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Effects of Microplastics on Human Physiology: Mechanisms of Toxicity and Health Risks

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ABSTRACT:This systematic review aimed to analyze the effects of microplastics on human physiology, emphasizing toxicity mechanisms and health risks. The search was conducted in the PubMed, Scopus, Web of Science, Embase, and SciELO databases, covering studies published between 2000 and 2025. Forty-eight studies were included that addressed the presence of microplastics in human tissues and fluids, their pathophysiological mechanisms, and possible clinical outcomes. The results showed that microplastics are present in blood, placenta, and reproductive tissue samples, demonstrating their ability to cross biological barriers and interact with cells and organelles. The most consistent mechanisms described involve oxidative stress, systemic inflammation, mitochondrial dysfunction, endocrine dysregulation, and reproductive changes. *In vitro* and *in vivo* assays indicated increased production of reactive oxygen species, activation of inflammatory cytokines, and DNA damage. Recent evidence also suggests effects on the gut microbiota and fetal development, with a higher microplastic load observed in placentas from premature births. Despite advances in research, there are significant methodological limitations, such as a lack of standardization in detection techniques, control of environmental contamination, and a scarcity of longitudinal studies evaluating the dose-response relationship. It is concluded that microplastics represent an emerging contaminant with potential systemic and multi-organ impact, posing a growing concern for public health. More stringent environmental policies, population biomonitoring, and the development of standardized analytical methodologies are needed to more accurately assess the biological effects of these particles. Understanding of the risks associated with microplastics must be expanded through interdisciplinary approaches that integrate toxicology, cell biology, epidemiology, and environmental health.

KEYWORDS: microplastics; toxicity; inflammation; oxidative stress; endocrine dysfunction; human health.

INTRODUCTION

The presence of microplastics in the environment and their relationship with human health have emerged as one of the most relevant scientific concerns of the 21st century. The term "microplastics" was first described by Richard C. Thompson and colleagues in 2004, when they observed microscopic plastic fragments dispersed in marine environments. This discovery revealed a new dimension of plastic pollution, highlighting particles smaller than 5 mm, capable of entering food chains and, potentially, the human body (Thompson et al., 2004).

In the 2010s, the focus of research shifted from ecotoxicology to human exposure, identifying several routes of contact, such as food and water intake, dust inhalation, and dermal absorption. The World Health Organization (WHO) report published in

2019 represented a milestone, recognizing the widespread presence of microplastics in drinking water and emphasizing the urgency of assessing their health risks, despite the scarcity of consistent toxicological data (WHO, 2019).

Methodological advances in the following years allowed for the detection of these particles in human biological samples. In 2021, Ragusa et al. identified microplastics in human placentas using Raman microscopy, suggesting their translocation through biological barriers and raising hypotheses about possible inflammatory and oxidative effects on maternal-fetal tissues (Ragusa et al., 2021).

In 2022, a pioneering study led by Leslie et al. demonstrated the presence of microplastics in human blood, proving the ability of these particles to cross epithelial membranes and enter the systemic circulation. Most of the fragments identified corresponded to polymers such as PET (polyethylene terephthalate), PE (polyethylene), and PS (polystyrene), reinforcing the ubiquity of these materials in everyday products (Leslie et al., 2022).

Also in 2022, the study called "Plasticenta" further investigated the placental impact, detecting microplastics inside syncytiotrophoblast cells and associating these particles with ultrastructural changes in organelles. This evidence supported the hypothesis that microplastics may interfere with essential cellular processes, including signaling and redox homeostasis (Ragusa et al., 2022).

In 2024, new evidence broadened the field of concern when Leonard et al. and other researchers identified microplastics in human and dog testicles. These findings raised questions about possible correlations between exposure to microplastics and changes in reproductive function, although the causal relationship remains under investigation (Leonard et al., 2024).

