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INVISIBLE THREAT: THE IMPACT OF MICROPLASTICS ON HUMAN HEALTH

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ABSTRACT

Microplastics have become ubiquitous environmental pollutants with growing evidence of human exposure and possible danger. These particles, which are usually smaller than 5 mm, come from direct industrial manufacture or the breakdown of bigger plastics. They are found in food, water, air, and even human biological samples, according to recent research, which raises grave worries about their potential toxicological effects. This review critically examines exposure pathways, toxicokinetic, cellular and molecular mechanisms, and associated health risks. Emphasis is placed on oxidative stress, inflammation, endocrine disruption, and organ-specific toxicity. Significant gaps in our knowledge of long-term effects persist despite mounting evidence, calling for additional study and regulatory attention.

1.INTRODUCTION

Over the past century, the widespread manufacture and use of plastics have improved industrial development, healthcare systems, transportation, agriculture, food packaging, and consumer convenience, all of which have revolutionized modern society [1]. But the same resilience and durability that made plastics profitable have also led to serious environmental degradation. Since the 1950s, the amount of plastic produced worldwide has grown dramatically, reaching hundreds of millions of metric tons each year. A significant amount of plastic garbage eventually builds up in terrestrial and aquatic environments because it is not properly recycled or managed. Microplastics are created when larger plastic materials fragment over time as a result of environmental weathering processes as UV radiation, mechanical abrasion, hydrolysis, and microbial deterioration (2). Plastic particles less than five millimetres are commonly referred to as microplastics. These particles fall into two general categories: primary and secondary microplastics. Primary microplastics are purposefully produced in tiny sizes for use in pharmaceuticals, cosmetics, industrial applications, and synthetic textiles. The breakdown of bigger plastic waste, including bottles, fishing nets, food containers, and packaging materials, produces secondary microplastics [3]. Ongoing environmental fragmentation results in ever-tinier particles, such as nanoplastics, which have increasingly higher toxicological and biological reactivity. Due to their extensive distribution and mounting evidence of human exposure, microplastics have become a significant environmental and public health concern in recent years. Microplastics have been found in drinking water, freshwater systems, marine ecosystems, agricultural soil, seafood, salt, fruits, vegetables, and processed foods, according to studies [4]. Their omnipresence indicates that human exposure is virtually unavoidable. More importantly, recent investigations have detected microplastics in human blood, lung tissues, placenta, breast milk, and fecal samples, confirming their entry into the human body and raising concerns regarding systemic accumulation and toxicity. Beyond the actual particles, microplastics have significant toxicological implications [5]. Heavy metals, persistent organic pollutants, polycyclic aromatic hydrocarbons, bisphenols, and phthalates are just a few of the dangerous substances that these materials transport. Microplastics absorb pollutants from the environment and transfer them into biological systems because of their hydrophobic surface characteristics and high surface-area-to-volume ratio. The "Trojan horse effect" is the term used to describe this phenomenon, in which microplastics increase the toxicity and bioavailability of related contaminants [6]. The way that microplastics interact with biological membranes and cellular processes is another significant issue. Microplastics cause oxidative stress, mitochondrial malfunction, inflammatory reactions, apoptosis, endocrine disruption, and genotoxicity, according to experimental research [7]. Because of their small size, they can pass through epithelial barriers and reach the systemic circulation, where they may have an impact on the liver, lungs, kidneys, gastrointestinal tract, reproductive organs, and neurological system, among other organs. Because of their nanoscale size, nanoplastics may be more poisonous and more effective at penetrating biological membranes [8]. Concerns about the effects on the environment and food chain have also been raised by the growing load of plastic pollution. Microplastics are commonly consumed by aquatic organisms, either on purpose or by accident, which causes bioaccumulation and trophic transfer. Consuming seafood and drinking tainted water exposes humans to these particles. Inhalation exposure is also influenced by atmospheric microplastics, especially in urban and occupational settings [9]. There are still a number of questions about the long-term health effects of chronic microplastic exposure, despite increased scientific interest [10]. Toxicological interpretation is complicated by variable exposure evaluations, variations in polymer composition, inconsistent study procedures, and a lack of standardized detection tools. The majority of the evidence that is currently available comes from animal research and in vitro trials; human epidemiological data is still scarce [11]. Thus, the purpose of this study is to critically assess the available data on microplastics and human health. Environmental distribution, exposure routes, toxicokinetics, molecular mechanisms of toxicity, organ-specific effects, current scientific discoveries, and prospects for future study are the main topics of the review. This article emphasizes the critical need for all-encompassing techniques to lower human exposure and lessen the possible health burden associated with microplastics by combining data from toxicology, environmental science, molecular biology, and public health research [12].

