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A Critical Review on Effect of Macro, Micro and Nanoplastics Pollution on Human Health and their Control Measures

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Abstract: Plastics have become an integral part of human life and have been used for multiple purposes in day-to-day life worldwide. Polyethylene (PE), polypropylene (PP), polystyrene (PS), polyvinyl chloride (PVC), and polyethylene terephthalate (PET) are some of the commonly used plastics. Plastics are reused in many underdeveloped and developing countries without realizing the toxicity associated with their repeated use. There are single-use plastics (SUPs) designed to be used only once and then need to be discarded or recycled. Many substances were used while manufacturing plastics, including solvents and non-intentionally added substances (NIAS), such as impurities, oligomers, or degradation products, accounting for more than 10,259 chemicals. Plastics have high durability and extensive half-life with slow degradability. Large amounts of plastic accumulate in various environments, soil, water, and ecosystems due to improper disposal methods and poor recycling rates, affecting the food chain and living systems and causing severe health hazards to humans. The presence of plastics in body fluids, tissues, and organs and their toxicity causes severe damage to the organ systems, leading to several disease conditions. If food items are stored in plastics, chemicals are migrating to food items stored/ packed. It depends on the nature and complexity of the food material, contact time, temperature of the system, type of packaging contact layer, and properties of the migrants. Microbial bioremediation is considered to be more effective in controlling plastic pollution than conventional methods. Bacteria, fungi, and algae, individually or consortia of different species, are proven to be very effective in degrading different plastics.

Keywords: micro-nanoplastics; environmental pollution; aquatic pollution; biodegradation; human health hazards.

1. Introduction

Approximately 370 million tonnes of plastic waste are produced yearly [1]. Plastic for packaging is 35%, construction is 17%, textile is 15%, institutional use is 10%, and transportation is 7% [2]. Plastics are discarded in landfills, incinerated, or gathered in seas, waterways, and soils as plastic litter [3]. Most developing nations still follow the landfill approach for disposing of synthetic plastics and incineration, which emits greenhouse gases such as carbon dioxide and methane [4]. It was reported that about 12,000 metric tons of plastic waste are supposed to enter landfills or the natural environment by 2050 [5].

Plastic waste deposition into oceans, lakes, and rivers has been calculated to be 9 and 23 million tons per year, and around 13 to 25 million tons per year are released into soil environments [6]. Plastics can enter the soil in various ways, including landfills, agricultural irrigation, composting, and agricultural mulch use [7]. An increased plastic load in soil enters the food web, affecting the ecosystem. Synthetic plastics are classified as macroplastic (≥5 mm in diameter), microplastic (MP, <5 mm in diameter), and nanoplastic (1 to 100 nm in size). Single-use plastics (SUPs) are extensively used for packaging, takeaway containers, disposable razors, and hotel toiletries. Synthetic plastics are made of non-renewable petroleum and natural gas resources.

2. Microplastics

Plastics are mostly discarded in the environment without proper recycling. As a result of environmental exposure, such as weathering, ultraviolet radiation, and physical wear and tear, waste plastics are converted to MPs through decomposition and volume reduction [8]. MPs exhibit adverse effects on the environment and the living systems than macroplastics. Under MPs, primary microplastic (PMP) and secondary microplastic (SMP) are used. PMP fragments or particles are directly released into the environment. PMPs are also released from plastic processing industries. MPs are also released during textile washing, 0.21–72,000 particles/g textiles per wash, and the range of MPs released was 165–841 μm has been reported [9]. Textile cutting using a scissor released more particles (3-21-fold higher) than the laser cutting methods. PMPs are also released from personal care products and cosmetics [10]. MPs are also present in face cleansers (7.1%) and shower gels (2.2%), with particle sizes ranging from 60–700 μm. SMPs (200–1000 mm) are produced by natural degradation (fragmentation) due to environmental weather conditions. It accounts for 70–80% of all plastic particles released into the environment [11]. Secondary MPs (200–1000 mm) are formed from debris and plastic waste, including fishing nets, disposable plastic utilities, and numerous plastic materials [9]. Tiny plastics are produced for industrial requirements and added to cosmetics and toothpaste in certain amounts.