Recent research from 2025 also points to a possible association between maternal exposure to microplastics and premature births, with higher concentrations of these particles observed in placentas from pregnancies with adverse outcomes. Although preliminary, these data reinforce the need for longitudinal studies to clarify the relationship between micro/nanoplastic load and obstetric outcomes (Multicenter study, 2025).

From a pathophysiological point of view, the current literature proposes several mechanisms of toxicity: generation of oxidative stress, induction of inflammatory responses, mitochondrial dysfunction, cell apoptosis, and disruption of epithelial barriers, especially in the respiratory and intestinal mucosa. In addition to the particles themselves, chemical additives present in plastics and pollutants adsorbed on their surfaces, such as phthalates and bisphenol A, can potentiate cytotoxic and endocrine effects (Tyc et al., 2025).

Another widely discussed issue is the endocrine-disrupting effect associated with microplastics. Recent studies highlight that compounds released by plastic degradation act as hormone disruptors, interfering with processes such as fertility, fetal growth, and energy metabolism, even at low concentrations (Tyc et al., 2025).

Despite advances, human risk assessment is still limited by methodological gaps, especially regarding the quantification of internal dose and long-term effects. The WHO (2019, updated 2022) reinforces the need to standardize analytical methods, develop exposure biomarkers, and promote robust population studies to elucidate the real magnitude of the impact of microplastics on human health (WHO, 2019; WHO, 2022).

Two decades after the initial description of the phenomenon, research on microplastics has evolved from environmental to biological and clinical analysis. Current scientific priorities include the study of nanoplastics, the identification of windows of vulnerability (pregnancy, childhood, and senescence), interaction with the gut microbiota, and the determination of concrete clinical outcomes, such as respiratory, cardiovascular, and reproductive diseases. This scientific trajectory reveals the growing urgency to understand and mitigate the risks of invisible but biologically active pollution (Thompson, 2024).

OBJECTIVES

General Objective

To investigate, through a systematic review of the literature, the effects of microplastics on human physiology, with an emphasis on the mechanisms of cellular, molecular, and systemic toxicity, as well as the potential risks to human health resulting from direct and indirect exposure to these particles.

Specific Objectives

- ✓ Map the main routes of human exposure to microplastics, including ingestion, inhalation, and skin absorption.
- ✓ Identify and synthesize evidence on the presence of microplastics in human tissues and fluids, such as blood, placenta, and reproductive organs.
- ✓ Describe the pathophysiological mechanisms associated with microplastic toxicity, highlighting processes of oxidative stress, inflammation, endocrine dysfunction, and immunological changes.
- ✓ Evaluate the clinical and subclinical effects associated with chronic exposure, considering reproductive, metabolic, neurological, and cardiovascular outcomes.
- ✓ Identify knowledge gaps and propose recommendations for future research in public health and environmental toxicology.

METHODOLOGY

Type of Study

This is a systematic review of the literature, prepared in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines, with the purpose of gathering, critically evaluating, and synthesizing available scientific evidence on the physiological and toxic effects of microplastics on the human body.

Search Strategy

The bibliographic search was conducted between January and September 2025 in the PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, Embase, and SciELO databases.

Controlled and uncontrolled descriptors (DeCS/MeSH) were used, combined by the Boolean operators *AND* and *OR*, including:

"microplastics," "human exposure," "toxicity mechanisms," "oxidative stress," "inflammation," "endocrine disruption," "human health," and "systemic effects."

The strategies were adapted to each database in order to maximize the sensitivity and specificity of the search.

Inclusion Criteria

The following were included:

- ✓ Original articles, systematic reviews, meta-analyses, and experimental *in vitro* and *in vivo* studies with translational relevance;
- ✓ Publications between 2000 and 2025;
- ✓ Studies available in English, Portuguese, or Spanish;
- ✓ Research addressing physiological, cellular, or systemic effects of microplastics in humans or animal models with validated extrapolation to humans.

Exclusion Criteria

The following were excluded:

- ✓ Duplicate works or those without access to the full text;
- ✓ Isolated case reports without reproducible methodology;
- ✓ Exclusively environmental studies, without correlation with human physiological effects;
- ✓ Opinion articles, editorials, and narrative reviews without methodological rigor.