2.SOURCES AND ENVIRONMENTAL DISTRIBUTION OF MICROPLASTICS

Numerous human activities and environmental deterioration processes are the source of microplastics. Their sources are typically divided into primary and secondary origins, both of which have a substantial impact on environmental contamination [13]. Primary microplastics are purposefully produced in minute sizes and are released into the environment through commercial and industrial processes. Typical examples include pellets used in the production of plastic, industrial abrasives, synthetic fibers released from textiles, and microbeads used in cosmetic and personal care goods. These particles are frequently released into wastewater systems, where their release into natural ecosystems is made possible by insufficient filtering and treatment procedures [14].

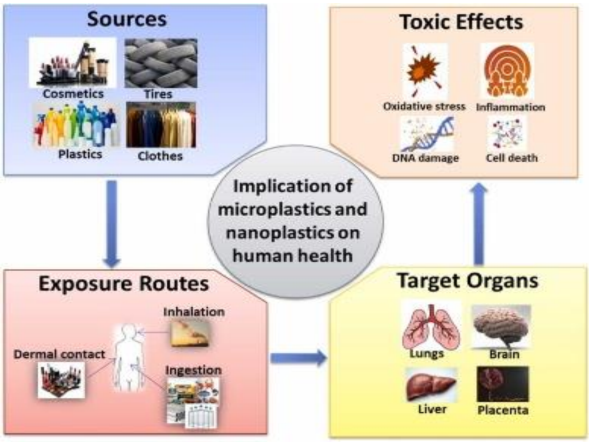

FIG-1 Sources and exposure of microplastics and nano plastics to human beings and its possible harmful health outcomes are major threats to public health [15].

Larger plastic items break down and fragment, producing secondary microplastics. Plastic waste gradually breaks down into tiny particles due to environmental exposure to UV light, wind abrasion, water turbulence, temperature changes, and microbiological activity [16]. Tires, bottles, household goods, fishing nets, packing materials, and used plastic bags are typical sources. Because of the constant friction between car tires and road surfaces, tire wear particles are now known to be a significant source of urban microplastic pollution [17]. Microplastics are extremely widely distributed in the environment. One of the most researched sources of microplastic pollution is marine environments. Every year, rivers, sewage systems, industrial discharge, and coastal activities send millions of tons of plastic debris into the ocean [18]. Depending on their density and surface characteristics, microplastics may float, stay floating, or sink into sediments once they are introduced into aquatic systems. These particles are consumed by marine species at various trophic levels, which causes biomagnification through food chains and ecological disruption [19]. Freshwater habitats are just as susceptible to pollution. Plastic waste is transported from terrestrial areas into marine environments mostly by rivers and lakes. Because many filtering systems are unable to entirely remove small particles, wastewater treatment plants are important sources. Freshwater contamination is also a result of sludge application and agricultural runoff. Another significant source of microplastics is terrestrial ecosystems [20]. Large amounts of plastic particles are introduced into soil through agricultural practices like sewage sludge application, greenhouse films, and plastic mulching. These particles change the microbial diversity, nutrient cycling, water retention capacity, and soil structure throughout time. Microplastics may be consumed by soil organisms like earthworms, which makes it easier for them to move throughout terrestrial ecosystems [21]. Microplastics in the atmosphere have become a major source of worry. Textile fibers, industrial emissions, car wear, household dust, and urban pollution are the main sources of airborne microplastics. Before being deposited, these particles might traverse great distances while suspended in the atmosphere. Microplastics have been found in far-off places like deep-sea sediments, polar ice, and mountainous areas, indicating their widespread distribution [22]. Because humans spend a lot of time indoors, indoor surroundings are thought to be especially significant sources of human exposure. Inhalable fibers and particles are released by furniture, upholstery, synthetic clothing, carpets, and household dust. Because of inadequate ventilation and ongoing particle shedding from household items, indoor concentrations may be higher than outdoor levels [23]. The chemical stability of synthetic polymers is a major factor in the environmental persistence of microplastics. The majority of plastics can last for decades or even centuries and are resistant to biodegradation. Particle shape and surface chemistry can be changed by environmental weathering, which increases the particles' capacity to interact with biological systems and absorb harmful substances [24]. The relationship between microplastics and environmental pollutants is another important issue. Microplastics absorb pollutants like pesticides, heavy metals, antibiotics, and persistent organic pollutants because they are hydrophobic. Because of their ability to adsorb, microplastics become vectors that can carry dangerous substances into living things and throughout ecosystems. The dispersal of microplastics around the world emphasizes the critical need for efficient waste management techniques, enhanced recycling infrastructure, a decrease in single-use plastics, and environmental monitoring initiatives. Without significant intervention, environmental contamination and human exposure are expected to increase substantially in the coming decades [25].