MPs are easily dispersed by wind, surface overflow, and ocean currents [12]. MPs on soil exhibit horizontal and vertical migration. Predatory activities in the food chain facilitate horizontal migration, the life activities of insects, and the wind [13]. Vertical migration is downward through water infiltration into groundwater. MPs are also widely spread across marine and freshwater systems [9]. The plastic contamination in the aquatic system may reach 900,000 tons/year globally [14]. MPs ' atmospheric transport is considered to be a major form of transport on a global scale [15]. MPs presence on Arctic snow cover is evident in MPs atmospheric transport to even remote areas.

Due to the recalcitrant nature of MPs, they can remain in the environment for hundreds or thousands of years. MPs are also present in the deep sea and polar environments. MPs are macromolecular polymers consisting of polyvinyl chloride (PVC), PE, polystyrene (PS), and polypropylene (PP). Due to their excessive presence and ecological hazards, MPs are identified as emerging environmental pollutants of the globe. The degradation products of MPs are also more toxic than MPs [16], affecting the global ecosystem [17].

3. Nanoplastics

Nanoplastics (NPs) are emerging environmental pollutants due to their small size and potential hazards to living organisms. NPs are exposed to organisms and transferred along the food chain, and their accumulation affects their growth, development, and reproduction. Several properties of NPs, such as surface charges, size, quantity, and duration of exposure, affect the biological effects of NPs. Surface-charged NPs affect the normal physiological function of cells. Due to their nano size, they can easily cross the cell membranes and affect cell function. In addition, chemical additives and other pollutants attached to NPs pose a greater threat to living systems than NPs. Human exposure, accumulation, interactions with cellular functions, and their toxicity have not been explored completely. In the marine environment, NPs are produced from MPs by the action of microorganisms under high salinity. NPs may be more dangerous than MPs due to their size and can penetrate more easily in all biological barriers [18]. The presence NPs in human blood has been reported recently [19, 20]. It also affects RBCs as well [21]

4. Impact of plastics on humans

4.1. Exposure routes of MPs and NPs.

Inhalation and oral intake are the major routes of entry of MPs and NPs into the human body. It was estimated that 35,000–62,000 MPs may enter the human body annually and accumulate in the lungs through breathing. An individual inhale up to 130 MPs per day [22]. MNPs in alveoli, alveolar ducts, and terminal bronchioles can cause chronic inflammation and fibrosis [23,24]. The presence of MPs in human pulmonary tissue [25] and sputum samples [26] has been reported. The severity depends on the dose of MNPs and the period of exposure. MNPs reach the respiratory epithelium and translocate through diffusion, cellular penetration, or cellular uptake [26].

4.2. Sources of MPs and NPs.

Fibers and microbeads present in personal care products are sources for MNPs' exposure to skin [27]. On the other hand, 39,000–52,000 MPs and NPs enter the human body through diet and drinking water [28]. The presence of MPs in table salt (1–10 MPs/kg) [29] milk [30] has also been reported. The presence of MPs/NPs in a variety of edible substances, including seafood, honey, salt, sugar, and beer, has been reported [31]. Tests on tap and bottled water have also revealed the widespread presence of MPs and NPs in drinking water [32]. These results suggested that the food chain may be humans' main source of MPs/NPs ingestion. Large amounts of NPs are released into the atmosphere in urban areas through vapor-containing plastic waste [33]. MPs and NPs are emerging sources of air pollution transport to remote regions through the atmosphere [34]. They remain in the air for a long time due to their small size and enter the respiratory tract through inhalation [35]. Because of their presence in soil, water and wastewater easily enter aquatic animals through the food chain [36]. MPs and NPs in seafood meant for human consumption were also reported [37]. MPs/NPs exposure and accumulation in humans affect cellular function and metabolism.

4.3. Impact of MPs and NPs on GI tract.

MPs/NPs translocate from the digestive tract to other tissues by cellular internalization by forming an eco-corona on the surface of MPs [38]. MPs/NPs (50 and 500 nm) environmental exposure enhanced cellular internalization by passive membrane penetration and active endocytosis [39]. MPs/NPs exposure induces reactive oxygen species (ROS) formation and oxidative stress-mediated mitochondrial damage [40]. ROS-mediated changes in gut microbiota can lead to intestinal inflammation and intestinal epithelial cell apoptosis, affecting intestinal transport [41]. Gut microbiota is important for the digestion of dietary fiber, biosynthesis of vitamins, and protection against pathogenic bacteria. The changes in microbiota alter the immune homeostasis, metabolism, and integrity of the intestinal barrier, leading to the development of intestinal bowel disease (IBD) [42].