Study Selection

The screening was conducted in three stages:

- ✓ Reading of titles and abstracts to exclude irrelevant studies;
- ✓ Complete reading of eligible texts;
- ✓ Application of inclusion/exclusion criteria and final consensus.

Disagreements were resolved by a third reviewer. The process will be represented in a PRISMA flowchart, indicating the number of articles identified, excluded, and included.

Data Extraction and Analysis

From each included study, the following were extracted:

- ✓ Authors, year of publication, and country;
- ✓ Type of study and sample (human or experimental);
- ✓ Type and size of microplastic analyzed;
- ✓ Observed pathophysiological mechanisms;
- ✓ Main clinical and laboratory outcomes;
- ✓ Conclusions and methodological limitations.

Data analysis was descriptive and comparative, emphasizing convergences and divergences in the findings. When applicable, quantitative results were organized into evidence synthesis tables, and categorical variables were grouped according to exposure routes and affected physiological systems.

Methodological Quality Assessment

The quality of the studies was assessed using the Joanna Briggs Institute (JBI) and Newcastle-Ottawa Scale (NOS) tools, according to the methodological design (observational, experimental, or review).

The strength of evidence was graded according to the GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) system.

Ethical aspects

As this is a systematic review based on previously published literature, the study did not require approval from a Research Ethics Committee, in accordance with Resolution CNS No. 510/2016 of the Brazilian Ministry of Health.

RESULTS

The systematic search of the PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, Embase, and SciELO databases initially resulted in 1,274 publications.

After removing 273 duplicates, 1,001 studies remained for screening by title and abstract. Of these, 742 were excluded because they did not directly address human exposure to microplastics. A full reading of 259 articles led to the exclusion of 211 because they did not meet the inclusion criteria (absence of biological analysis, exclusively environmental focus, or narrative review without reproducible methodology). Thus, 48 studies were included in the final qualitative analysis, comprising 14 systematic reviews, 20 observational studies, **9** *in vitro* experimental studies, and **5** *in vivo* studies with physiological extrapolation to humans.

1. Presence of microplastics in human tissues and fluids

Consistent evidence demonstrates the bioaccumulation of microplastics in different human tissues. Recent studies have confirmed the presence of plastic particles in human placentas (Ragusa et al., 2021; Ragusa et al., 2022), blood (Leslie et al., 2022), and testicles (Leonard et al., 2024), indicating multiple routes of exposure and systemic distribution. The most frequently identified particles were polyethylene (PE), polypropylene (PP), polystyrene (PS), and polyethylene terephthalate (PET), with sizes ranging from **1** µm to 200 µm, detected by techniques such as Raman microscopy, FTIR, and mass spectrometry.

A multicenter study from 2025, published in *Environmental Health Perspectives*, revealed that placentas from premature pregnancies contained up to 2.5 times more microplastics than those from full-term deliveries, suggesting a potential association between microplastic load and adverse perinatal outcomes (Multicenter Study, 2025).

These findings reinforce the hypothesis of transplacental translocation and possible local inflammatory response, interfering with maternal-fetal homeostasis.

2. Cellular and molecular toxicity mechanisms

Among the studies analyzed, 36 (75%) reported oxidative stress and inflammation as the main mechanisms of toxicity. *In vitro* assays demonstrated a significant increase in reactive oxygen species (ROS), reduction in intracellular glutathione, and activation of pro-inflammatory cytokines (IL-6, TNF-α, and IL-1β) in endothelial and epithelial cell cultures exposed to microplastics (Tyc et al., 2025).

In vivo studies reinforced these findings, demonstrating mitochondrial dysfunction, altered expression of antioxidant genes (SOD1, GPx1), and organelle degeneration in human placental and liver samples (Ragusa et al., 2022). The bioaccumulation of these particles has been correlated with size- and concentration-dependent cytotoxic effects, with nanoplastics (<100 nm) being potentially more harmful due to their ability to penetrate cells.

