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Review

The Impact of Micro- and Nanoplastics on Aquatic Organisms: Mechanisms of Oxidative Stress and Implications for Human Health—A Review

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Abstract: In recent years, interest in the impact of environmental pollutants on the ecosystem has increased significantly, with particular attention being paid to the relationship between climate change and the aquatic world. This is because increasing pollution is causing fundamental changes to the welfare of animals and the marine ecosystem. A primary focus is on the impact of microplastics (MPs) and nanoplastics (NPs), as evidenced by our bibliometric network analysis (BNA). However, while research is focused on the accumulation of these pollutants in aquatic organisms, their effects on redox homeostasis are still seldom discussed despite the role played by reactive oxygen species and mitochondrial well-being in maintaining an optimal state of health. However, some scientific evidence suggests that the accumulation of MPs and NPs in organisms at the base of the trophic chain can cause a transfer of these substances towards more complex organisms, reaching humans through the consumption of aquatic fauna as food. Therefore, in this review, we have tried to discuss the effects of these substances on oxidative stress in aquatic organisms, even if studies in this regard are still scarce.

Keywords: microplastics; nanoplastics; oxidative stress; toxicity; trophic transfer

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1. Pollution and the Environment

The increase in the world population and, consequently, human activities, such as agricultural intensification, industrial development, and urbanization, have led to a sharp increase in waste production and environmental pollution [1,2]. Coastal, marine, and fluvial environments are constantly under pressure from anthropogenic release into the environment of heavy metals, hydrocarbons, pesticides, persistent organic pollutants (POPs), organophosphorus flame retardants (OPFRs), pharmaceuticals and personal care products (PPCPs), products of metallurgical processes, nanoparticles, plastics and microplastics, etc. [3–8]. Organisms living in the aquatic environment, such as plants and animals, can transfer pollutants to humans through bioaccumulation and trophic transfer, as well as exert harmful effects on them following their exposure [5,7]. In recent decades, there has been a rise in the emission of nanoparticles and heavy metals into the environment with the intensification of industrialization [9–11]. Nanoparticles are a group of materials that have a size between 1 and 1000 nm in diameter [12–14]. The nanoparticles are widespread in aquatic ecosystems through aerial deposition, effluent discharge, dumping, and run-off [11]. They can be divided into two groups: natural (i.e., desert dusts, aerosols, emissions from volcanic activities, etc.) and anthropogenic (i.e., metal oxides, drug production, burning

fossil fuels, vehicle exhaust release, mining demolition emissions, etc.) [11–13]. One of the most widespread nanoparticles in the aquatic environment is titanium dioxide (TiO_2), which the European Union reports as one of the main pollutants of surface waters, with a value of 2.2 μgL^{-1} [12]. Lovern and Klaper, showed an increase in the mortality rate of *D. magna* due to exposure to TiO_2 [14]. Heavy metals also pose a serious threat to the aquatic environment. Twenty-three metals and metalloids out of seventy present in the environment are identified as heavy metals/trace metals, some of which are considered dangerous [9]. Heavy metals can be divided into two categories: essential and non-essential. The non-essential metals (aluminum (Al), cadmium (Cd), mercury (Hg), tin (Sn), lead (Pb), etc.) are elements that have no known biological function and can be considered toxic at high concentrations. Essential metals (copper (Cu), zinc (Zn), chromium (Cr), nickel (Ni), cobalt (Co), molybdenum (Mo), and iron (Fe)) play a specific role in the body's metabolism and can be toxic both under metabolic deficiency conditions and at high concentrations [9,10,15]. Contaminants/heavy metals can have a negative impact on the physiology of aquatic organisms; fish especially are very sensitive to such changes [15]. The toxic effects of heavy metals on fish are numerous [16]. They enter the fish body via the gills, digestive tract, and body surface and accumulate in the liver, kidney, muscle, intestine, skin, and bones [16]. It is shown that in fish heavy metals can alter several physiological and biochemical processes, growth rates, mortality, and reproduction. Moreover, they can cause serious problems in the body by producing reactive oxygen species (ROS), which cause oxidative stress and DNA damage [17–19].

Another issue that has plagued the environment in recent decades is the spread of pharmaceuticals and personal care products (PPCPs) in aquatic environments. This group of pollutants includes several substances from different sources, mainly anti-inflammatory drugs, as well as antibiotics (antimicrobials or antibacterials), antiepileptics, and personal care products [20]. The increase in the consumption of PPCPs is caused by the average use in the use human population and the upward demand for animal protein [21–24]. These substances find their way into the environment, where they can negatively impact organisms in aquatic ecosystems. Antibiotics are the most widely used pharmaceuticals due to human and veterinary applications [25]. Environmental antibiotics are derived from different sources, such as wastewater treatment plants, hospitals, water from urban canals, agriculture (aquaculture, husbandry), and the pharmaceutical industry [26,27]. Recently, the use of antibiotics has increased with the rise in aquaculture; in fact, their use allows for the reduction in possible threats from microorganisms. However, on the other hand, antibiotics can promote the resistance of bacteria in aquaculture and lead to resistance even in wild fish populations [28]. They are continually discharged into the aquatic environment and are bioavailable for animals, crops, and aquatic plants [26–28]. Primary producers, like microalgae and decomposers, are more sensitive than crustaceans and fish to antibiotic agents. Consequently, antibiotics could negatively impact microbial processes and lead to variations in biogeochemical cycling and aquatic ecosystems [4,26].