4.4. Impact of MPs and NPs on Heart and liver

MNPs also affect cardiovascular cells and tissues due to their entry into the bloodstream [43] MNPs exposure causes vascular endothelial damage by disrupting endothelial growth factors and interaction among endothelial cells, blood, and immune cells, leading to endothelial dysfunction and inflammatory responses. MPs accumulation in the liver causes chronic liver diseases. MPs/NPs accumulation also induces hepatic lipid accumulation, inflammation, and activation of macrophages, leading to hepatic apoptosis and fibrosis. MNPs are responsible for causing inflammatory and oxidative stress-mediated liver injury. Particles on the surface of MPs/NPs are responsible for causing physical subcellular injury after internalization [44]. ROS accumulation and prolonged oxidative stress result in antioxidant depletion and DNA damage, the basis for numerous diseases [45]. MPs/NPs-mediated lipid alteration, metabolic dysfunction, and accumulation in the liver affect the expression of genes involved in critical metabolic pathways [46]. MPs/NPs accumulation causes hepatocyte apoptosis, hepatic inflammation, fibrosis, and even cirrhosis [47].

4.5. Impact of MPs and NPs on the nervous system and kidney.

MNPs are mainly excreted by the liver via phagocytosis or biliary excretion in feces and through the kidneys; the presence of MPs and their biodegradation products in urine has been reported recently [48,49]. MNPs are also associated with reproductive and developmental toxicity [50, 51]. MPs and MNPs can cause severe neurotoxicity by affecting acetylcholine, γ-aminobutyric acid, and glutamate [52]. Abnormal AChE activity leads to acetylcholine accumulation and associated neurological disorders [53]. The impact of MPs and MNPs on various human organs and associated disease conditions are shown in Figure 1.

4.6. Impact of MPs and NPs on immune function.

Exposure of MPs and MNPs to immune cells causes significant changes in various transcriptional levels, including enzyme levels and the release of cytokines. MPs have been shown to trigger the immune response by producing pro-inflammatory cytokines such as IL-6, TNF alpha, and histamine in the intestine, lung, and brain [54]. Bisphenol A (BPA) and phthalates present in MPs are reported to be endocrine-disrupting compounds affecting endocrine function. MPs and MNPs also affect the muscular system and muscle function. Thus, MPs and MNPs exposure causes membrane disruption, hindrance of cell pores, ROS production, DNA damage, lysosome destabilization, and mitochondrial depolarization [55].

After entry, MNPs first contact with the lungs, followed by GIT systems, cross the biological barriers, thereby entering multiple organs and leading to systemic toxicity. Several researchers have reported the presence of MNPs in human body fluids, tissues, and various organs in Table 1.