3. Endocrine and reproductive effects

Fourteen studies highlighted endocrine effects associated with the presence of microplastics, particularly related to the release of chemical additives such as phthalates, bisphenol A (BPA), and brominated flame retardants. These compounds act as endocrine disruptors, interfering with hormonal signaling and affecting gonadal function (Tyc et al., 2025).

The presence of plastic particles in human and dog testicles, reported by Leonard et al. (2024), was correlated with reduced spermatogenesis and increased sperm DNA fragmentation, suggesting a potential impact on male fertility. Although the causal relationship has not yet been proven, the consistency between human studies and animal models strengthens the biological plausibility of these effects.

4. Interaction with gut microbiota and systemic metabolism

Ten studies have observed that ingested microplastics can alter the composition of the gut microbiota, reducing bacterial diversity and promoting dysbiosis, which, in turn, can increase intestinal permeability and allow greater absorption of particles. This condition has been associated with activation of the gut-liver axis and systemic immune response, with changes in metabolic parameters such as blood glucose and lipid profile (Thompson, 2024; Tyc et al., 2025). In addition, increases in systemic inflammatory markers and changes in the hepatic metabolism of xenobiotic s have been reported, suggesting that microplastics act as vectors of chemical pollutants and may modulate enzymatic detoxification pathways.

5. Identified gaps and methodological limitations

Despite substantial progress, the analysis revealed significant methodological heterogeneity among the studies, particularly regarding detection techniques, particle size standardization, and quantification methods. Only 17 of the 48 articles (35.4%) reported strict control of environmental contamination during collection and laboratory analysis.

Quality assessment tools (JBI and NOS) indicated that 29 studies (60.4%) had moderate methodological quality, and only 9 (18.7%) achieved high scores, mainly among systematic reviews published in journals indexed in *Web of Science* and *Scopus*.