3.HUMAN EXPOSURE PATHWAYS

Microplastics are constantly ingested, inhaled, and come into touch with human skin. Ingestion and inhalation are thought to be the main ways that these pathways contribute to systemic exposure. Because microplastics are so common in the environment, people come into contact with them on a regular basis through consumer goods, food, water, and the air [26]. Currently, the most important exposure mechanism is thought to be dietary consumption. Seafood, shellfish, drinking water, bottled drinks, salt, fruits, vegetables, honey, sugar, and processed foods have all been found to contain microplastics in several studies. Because marine creatures directly consume microplastics from contaminated aquatic ecosystems, seafood exposure is especially significant. Microplastics are accumulated in significant quantities by filter-feeding species like mussels and oysters, and then consumed by humans [27]. Drinking water is yet another significant exposure source. The amount of microplastics in tap and bottled water varies. Because of the plastic packaging and bottling procedures, bottled water frequently exhibits higher levels of contamination. Particle shedding may result from repeatedly opening and shutting plastic containers [28]. Furthermore, prolonged storage and exposure to heat accelerate the deterioration of packaging materials. Materials used in food packaging are a major source of contamination. Particles are released by microwaveable plastic items, disposable cutlery, takeout packaging, and plastic containers under heat and mechanical stress. Even tea bags made of nylon or polyethylene terephthalate may release billions of microplastic particles during the brewing process, according to recent research [29]. Exposure to inhalants has grown in importance, particularly in urban and professional settings. Textile fibers, synthetic carpets, road dust, building materials, industrial emissions, and household dust are the sources of airborne microplastics. Indoors, fibrous particles generated by synthetic garments are more prevalent. People who work in waste management facilities, plastic industries, and textile manufacturers may be more exposed at work [30]. Depending on their size and aerodynamic properties, microplastics can settle in various parts of the respiratory system after being inhaled. Bigger particles are frequently caught in the upper respiratory tract and eliminated by mucociliary clearance processes. Smaller particles and nanoplastics, on the other hand, have the ability to reach systemic circulation by penetrating deeper into alveolar areas [31]. In comparison to ingesting and inhalation, dermal exposure has not received as much research. Under some circumstances, nanoplastics may penetrate damaged or compromised human skin, despite the fact that unbroken skin serves as an effective barrier against larger particles. Microplastic-containing cosmetics may increase localized exposure [32]. Microplastics interact with biological tissues and epithelial barriers once they enter the body. Mechanisms like endocytosis, persorption, and paracellular transport facilitate intestine absorption. Because of their tiny size and higher surface reactivity, nanoplastics have improved translocation capabilities. Research has shown that the liver, kidneys, lungs, and placenta are among the organs where these particles may build up [33]. Studies using human biomonitoring have verified the existence of microplastics in lung tissues, blood, faeces, and placentas. While placental accumulation raises questions about foetal exposure and developmental damage, detection in blood indicates systemic dissemination. These results point to the potential for long-term internal exposure even in populations that are healthy [34]. Age, occupation, food habits, social position, and geographic location all affect exposure levels. Due to their frequent hand-to-mouth behaviour and use of plastic feeding bottles, infants and young children may be more exposed. It has been demonstrated that heating feeding bottles made of polypropylene releases significant amounts of microplastics into baby formula. Because people come into contact with several sources at once during their lives, the cumulative aspect of exposure is particularly worrisome. Subtle biological effects from long-term low-dose exposure may eventually become clinically significant [35].