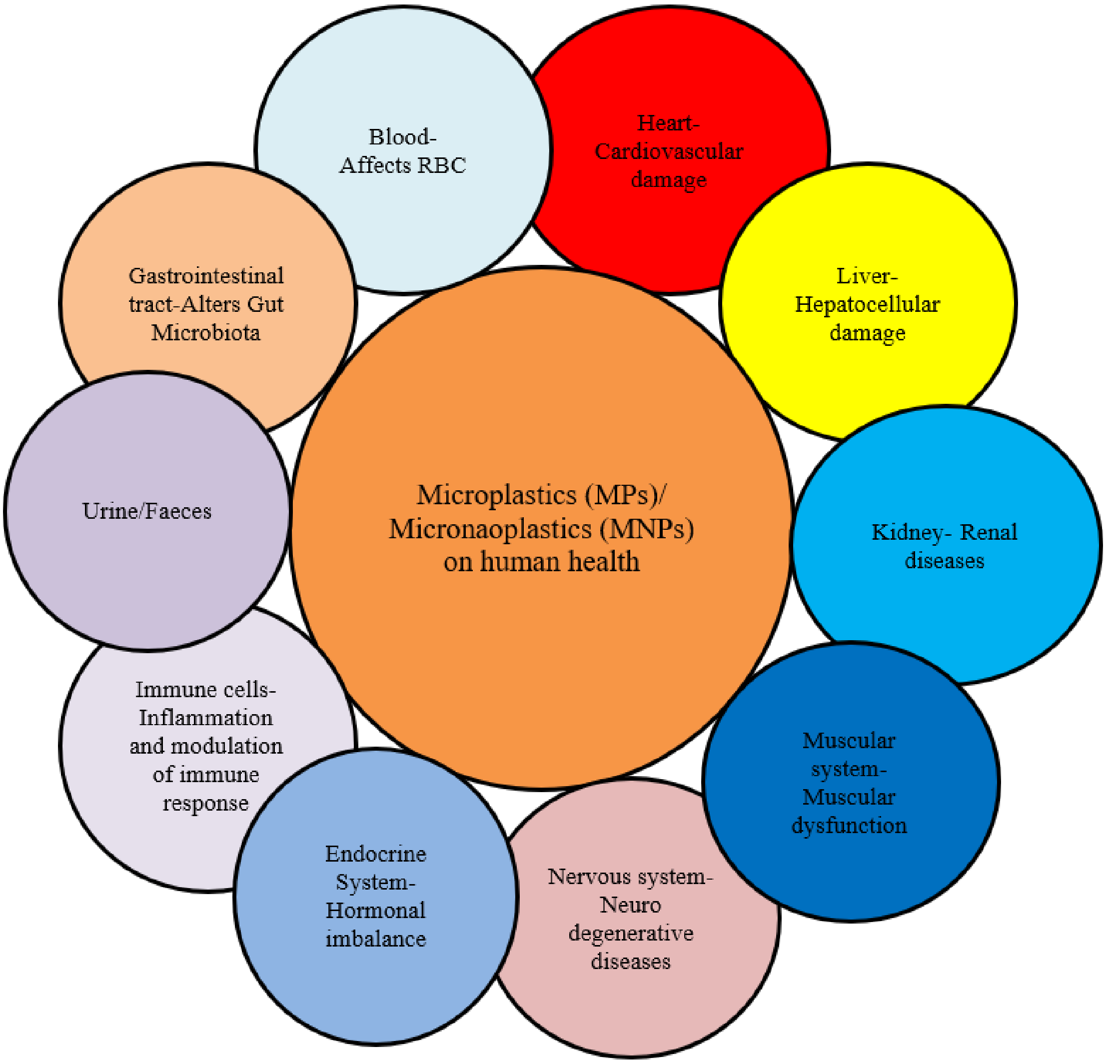

Figure 1. Effect of microplastics and microplastics on various organs of humans and associated disease conditions.

Table 1. Micro and nanoplastics present in human tissues, organs, and samples.

Human organs and samples	**Type of MNPs plastics**	**References**
Sputum	Polyurethane	[24]
Blood	Polystyrene	[56]
Urine	Polyvinyl chloride, Polypropylene and Polyethylene	[57]
Feces	Polypropylene (PP) and Polyethylene terephthalate (PET)	[58]
Muscles	Polypropylene and Polyethylene	[59]
Brain	Polystyrene	[60]
Lung	polyethylene and polypropylene	[25]
Intestine	Polystyrene	[61,62]
Heart	Polypropylene and Polystyrene	[63]
Liver	Polystyrene	[64]
Kidney	Polypropylene and Polyethylene	[57]
Placenta	Polypropylene	[65]

5. Natural microorganisms capable of degrading plastics

5.1. Bacteria.

Bacteria decompose plastics into low molecular weight compounds by forming biofilms and a series of enzymatic reactions [43]. *Pseudomonas* spp., *Bacillus* spp., and *Streptomyces* spp. are reported to be effective plastic-degrading bacteria [66]. The majority of plastic biodegrading organisms are bacteria [67]. Cold-tolerant bacteria are advantageous for degrading plastics due to lower energy requirements at lower temperatures. Salt-tolerant and thermophilic bacteria produce many enzymes that can degrade plastics for a longer time. *Bacillus velezensis* C5 forms biofilm on low-density polyethylene (LDPE) surface by reducing the water contact angle by 46° and increasing the hydrophilicity of LDPE films. It decreases the weight and thickness of the LDPE by 8.01 % and 26 %, respectively, after 90 days of incubation [68]. *B. paralicheniformis,* capable of degrading polystyrene (PS) films by 34 % within 60 days, was already been reported [69]. *Pseudomonas aeruginosa* WZH-4 and *P. aeruginosa* WGH-6 produce weight loss (9.35 % and 17.20 %) on PP particles [8]. A thermophilic bacterium *Brevibacillus agri* btDSCE02, capable of degrading high-density polyethylene (HDPE) strips by 24.66 % (in thickness) after 140 days at 50 °C [70].