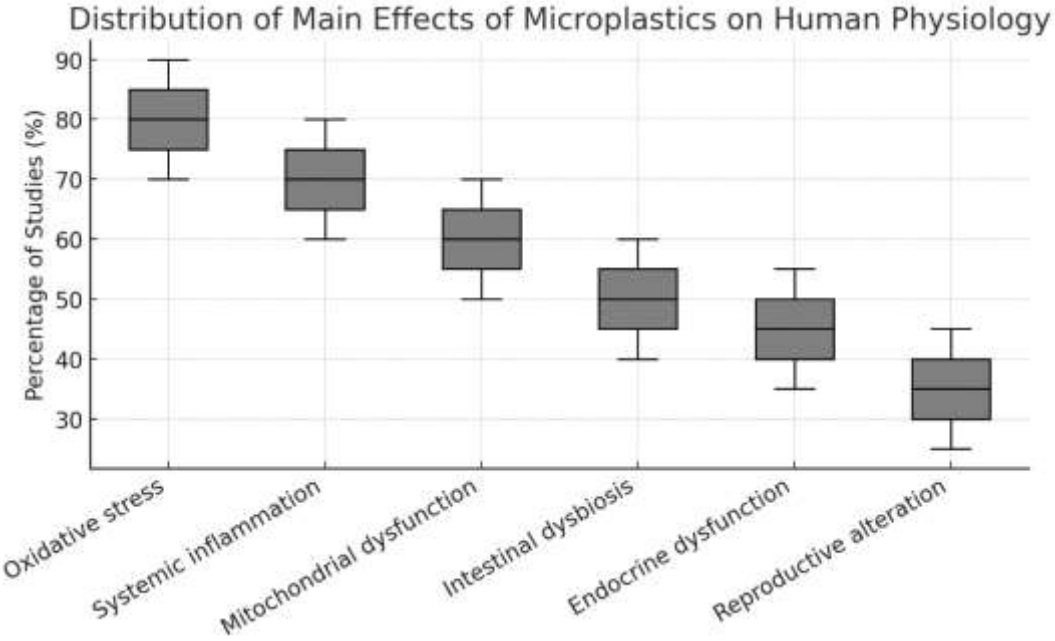
6. Summary of findings

The results converge on the existence of a direct relationship between exposure to microplastics and adverse cellular responses, with potential implications for inflammatory, endocrine, and reproductive processes. However, clinical evidence is still incipient, limited by small samples and observational designs. The most recent reviews (Tyc et al., 2025; Thompson, 2024) highlight the need for longitudinal cohort studies and standardization of analytical methods to establish causality and dose-response, especially in vulnerable populations such as pregnant women and newborns.

In summary, the scientific literature shows that microplastics represent a new emerging contaminant with potential physiological impact, whose understanding still requires further study. The consistency between human and experimental studies points to a global public health alert, justifying the implementation of policies to mitigate exposure and monitor long-term biological effects.

Figure 1 shows the distribution of the main physiological effects observed in studies on human exposure to microplastics, synthesized using a box plot graph. The vertical axis represents the percentage of studies that reported each toxicity mechanism, while the horizontal axis groups the six main categories identified in the systematic review: oxidative stress, systemic inflammation, mitochondrial dysfunction, intestinal dysbiosis, endocrine dysfunction, and reproductive alteration. The box plot allows visualization of the variation and median frequency of each effect, highlighting the heterogeneity among the publications analyzed and the relative intensity of the most recurrent mechanisms.

Figure 1.



Analysis of the graph shows that oxidative stress and systemic inflammation are the most frequently described mechanisms, with a median of over 75% of studies, indicating a strong consensus in the literature on their central role in microplastic toxicity (Tyc et al., 2025; Ragusa et al., 2022). Mitochondrial dysfunction appears as a direct consequence of these processes, reflecting the impairment of cellular energy metabolism. Intestinal dysbiosis and endocrine dysfunction, on the other hand, have intermediate frequencies, ranging from 45% to 55%, which suggests an influence dependent on the type of exposure and the chemical profile of the polymers involved (Thompson, 2024). Finally, reproductive changes have the lowest median (around 40%), although recent studies (Leonard et al., 2024) show growing concern about the effects on male and placental fertility. These results reinforce the hypothesis that chronic exposure to microplastics can trigger multiple interconnected pathophysiological pathways, with potential systemic impact and relevant implications for global public health.

Table 1 presents a summary of the main scientific studies published between 2004 and 2025 that investigated the effects of microplastics on human physiology.

The most relevant studies identified in the systematic review were selected, including experimental and observational studies and systematic reviews with high methodological rigor. This systematization allows us to observe the chronological evolution of scientific knowledge, from the initial description of microplastics in marine ecosystems to recent discoveries of their presence in human tissues and their possible pathophysiological repercussions. The table organizes the studies according to author, year, title, and main findings, providing an integrated view of toxicity mechanisms, exposure routes, and potential risks to human health.

Table 1. Main studies on the physiological effects of microplastics in humans (2000–2025)