4.TOXICOKINETICS OF MICROPLASTICS

The mechanisms of absorption, distribution, metabolism, and excretion are all part of the toxicokinetics of micro plastics, which dictate their biological fate and toxicological effects in the human body. Microplastics have distinct physical and chemical properties that affect how they interact with tissues and biological systems, in contrast to traditional chemical toxicants. Their toxicokinetic behaviour is influenced by the size, shape, type of polymer, surface charge, hydrophobicity, and related pollutants of the particles [36]. Microplastics are mostly absorbed through the respiratory and gastrointestinal systems. Particle size has a major impact on intestinal absorption. Smaller particles, especially nanoplastics, show more potential for intestinal absorption, while larger particles are often eliminated by stool. Particle translocation across the intestinal epithelium may be aided by specialized intestinal cells like M cells found in Peyer's patches [37]. Diffusion via damaged epithelial barriers, endocytosis, phagocytosis, and micropinocytosis are some of the processes for cellular uptake that have been suggested. Cellular interactions and absorption efficiency may also be impacted by surface changes brought on by environmental weathering or protein adsorption [38]. According to distribution studies conducted on experimental animal models, absorbed particles might aggregate in different organs and enter the systemic circulation. The liver, spleen, kidneys, lungs, intestines, heart, and brain have all been found to have microplastics. Particle properties and exposure time determine accumulation patterns [39]. Because of its function in blood filtration and detoxification, the liver is regarded as a primary target organ. Microplastic buildup in the liver has been linked to oxidative stress, aberrant lipid metabolism, inflammation, and mitochondrial malfunction. Similarly, nephrotoxicity and reduced filtration capacity may result from kidney buildup [40]. The metabolism of microplastics is still poorly known due to the remarkable resistance of synthetic polymers to enzymatic breakdown. However, in biological settings, particles might experience physicochemical changes. Protein corona formation on particle surfaces can alter toxicity, biodistribution, and cellular recognition. Environmental aging processes may also improve reactivity and alter surface chemistry. The processes involved in excretion are not fully understood. Smaller particles can be expelled by bile or urine, but larger particles are mostly removed through fecal excretion. Additionally, microplastics serve as carriers of harmful substances. Their surfaces absorb pesticides, endocrine-disrupting substances, heavy metals, and persistent organic pollutants. These pollutants may desorb and contribute to compounded toxicological effects once they enter biological systems. The biological intricacy of microplastic exposure is greatly increased by this carrier characteristic [41]. According to recent studies, nanoplastics may have more toxicological potential than bigger particles due to their improved cellular penetration and bioavailability. Significant cellular dysfunction may arise from their direct interaction with intracellular organelles like nuclei and mitochondria. In general, toxicokinetic research indicates that microplastics may accumulate and spread throughout the human body. However, there are still a lot of unanswered questions about dose-response relationships, tissue persistence, elimination rates, and chronic exposure. Accurate risk assessment and comprehension of long-term health repercussions require further human research [42].