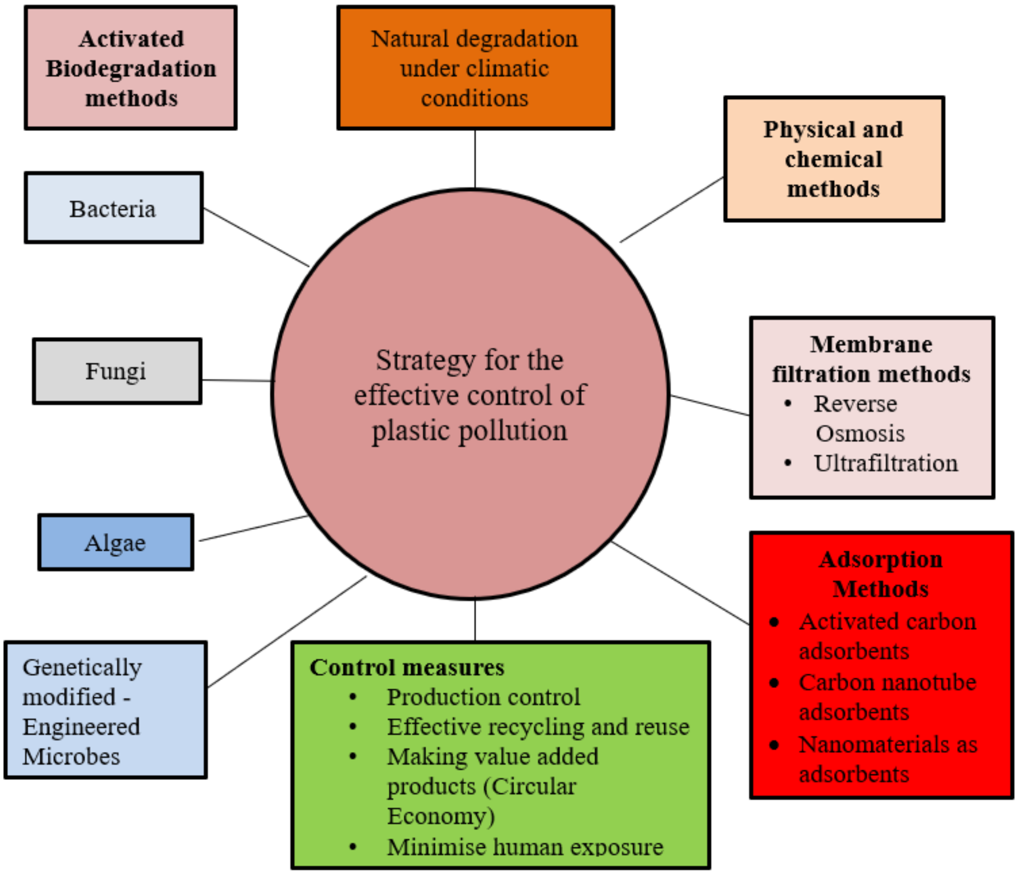

Figure 2. Strategies used for the effective control of plastic pollution and their exposure to humans.

