Fátima Felgueiras, Zenaida Mourão, Catarina Morais, Hugo Santos, Marta Fonseca Gabriel, Eduardo de Oliveira Fernandes. Comprehensive assessment of the indoor air quality in a chlorinated Olympic-size swimming pool. Environment International. Volume 136, March 2020, 105401 - https://doi.org/10.1016/j.envint.2019.105401
26. Leslie H.A., van Velzen M.J.M., Brandsma S.H., et al. (2022). Discovery and quantification of plastic particle

pollution in human blood. Environment International, 163:107199.
27. Jenner L.C., Rotchell J.M., Bennett R.T., Cowen M., Tentzeris V., Sadofsky L.R. (2022). Detection of microplastics in human lung tissue using μFTIR spectroscopy. Science of the Total Environment, 831:154907.
28. Ragusa A., Svelato A., Santacroce C., et al. (2021). Plasticenta: First evidence of microplastics in human placenta. Environment International, 146:106274.
29. Maurizi L, Simon-Sánchez L, Vianello A, Nielsen AH, Vollertsen J. Every breath you take: High concentration of breathable microplastics in indoor environments. Chemosphere. 2024 Aug;361:142553. doi: 10.1016/j.chemosphere.2024.142553. Epub 2024 Jun 6. PMID: 38851509.
30. Tainio M., et al. (2021). Air pollution, physical activity and health: A mapping review of the evidence. Environment International, 147:105954.
31. Yong CQY, Valiyaveettil S, Tang BL. Toxicity of Microplastics and Nanoplastics in Mammalian Systems. Int J Environ Res Public Health. 2020 Feb 26;17(5):1509. doi: 10.3390/ijerph17051509. PMID: 32111046; PMCID: PMC7084551.
32. Hirt N., Body-Malapel M. (2020). Immunotoxicity and intestinal effects of nano- and microplastics: A review of the literature. Particle and Fibre Toxicology, 17:57.
33. Saha SC, Saha G. Effect of microplastics deposition on human lung airways: A review with computational benefits and challenges. Heliyon. 2024 Jan 11;10(2):e24355. doi: 10.1016/ j.heliyon.2024.e24355. PMID: 38293398; PMCID: PMC10826726.
34. Lu K, Zhan D, Fang Y, Li L, Chen G, Chen S, Wang L. Microplastics, potential threat to patients with lung diseases. Front Toxicol. 2022 Sep 28;4:958414. doi: 10.3389/ftox.2022.958414. Erratum in: Front Toxicol. 2022 Dec 20;4:1119994. doi: 10.3389/ftox.2022.1119994. PMID: 36245793; PMCID: PMC9555848.
35. Epeslidou E, Scott JS, de Klein B, Cudia JT, Melgert B, Prekovic S. Microplastics as environmental modifiers of lung disease. EMBO Mol Med. 2026 Feb;18(2):381-395. doi: 10.1038/s44321-025-00353-w. Epub 2025 Dec 11. PMID: 41381807; PMCID: PMC12905266.
36. Vasse GF, Melgert BN. Microplastic and plastic pollution: impact on respiratory disease and health. Eur Respir Rev. 2024 Jun 12;33(172):230226. doi: 10.1183/16000617.0226-2023. PMID: 39009408; PMCID: PMC11262622.
37. Baroni A, Moulton C, Cristina M, Sansone L, Belli M, Tasciotti E. Nano- and Microplastics in the Brain: An Emerging Threat to Neural Health. Nanomaterials (Basel). 2025 Sep 4;15(17):1361. doi: 10.3390/nano15171361. PMID: 40938039; PMCID: PMC12429919.
38. Liu S, He Y, Yin J, Zhu Q, Liao C, Jiang G. Neurotoxicities induced by micro/nanoplastics: A review focusing on the risks of neurological diseases. J Hazard Mater. 2024 May 5;469:134054. doi: 10.1016/j.jhazmat.2024.134054. Epub 2024 Mar 15. PMID: 38503214.