5.CELLULAR AND MOLECULAR MECHANISMS OF TOXICITY

Through a variety of interrelated cellular and molecular processes, microplastics have toxicological effects. According to available data, these particles actively interact with cells, tissues, and metabolic processes rather than being physiologically inert. Particle size, concentration, polymer composition, shape, surface properties, and related chemical pollutants all affect their potential for toxicity. One of the main processes thought to underlie the damage caused by microplastics is oxidative stress. Microplastic exposure increases the production of reactive oxygen species, which overwhelms the body's natural antioxidant defense mechanisms, including glutathione peroxidase, catalase, and superoxide dismutase. Lipid peroxidation, protein oxidation, mitochondrial malfunction, and DNA damage are all consequences of increased oxidative stress [43].

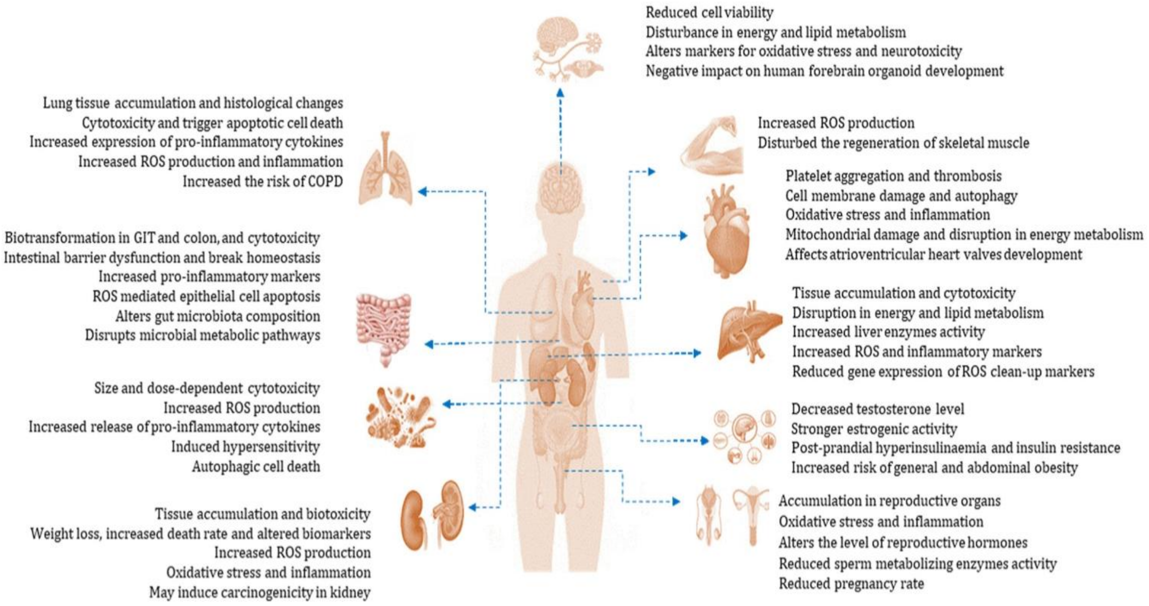

FIG-2 Potential effects of MNPs on different organ systems in humans [44].

Because oxidative stress interferes with electron transport chains and ATP synthesis, mitochondria are especially susceptible. Damage to the mitochondrial membrane may cause cytochrome c to be released and apoptotic pathways to be activated. Cellular deterioration and chronic inflammation are caused by persistent mitochondrial malfunction. Another significant mechanism of toxicity is inflammation [45]. Tumor necrosis factor-alpha, interleukin-6, and interleukin-1 beta are among the pro-inflammatory cytokines that are produced when immune cells including macrophages, neutrophils, and dendritic cells are activated by microplastics. Immune responses are further enhanced by activation of inflammatory signaling pathways, such as MAPK and NF-κB. Tissue damage, fibrosis, and altered immune modulation can result from persistent inflammation. Continuous inflammatory reactions in respiratory tissues may lead to pulmonary fibrosis and airway remodeling. In a similar vein, intestinal inflammation may damage the integrity of the epithelial barrier and upset gut homeostasis [46]. Another mechanism linked to microplastic toxicity is autophagy dysregulation. Exposure may either excessively activate or inhibit autophagic pathways, resulting in the accumulation of damaged proteins and organelles, which contributes to cellular stress and degeneration. Adsorption of environmental pollutants onto microplastic surfaces further intensifies toxicity; combined exposure to microplastics and associated chemicals may produce synergistic effects that exceed toxicity caused by individual contaminants alone [47]. Taken together, these molecular mechanisms show the complexity of microplastic toxicity. Damaged proteins and organelles may accumulate as a result of exposure that either overly activates or inhibits autophagic mechanisms. Cellular stress and degeneration are exacerbated by this. Toxicity is increased when environmental contaminants adhere to microplastic surfaces. The synergistic effects of combined exposure to microplastics and related compounds may outweigh the toxicity of individual pollutants. When taken as a whole, these molecular processes show how complex microplastic toxicity is. To ascertain dose-response correlations, long-term exposure effects, and significance to the development of human diseases, more research is required [48].