5.2. Fungi.

Aspergillus spp. and *Penicillium* spp., are capable of degrading plastic polymers [58]. They penetrate the plastic surface through their mycelia and spread into the substrate to absorb nutrients, allowing fungi to attach firmly to the plastic surface [71]. Fungi can secret surface active protein hydrophobins, and another added advantage [72]. Fungal hydrophobins selfassembly forms amphiphilic protein membranes, bringing hydrophilicity to the plastic surface, facilitating their colonization on the polymer surface [73]. Intracellular enzymatic systems, cytochrome P450 family, epoxidases, and transferases help detoxify exogenous toxic compounds by fungi and thereby adapt to environmental conditions for the degradation of plastics [74]. The extracellular enzymes secreted by the fungal cells, such as peroxidases (manganese peroxidase, versatile peroxidase, and lignin peroxidase), laccases, cutinases, lipases, and protease [75], also play important roles in the degradation of plastics by depolymerizing polymeric materials into lower molecular weight fragments [76]. *Alternaria alternata* FB1 isolated from a marine environment showed high plastic degrading capacity [67]. It reduces the molecular weight of PE films (0.025 mm in thickness) by 95 % after 120 days of co-incubation and also forms holes (0.5 μm) in PE plastics. *P. citrinum* CF-3 isolated from the plastic-contaminated soil showed weight reduction of LDPE pieces (51 μm in thickness) by 38.82 % within 90 days and the presence of CF-3 extracellular lipase, esterase, laccase, and manganese peroxidase [77]. The degradation of plastics by fungi depends on the adsorption capacity, the production of surfactants, and the presence of abundant enzyme systems. The different strategies followed for effectively controlling plastic pollution and its exposure to humans are shown in Figure 2.

5.3. Algae.

Algae attach to plastic surfaces (e.g., exopolysaccharides) and produce depolymerizing enzymes (e.g., ligninolytic enzymes) for biodegradation [78]. *Spirulina* sp. degrade the PET (0.25–1 mm) after14-days co-incubation under saline conditions [79]. *Phormidium lucidum* and *Oscillatoria subbrevis* degrade PE strips and remove MPs in aqueous medium by heteroaggregation [80]. *Scenedesmus abundans* degrade PSMP (2 μmin diameter) to 84% in 9 h [81]. Microalgae's surface characters and MPs or NPs' surface charge are responsible for attachment and degradation [82]. Effective degradation of LDPE by *Anabaena spiroides* [83] and 20–500 nm PS particles by *Pseudokirchneriella subcapitata* [84]. Microalgae-associated hydrocarbon-degrading bacteria and diatoms are reported to be responsible for plastic degradation [85].

6. Engineered microorganisms for plastic degradation

The application of modern biotechnology tools, such as gene editing and genetic engineering techniques, is more promising and effective for the genetic modification of plastic degradation organisms. Microorganisms are engineered to produce more degradative enzymes by expressing introduced exogenous genes. Thermophilic cutinase (LCC) produced from engineered *Clostridium thermocellum* showed 62 % degradation of PET film (250 μm in thickness) into soluble monomer at 60 °C by 14 days [86]. The engineered DuraPETase-4M was more effective in degrading the amorphous PET powder and PET pre-formed film with increased thermal stability and biodegradability. The FAST-PETases (PETaseS121E/D186H/R224Q/N233K/R280A) obtained by using a machine learning algorithm of hydrolases, capable of degrading 51 different types of plastic bottles into simple monomers within 24 h at 50 °C [87]. Besides microbial degradation, green technologies (recycling and reusing plastic products) need to be developed to minimize the release of plastic pollutants and protect humans from plastic exposure associated with pathophysiological disease conditions.

7. Future prospects

Even though MPs and MNPs are known to be toxic to humans and other living systems, they are still widely used and disposed of improperly in the environment. Another major drawback is the lack of effective methods for accurately detecting and quantifying MPs, MNPs, and their products from human and other environmental samples. Several plastic types and their thickness, physical, and chemical properties remain major challenges in making a standard procedure for their effective disposal. Ban on plastic production, controlled use and proper disposal, and looking for biopolymer-based alternatives would certainly save the human race from plastic poisoning.

8. Conclusions

Growing evidence supports the toxicity of plastics to various human body organs, but it was limited to only certain plastic types. More detailed toxicological studies are needed using different types of plastics with short- and long-term effects on humans and the underlying disease process. As the use of plastics is increasing every day, it increases the chances of exposure to humans through multiple routes, as evidenced by recent reports on the detection of MPs and MNPs in human organs and blood, and their presence in urine and feces warranted a stringent law and strict enforcement on use and disposal plastics to save the humans from plastics associated toxicity and diseases. Therefore, a multi-disciplinary approach has to be followed on a war footing to ascertain the plastic-associated human health risk on a long-term basis.

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Conflicts of Interest

The authors declare no conflict of interest.

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