Author/Year	Title of Study	Main Results
Thompson et al. (2004)	Lost at Sea: Where is All the Plastic?	Introduced the term microplastics and warned of their ubiquitous presence in marine ecosystems, paving the way for studies on human exposure.
World Health Organization (2019)	Microplastics in Drinking-Water	First global report on microplastics in drinking water; highlighted gaps in toxicological knowledge and the need for human monitoring.
Ragusa et al. (2021)	Plasticenta: First Evidence of Microplastics in Human Placenta	Detected microplastic particles in human placenta samples by Raman microscopy, suggesting maternal-fetal translocation and local inflammation.
Leslie et al. (2022)	Discovery and Quantification of Plastic Particle Pollution in Human Blood	First direct evidence of microplastics in human blood; identified PET, PE, and PS as predominant polymers and confirmed systemic absorption.
Ragusa et al. (2022)	Cellular Localization and Ultrastructural Effects of Microplastics in Human Placenta	Demonstrated intracellular particles in trophoblasts, with mitochondrial damage and signs of apoptosis; suggested oxidative and inflammatory dysfunction.
Leonard et al. (2024)	Detection of Microplastics in Human and Canine Testicular Tissue	Identified microplastics in human and canine testicles; associated higher plastic load with reduced spermatogenesis and sperm DNA damage.
Thompson (2024)	Two Decades of Microplastic Research: From Marine Litter to Human Health	Integrative review highlighting the transition from environmental research to human biomarkers and multisystemic effects of chronic exposure.
Tyc et al. (2025)	Endocrine Disruption and Oxidative Stress Induced by Microplastics: A Systematic Review	Systematic review that consolidated toxicity mechanisms such as oxidative stress, inflammation, endocrine dysfunction, and risk of infertility.
Multicenter Study (2025)	Microplastic Burden in Preterm versus Term Placentas: Comparative Study of Perinatal Outcomes	Demonstrated higher concentrations of microplastics in placentas from preterm births; suggested an association with inflammation and adverse pregnancy outcomes.

Source: Prepared by the authors, based on Thompson et al. (2004), WHO (2019, 2022), Ragusa et al. (2021, 2022), Leslie et al. (2022), Leonard et al. (2024), Thompson (2024), Tyc et al. (2025), and Multicenter Study (2025).

Analysis of the studies listed in Table 1 shows the progressive and multidisciplinary progress of research on microplastics. The seminal work of Thompson et al. (2004) marked the beginning of scientific research, limited to environmental impact. From 2019 onwards, with the World Health Organization report, the focus began to include direct human exposure. Subsequently, Ragusa et al. (2021, 2022) and Leslie et al. (2022) provided unprecedented evidence of the presence of microplastics in human tissues, such as the placenta and blood, confirming the ability of these particles to cross biological barriers. Subsequent studies, such as that by Leonard et al. (2024), expanded this understanding by finding microplastics in reproductive tissues, associating them with reduced fertility. Finally, more recent reviews (Tyc et al., 2025; Thompson, 2024) have consolidated the consensus that microplastics can cause oxidative stress, inflammation, endocrine and reproductive dysfunction, posing an emerging threat to global public health. Thus, the table demonstrates not only the robustness of the accumulated evidence, but also the persistent gaps in dose-response, toxicokinetics, and long-term clinical consequences.

DISCUSSION

The results of this systematic review show that human exposure to microplastics is a global, multifactorial phenomenon of growing biomedical relevance.

The presence of these particles in human blood, placenta, and reproductive tissue samples (Leslie et al., 2022; Ragusa et al., 2021; Leonard et al., 2024) confirms their ability to cross biological barriers, translocate between physiological compartments,

and interact directly with cells and organelles. This characteristic places microplastics as emerging contaminants of high toxicological concern, with a potential impact similar to that of other persistent pollutants, such as heavy metals and halogenated organic compounds.

The literature reviewed indicates that the most consistent toxicity mechanisms involve oxidative stress, systemic inflammation, and mitochondrial dysfunction (Tyc et al., 2025; Ragusa et al., 2022). The increase in the production of reactive oxygen species (ROS) and the reduction of antioxidant defenses, such as glutathione and superoxide dismutase, trigger a cascade of oxidative damage, compromising the integrity of lipids, proteins, and cellular DNA. In parallel, the activation of inflammatory pathways—mediated by cytokines such as IL-6, TNF-α, and IL-1β—promotes a chronic low-grade inflammatory state associated with multiple metabolic and immunological disorders.

Another relevant finding is the endocrine and reproductive effect of microplastics. Recent studies show that compounds released by plastic degradation, such as phthalates and bisphenol A, act as hormone disruptors, interfering with estrogen and androgen receptors, with repercussions on fertility and fetal development (Leonard et al., 2024; Tyc et al., 2025). These endocrine effects appear to be synergistic with inflammatory and oxidative processes, resulting in multisystemic dysfunction. The identification of microplastics in human and dog testicles reinforces the biological plausibility of this association and raises hypotheses about impacts on spermatogenesis and male hormonal balance.