Among the toxic pollutants with the greatest impact on the aquatic environment, we cannot fail to include pesticides [29–32]. Pesticides are important for the protection of crops from infection by many microorganisms and for eradicating pests and organisms, such as mice, rats, ticks, and mosquitoes, that are dangerous for the environment and the habitat of humans [30,32,33]. Among these, the best known and most discussed is DDT (1,1,1-trichloro-2,2-bis(4-chlorophenyl) ethane) [30,33]. However, due to its high toxicity, it has been banned in many Western countries since the 1970s [33]. Due to its ecotoxic effects, bioaccumulation, and environmental persistence, it was also considered by the international agreement of the Stockholm Convention 2001 (World Health Organization, 2011) as one of the world's 12 restricted persistent organic pollutants [34]. Nevertheless, the World Health Organization controlling vector-borne diseases allowed it in certain tropical countries in 2006 [30]. DDT can land many issues for aquatic organisms. It is so hydrophobic that it is absorbed into organic sediment particles, where it can persist for many years. In the aquatic environment, it can accumulate in benthic organisms and

subsequently be transferred to higher trophic levels through biomagnification [30]. Owens e Baer, 2000, revealed a dose-dependent increase in the lethality of the Japanese medaka (*Oryzias latipes*) exposed to DDT [35], while Ton et al., 2006, showed the teratogenic effects on zebrafish (*Danio Rerio*) larvae exposed to the organochlorine DDT [36].

Nowadays, new pollutants are causing harm in aquatic environments and beyond, namely micro- and nanoplastics [4–6,37,38]. Figure 1 shows how microplastic pollution has recently become one of the main topics in studies on the aquatic environmental stress. Figure 1 has been generated using the Scopus database and the software Vosviewer (version 1.6.19) which allows for the creation, viewing, and exploration of maps based on bibliometric network data [39]. Scopus database provided 3235 scientific documents published from 1976 to 2022 by selecting (Aquatic environment*) AND ("Oxidative stress" OR "ROS" OR "REDOX") AND ("Pollution*" OR "Pollutant*") as keywords.