6.ORGAN-SPECIFIC TOXICITY

Microplastics have the ability to damage a variety of organ systems after being absorbed and distributed systemically. According to experimental investigations, particle size, exposure time, concentration, and related pollutants all have an impact on toxicity. Although much of the present information comes from animal and in vitro studies, the increased discovery of microplastics in human tissues raises serious concerns about organ-specific health effects [49]. The gastrointestinal tract is regarded as the principal site of exposure and toxicity since ingestion is a key exposure pathway. Microplastics interact directly with intestinal epithelial cells, altering tight junction proteins and jeopardizing barrier integrity. Increased intestinal permeability allows particles and bacterial endotoxins to enter the bloodstream more easily [50]. Exposure also affects the gut microbiome makeup, lowering beneficial bacterial populations and promoting dysbiosis. These microbial alterations affect immunological responses, food metabolism, and inflammatory pathways. Chronic intestinal inflammation caused by microplastics may be linked to inflammatory bowel illnesses and metabolic problems. Airborne microplastic exposure causes respiratory toxicity, which is becoming more widely recognized. Depending on their aerodynamic qualities, inhaled particles may deposit in the bronchi and alveoli. Fibrous microplastics are especially problematic since they are structurally similar to asbestos fibres [51]. Reproductive toxicity has received significant scientific study. Animal studies show that exposure leads to decreased spermatogenesis, lower sperm quality, ovarian dysfunction, hormonal imbalance, and altered reproductive behaviour. The presence of microplastics in placental tissue suggests possible foetal exposure during pregnancy [52]. Developmental toxicity is especially concerning since the prenatal and neonatal phases are extremely vulnerable to environmental toxins. Placental transfer may disrupt foetal growth, neurodevelopment, endocrine function, and immune system maturation. Neurological toxicity has emerged as a significant study topic. Nano plastics can pass the blood-brain barrier and accumulate in neural tissues. Experimental research suggests neuroinflammation, oxidative stress, neurotransmitter imbalance, and behavioural changes.

Long-term neurotoxic consequences are poorly known; however, they may contribute to neurodegenerative diseases [53]. Another significant side effect of exposure is immune system malfunction. Chronic activation of inflammatory pathways may impair immunological control, increasing susceptibility to infections, allergies, and autoimmune diseases. The multisystem character of microplastic toxicity highlights the necessity for a thorough assessment of chronic exposure concerns. Although clear human evidence is still sparse, available research strongly imply that chronic exposure may contribute to broad physiological dysfunction [54].