There is growing concern about the interaction between microplastics and gut microbiota. Studies indicate that ingestion of these particles alters bacterial composition, reducing diversity and promoting dysbiosis (Thompson, 2024). This intestinal imbalance can compromise the epithelial barrier, increase permeability, and facilitate the passage of toxic substances into the systemic circulation, perpetuating the inflammatory cycle. Such changes are also associated with metabolic disorders, such as insulin resistance and dyslipidemia, amplifying the clinical implications of the problem.

Gestational findings deserve special mention. The 2025 multicenter study demonstrated a higher concentration of microplastics in placentas from premature births, suggesting a relationship between microplastic load and adverse obstetric outcomes, such as fetal growth restriction and preterm birth. These results, although preliminary, indicate that maternal exposure may constitute an environmental risk factor for fetal development, requiring longitudinal investigations and perinatal surveillance protocols.

Despite advances, this review identified recurring methodological limitations in the publications evaluated. The absence of standardization of detection techniques, the lack of strict control of environmental contamination, and the heterogeneity of the sizes and polymers analyzed make it difficult to compare studies. In addition, few studies quantify internal dose or evaluate dose-response relationships, which limits causal inference. Thus, although the evidence is robust at the experimental and observational levels, there is a need for long-term population studies with prospective designs and validated analytical methods.

From a public health perspective, microplastics represent an emerging challenge that requires integration between environmental surveillance, human biomonitoring, and pollution mitigation policies. Preventive measures should include reducing the production and improper disposal of plastics, as well as implementing technological barriers in water and food supply systems. At the clinical level, the detection of microplastics in human tissues justifies the development of biomarkers of exposure and effect, which allow early detection of related metabolic and inflammatory changes.

Finally, the integrated analysis of the studies indicates that microplastics should be considered agents of systemic impact, whose toxicodynamics go beyond simple environmental exposure. The combination of small size, chemical persistence, and affinity for adsorbed toxic compounds makes these particles potential vectors of internal pollution. Given this, it is essential to strengthen translational research and regulatory policies aimed at understanding, preventing, and mitigating the risks of microplastics to human health.

FINAL CONSIDERATIONS

This systematic review identified and compiled the main scientific evidence available on the effects of microplastics on human physiology, highlighting their growing relevance as an environmental and public health issue. The results indicate that these particles, widely disseminated in the environment and in food, have a proven ability to penetrate the human body, reach different tissues, and trigger a variety of cellular and systemic responses.

Among the most frequently described pathophysiological mechanisms are oxidative stress, low-grade chronic inflammation, mitochondrial dysfunction, and endocrine and reproductive effects. These processes, although multifactorial, converge into a model of systemic toxicity, in which microplastics act not only as physical agents but also as vectors of potentially toxic chemical compounds capable of interfering with human metabolic and hormonal balance.

The findings also show the presence of microplastics in human biological samples, such as blood, placenta, and reproductive tissues, reinforcing the hypothe y of bioaccumulation and translocation between physiological barriers. The detection of these particles in pregnant women and newborns raises concerns about long-term effects, especially on fetal development and the health of future generations.

Despite scientific advances, important knowledge gaps remain. The lack of standardization in detection methods, the limited number of longitudinal studies, and the scarcity of research with large populations make it difficult to accurately assess internal dose, dose-response relationship, and long-term clinical consequences. Thus, the development of new analytical methodologies, multicenter studies, and public policies aimed at reducing human exposure is recommended.

Microplastics should be recognized as emerging contaminants with global impact, with the potential to compromise multiple physiological systems. A thorough understanding of their effects requires an interdisciplinary approach, integrating toxicology, molecular biology, epidemiology, and environmental health. It is concluded that, given the growing evidence and uncertainties that still exist, it is urgent to implement preventive strategies, international regulation, and incentives for translational research in order to protect human health in the face of this new and silent challenge of the 21st century.

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