39. Fang SJ, Yin ZD, Li LF, Cai Q, Zheng PF, Chen LZ. Overall effects of microplastics on brain. Front Toxicol. 2025 Nov 20;7:1619096. doi: 10.3389/ftox.2025.1619096. PMID: 41357964; PMCID: PMC12675269.
40. Zhang S, Wu J, Wu M, Qin Y, Zhu N, Shen Y, Chen R, Chen Z. From environment to brain: the role of microplastics in neurobehavioral disorders. Front Neurosci. 2025 Dec 1;19:1691461. doi: 10.3389/fnins.2025.1691461. PMID: 41404032; PMCID: PMC12702878.
41. Covello C, Di Vincenzo F, Cammarota G, Pizzoferrato M. Micro(nano)plastics and Their Potential Impact on Human Gut Health: A Narrative Review. Curr Issues Mol Biol. 2024 Mar 21;46(3):2658-2677. doi: 10.3390/cimb46030168. PMID: 38534784; PMCID: PMC10968954.
42. Bora SS, Gogoi R, Sharma MR, Anshu, Borah MP, Deka P, Bora J, Naorem RS, Das J, Teli AB. Microplastics and human health: unveiling the gut microbiome disruption and chronic disease risks. Front Cell Infect Microbiol. 2024 Nov 25;14:1492759. doi: 10.3389/fcimb.2024.1492759. PMID: 39669275; PMCID: PMC11635378.
43. Eichinger, J., Tretola, M., Seifert, J., & Brugger, D. (2024). Review: interactions between microplastics and the gastrointestinal microbiome. Italian Journal of Animal Science, 23(1), 1044–1056. https://doi.org/10.1080/1828051X.2024.2377642
44. Thin ZS, Chew J, Ong TYY, Raja Ali RA, Gew LT. Impact of microplastics on the human gut microbiome: a systematic review of microbial composition, diversity, and metabolic disruptions. BMC Gastroenterol. 2025 Aug 13 ; 25 (1): 583 . doi: 10 . 1186 / s12876-025-04140-2. PMID: 40804621; PMCID: PMC12351775.
45. Sofield CE, Anderton RS, Gorecki AM. Mind over Microplastics: Exploring Microplastic-Induced Gut Disruption and Gut-Brain-Axis Consequences. Curr Issues Mol Biol. 2024 Apr 30;46(5):4186-4202. doi: 10.3390/cimb46050256. PMID: 38785524; PMCID: PMC11120006.
46. Mohr AE, Jäger R, Carpenter KC, Kerksick CM, Purpura M, Townsend JR, West NP, Black K, Gleeson M, Pyne DB, Wells SD, Arent SM, Kreider RB, Campbell BI, Bannock L, Scheiman J, Wissent CJ, Pane M, Kalman DS, Pugh JN, Ortega-Santos CP, Ter Haar JA, Arciero PJ, Antonio J. The athletic gut microbiota. J Int Soc Sports Nutr. 2020 May 12;17(1):24. doi: 10.1186/s12970-020-00353-w. PMID: 32398103; PMCID: PMC7218537.
47. Martin D, Bonneau M, Orfila L, Horeau M, Hazon M, Demay R, Lecommandeur E, Boumpoutou R, Guillotel A, Guillemot P, Croyal M, Cressard P, Cressard C, Cuzol A, Monbet V, Derbré F. Atypical gut microbial ecosystem from athletes with very high exercise capacity improves insulin sensitivity and muscle glycogen store in mice. Cell Rep. 2025 Apr 22;44(4):115448. doi: 10.1016/j.celrep.2025.115448. Epub 2025 Mar 27. PMID: 40154488.
48. Menichetti A, Mordini D, Montalti M. Penetration of Microplastics and Nanoparticles Through Skin: Effects of Size, Shape, and Surface Chemistry. J Xenobiot. 2024 Dec 31;15(1):6. doi: 10.3390/jox15010006. PMID:

39846538; PMCID: PMC11755607.