7. COMPARATIVE ANALYSIS OF RECENT STUDIES

Recent scientific investigations have significantly increased our understanding of microplastics and their implications for human health. Studies conducted between 2018 and 2025 consistently show broad environmental contamination, increased human exposure, and a variety of toxicological impacts. However, studies vary significantly in terms of methodology, particle characterisation, exposure concentrations, and biological models. In vitro investigations offer valuable mechanistic insights into cellular toxicity. Human epithelial, macrophage, hepatocyte, and neuronal cells exposed to microplastics exhibit oxidative stress, inflammatory cytokine release, mitochondrial malfunction, apoptosis, and DNA damage. These findings indicate the possibility of direct cellular damage [55].Nonetheless, many in vitro research use substantially higher exposure concentrations than those observed in real-world circumstances. This raises concerns regarding extrapolation of findings to human exposure scenarios. Furthermore, cell culture systems lack the complexity of whole organisms and may not accurately represent chronic low-dose exposure. Animal studies provide evidence of systemic distribution and organ-specific toxicity. Rodent models exposed to polystyrene microplastics exhibit hepatic inflammation, gut microbiota disruption, reproductive toxicity, neurobehavioral alterations, and metabolic disturbances. Zebrafish studies additionally demonstrate developmental toxicity and oxidative stress [56]. Detecting microplastics in blood, placenta, lung tissues, breast milk, and feces confirms internal human exposure, but species differences complicate direct translation to humans due to differences in metabolism, exposure routes, and immune responses. A landmark study published in this area confirms this. Toxicological effects may be influenced by variations in metabolism, exposure pathways, and immunological responses [57]. Human biomonitoring studies have lately emerged as a significant research topic. Microplastics found in blood, placenta, lung tissues, breast milk, and feces indicate that humans have been exposed internally. A landmark study published in 2022 identified microplastics in human blood samples, providing strong evidence for systemic circulation. Placental investigations show particles moving from maternal circulation to fetal tissues, increasing worries about prenatal exposure. However, existing human investigations are restricted by small sample sizes and the absence of established analytical procedures [58]. Another difficulty is inconsistency in particle characterisation. The polymer kinds, particle sizes, shapes, and concentrations used in experiments differ between studies. Polystyrene particles are most frequently investigated because of experimental convenience, but environmental exposure involves highly heterogeneous mixtures. Analytical limitations also complicate interpretation. Detection and quantification of microplastics require advanced spectroscopic techniques such as Fourier-transform infrared spectroscopy and Raman spectroscopy. Variability in sampling procedures and contamination control further affects study reliability. Overall, current research firmly supports the health hazards associated with chronic microplastic exposure. However, future studies must focus on environmentally relevant concentrations, long-term exposure assessment, standardized methodologies, and epidemiological investigations to establish definitive causal relationships [59].

8. KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite advances in microplastics research, there are still uncertainties about their long-term impact on human health. While evidence suggests potential toxicity, methodological and conceptual limitations hinder accurate risk assessment and regulatory decision-making. One major challenge is the lack of standardized analytical methods for detecting and quantifying microplastics in biological and environmental samples. Existing evidence strongly suggests potential toxicity, yet several methodological and conceptual limitations hinder accurate risk assessment and regulatory decision-making [60]. One of the most significant challenges is the lack of standardized analytical methods for detection and quantification of microplastics in biological and environmental samples. Variability in sampling procedures, extraction methods, contamination control, and spectroscopic analysis contributes to inconsistent findings across studies. Another significant drawback is the poor categorization of ecologically relevant exposure situations. Many experimental investigations use homogeneous polystyrene particles at high concentrations, which may not adequately represent real-world exposure. Environmental microplastics vary greatly in terms of size, polymer content, form, weathering condition, and related pollutants [61]. The health effects of Nano plastics are especially unknown. Because of their incredibly small size, Nano plastics may display unusual biological behaviour such as increased cellular penetration, intracellular accumulation, and interaction with subcellular organelles. However, technical constraints in detection make their investigation particularly difficult. Another major research field is the interaction of microplastics with co-contaminants. Heavy metals, antibiotics, endocrine disruptors, and persistent organic pollutants are all often adsorbed by environmental particles. Combined exposure may result in synergistic toxicological consequences that differ significantly from isolated particle exposure. The function of gut microbiota disturbance in disease development warrants additional exploration. Microbial composition changes can affect immunological regulation, metabolism, and neurobehavioral function via gut-brain connections [62]. Future studies should identify reliable biomarkers of exposure and toxicity for biomonitoring, early detection, and risk assessment. Advanced omics technologies such as genomics, proteomics, metabolomics, and transcriptomics can provide deeper insights into molecular mechanisms. Many countries lack adequate regulatory frameworks to address microplastic contamination, so international collaboration is crucial. Reliable biomarkers would help with biomonitoring, early identification, and risk assessment. Advanced omics technologies such as genomics, proteomics, metabolomics, and transcriptomics have the potential to deliver deeper insights into molecular pathways [63]. Many countries still lack adequate regulatory frameworks for dealing with microplastic contamination. International collaboration is necessary for developing common norms for plastic production, waste management, environmental monitoring, and human exposure limits [64]. Biodegradable alternatives, improved recycling technologies, reductions in single-use plastics, and public awareness campaigns are all vital preventive measures. Furthermore, interdisciplinary collaboration among toxicologists, environmental scientists, doctors, epidemiologists, and politicians will be required to address this global health issue. Overall, future research must shift from exploratory to complete human risk assessment methodologies that may influence evidence-based policy and public health initiatives [65].