49. Tan E, Saha S, Niebel D. Plastics in dermatology: A review and solutions. J Eur Acad Dermatol Venereol. 2025;39:1715–1724.https://doi.org/10.1111/jdv.20537
50. Aristizabal M, Jiménez-Orrego KV, Caicedo-León MD, et al. Microplastics in dermatology: Potential effects on skin homeostasis. J Cosmet Dermatol. 2024;23:766-772. doi:10.1111/ jocd.16167
51. Amran NH, Zaid SSM, Mokhtar MH, Manaf LA, Othman S. Exposure to Microplastics during Early Developmental Stage: Review of Current Evidence. Toxics. 2022 Oct 10;10(10):597. doi: 10.3390/toxics10100597. PMID: 36287877; PMCID: PMC9611505.
52. Tyc HJ, Kłodnicka K, Teresińska B, Karpiński R, Flieger J, Baj J. Micro- and Nanoplastics as Disruptors of the Endocrine System-A Review of the Threats and Consequences Associated with Plastic Exposure. Int J Mol Sci. 2025 Jun 26;26(13):6156. doi: 10.3390/ ijms26136156. PMID: 40649932; PMCID: PMC12249724.
53. Ullah S, Ahmad S, Guo X, Ullah S, Ullah S, Nabi G, Wanghe K. A review of the endocrine disrupting effects of micro and nano plastic and their associated chemicals in mammals. Front Endocrinol (Lausanne). 2023 Jan 16;13:1084236. doi: 10.3389/fendo.2022.1084236. PMID: 36726457; PMCID: PMC9885170.
54. Bossio S, Ruffolo SA, Lofaro D, Perri A, La Russa MF (2025) Endocrine toxicity of micro-and nanoplastics, and advances in detection techniques for human tissues: a comprehensive review. Endocrines 6(2):23 https://doi.org/10.3390/endocrines6020023
55. Kehinde S.A., Fatokun T.P., Olajide A.T., Praveena S.M., Sokan-Adeaga A.A., Adekunle A.P., Fouad D., Papadakis M. Impact of polyethylene microplastics exposure on kallikrein-3 levels, steroidal-thyroidal hormones, and antioxidant status in murine model: Protective potentials of naringin. Sci. Rep. 2024;14:23664. doi: 10.1038/s41598-024-74637-5.
56. Muhammad Babar Khawar, Ali Afzal, Kashifa Jalil, Tayyba Jan, Zaira Ahmad, Amna Rehman, Hanan Afzal, Sadia Ahmad, Afzal Nimra, Muhammad Ahsan Ashraf. Unraveling the endocrine disruption potential of microplastics in testosterone regulation. Journal of Environmental Sciences, Volume 164, 2026, Pages 827-836, ISSN 1001-0742. Doi: 10.1016/j.jes.2025.12.063.
57. Li Z, Robaire B. Effects of Endocrine-Disrupting Chemicals on Adrenal Function. Endocrinology. 2025 Feb 27;166(4):bqaf045. doi: 10.1210/endocr/bqaf045. PMID: 40048632; PMCID: PMC11907101.
58. Wang M, Wu Y, Li G, Xiong Y, Zhang Y, Zhang M. The hidden threat: Unraveling the impact of microplastics on reproductive health. Sci Total Environ. 2024 Jul 20;935:173177. doi: 10.1016/j.scitotenv.2024.173177. Epub 2024 May 13. PMID: 38750730.
59. Fusagawa H, Youn A, Wilkerson E, Pandya N, Feeley BT. The Effects of Microplastics on Musculoskeletal Disorder; A Narrative Review. Curr Rev Musculoskelet Med. 2025 Feb;18(2):39-47. doi: 10.1007/s12178-024-09932-9. Epub 2024 Nov 22. PMID: 39572502; PMCID: PMC11775366.
60. Vethaak AD, Legler J. Microplastics and human health. Science. 2021 Feb 12;371(6530):672-674. doi: 10.1126/science.abe5041. PMID: 33574197.