9. CONCLUSION

Microplastics have developed as a widespread environmental pollutant, with serious consequences for human health. Their broad distribution in aquatic, terrestrial, and atmospheric ecosystems has resulted in ongoing human exposure by ingestion, inhalation, and possibly skin absorption. Recent scientific evidence shows the presence of microplastics in a variety of human tissues and biological fluids, demonstrating their capacity to penetrate the bloodstream and interact with important organs [66].

The toxicological impact of microplastics goes beyond their physical existence. These particles cause oxidative stress, inflammatory reactions, mitochondrial dysfunction, endocrine disruption, genotoxicity, and changes in the gut flora. Their position as transporters of dangerous environmental toxins exacerbates potential health hazards [67]. Smaller particles, particularly nanoplastics, have a higher biological reactivity and the ability to penetrate tissue, indicating that chronic exposure may contribute to long-term physiological effects. Experiments consistently show that organ-specific toxicity affects the gastrointestinal, respiratory, hepatic, renal, cardiovascular, reproductive, and nervous systems. Smaller particles, especially nanoplastics, exhibit enhanced biological reactivity and greater capacity for tissue penetration. Although definitive epidemiological evidence in humans remains limited, current findings strongly indicate that chronic exposure may contribute to long-term physiological dysfunction [68]. Several critical knowledge gaps continue to challenge risk assessment efforts. Lack of standardized methodologies, limited long-term human studies, variability in particle characteristics, and insufficient understanding of chronic low-dose exposure complicate interpretation of available data. Nevertheless, the growing body of evidence emphasizes the urgent need for preventive strategies and regulatory action [69]. Addressing the worldwide burden of microplastic contamination would necessitate concerted international efforts to reduce plastic manufacturing, improve waste management systems, improve recycling technology, and create safer alternatives to traditional plastics. Public understanding and policy implementation are both critical for minimizing environmental contamination and limiting human exposure. Future research should focus on epidemiological investigations, ecologically appropriate exposure models, biomarker development, and mechanistic studies including Nano plastics. Understanding the entire scope of microplastic-related health concerns will need interdisciplinary collaboration among environmental scientists, toxicologists, doctors, politicians, and public health experts [70].

In conclusion, microplastics pose an emerging and potentially significant threat to human health. While many uncertainties remain, the available evidence clearly demonstrates that these invisible pollutants can interact with biological systems in complex and harmful ways. Immediate scientific, regulatory, and societal action is required to mitigate exposure and protect future generations from the long-term consequences of plastic pollution. While many questions remain, available information clearly shows that these invisible pollutants can interact with biological systems in complex and damaging ways. Immediate scientific, legislative, and societal action is required to reduce exposure and shield future generations from the long-term effects of plastic pollution [71]. Furthermore, microplastics act as vectors for a wide range of environmental contaminants, including persistent organic pollutants (POPs), heavy metals, and microbial pathogens. This "Trojan horse" effect amplifies their toxicity and introduces complex interactions that are yet fully understood. Given the chronic nature of exposure and the potential for bioaccumulation, there is an urgent need to evaluate their long-term impact on human health through an integrated approach. This "Trojan horse" effect increases their toxicity and introduces intricate interactions that are still not fully understood. Given the chronic nature of exposure and the possibility of bioaccumulation, there is an urgent need to assess the long-term impact on human health using an integrated toxicology paradigm [72].

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