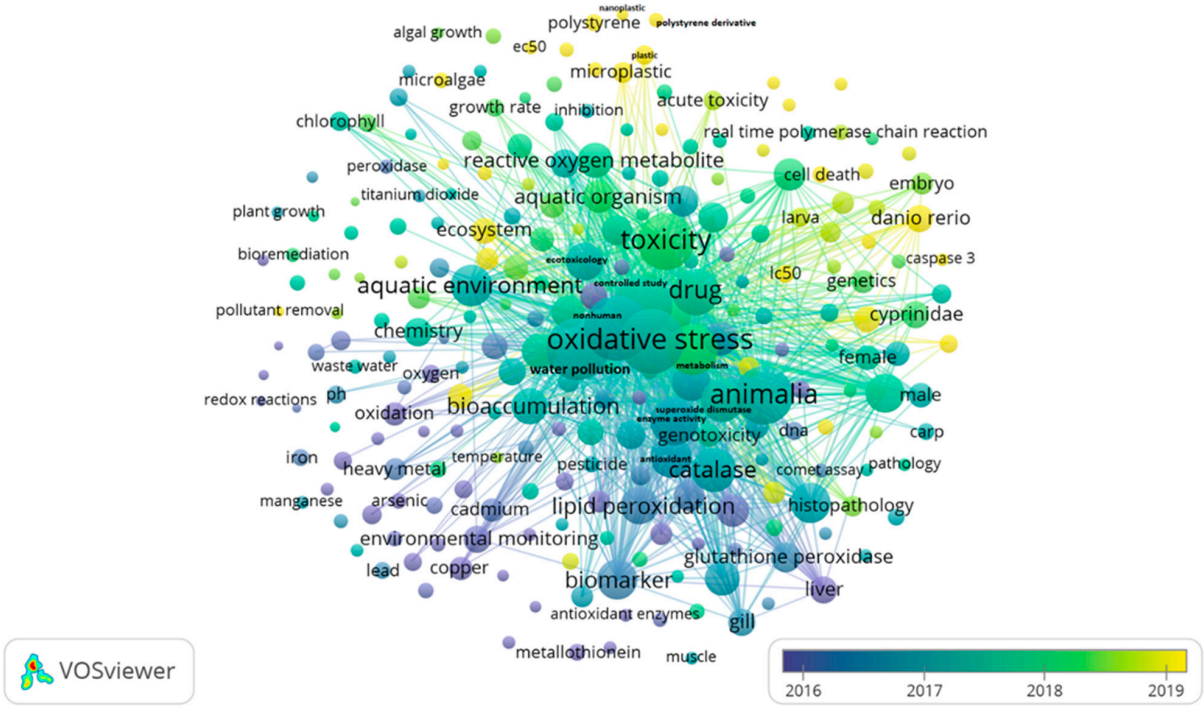

Figure 1. Bibliometric network analysis.

2. Micro- and Nanoplastics

Since the 1950s, plastic production has soared to a value of 322 million tons in 2015, and projections indicate it could exceed one billion tons by 2050 [40]. Microplastic pollution is widespread in every environment, from the atmosphere to the ocean depths [41,42]. Plastic makes up about 80 to 85% of marine litter, and it is estimated that an average of 5 to 13 million tons of plastic enter the oceans each year [6,37]. For example, around 229,000 tons of plastic are released every year in the Mediterranean Sea [43], which appears to be one of the areas with the highest concentration of floating debris [44]. This is due to the characteristics of the Mediterranean Sea, such as its semi-enclosed conformation and the general anti-estuarine circulation, which favors the accumulation of floating material [44].

3. Characteristics and Sources

Plastic materials are made from a wide variety of synthetic or semisynthetic organic compounds, such as polyethylene (PE), polyethylene terephthalate (PET), polystyrene (PS), polypropylene (PP), and polyvinyl chloride (PVC) [37,38,45]. Due to low end-of-life recycling, PVC is a very abundant contaminant in the aquatic environment. It has been reported that only 3% of PVC is recycled, while 82% is discarded in landfills, and 15% is incinerated [45]. Plastic materials may persist in the environment thanks to their high

ratio of aromatic compounds and consequent resistance to degradation by microorganisms [46]. Once released into the environment, plastic breaks down into smaller pieces called microplastics (MPs) and nanoplastics (NPs, <1 µm) due to the action of various environmental factors, such as ultraviolet (UV) radiation, mechanical abrasion, hydrolysis and biodegradation [5,6,37,47].

Microplastics can be divided into primary and secondary. These latter MPs, resulting from the fragmentation of larger plastic (e.g., plastic bags, food packaging, ropes, etc.), are tiny plastic particles with a size of less than 5 mm and are the main source of MPs in the aquatic environment [48,49]. Instead, primary MPs are not derived from the degradation of larger plastic objects but are manufactured directly in microscopic sizes, including products for industrial or domestic applications entering aquatic systems through wastewater [37,48,50]. Over time, microplastics can become even smaller, fragmenting and giving rise to nanoplastics, such as those recently detected in the seawater below the North Atlantic sub-tropical gyre [51].

Like microplastics, nanoplastics are also classified into primary and secondary sources. Primary NPs are plastic nanoparticles manufactured and released directly into the environment. Secondary NPs originate from fragments formed by the degradation of MPs, plastic litter, and bulk plastics. NPs have a high surface area-to-volume ratio, an important parameter when considering toxicity, and can enrich and transfer many hazardous pollutants to the environment [52,53]. These properties can promote processes such as dissolution, redox reactions, or the generation of ROS. Larger particles would not produce such biological effects even with the same chemical composition [54].

The pathways of microplastic from source to habitat can be different. The sources can be divided into four types: (I) large plastic waste, (II) medicines, (III) textiles, and (IV) cleaning products [50]. From these sources, they can enter aquatic habitats through wastewater treatment plants (WWTPs), which are considered the main route for MPs to enter freshwater, via runoff, through sewers and landfills, and then spread worldwide by wind and currents [5,37,47]. Activities directly related to the aquatic environment can also be sources of marine litter, such as in the case of aquaculture and fishing, which play a significant role in the increase in MPs in aquatic environments [55,56]. Plastic in aquaculture and fisheries are generally used to produce different products, such as cages or nets, buoys, gears, trawls, dredges, traps, and floats, and for different purposes, such as boat construction and maintenance [40,49]. In addition, ALDFGs (abandoned, lost, or otherwise discharged fishing gears) represent one of the main plastic marine litters in the world's oceans [49,57–59].

In summary, the widespread use of microplastics is becoming a serious issue primarily for freshwater and marine environments due to their overwhelming presence on surface and deep waters, on beaches, and in seabed sediments with their consequent ingestion by the organisms and biomagnification or bioaccumulation [37,48]. The diversity of microplastics based on the shape and source of production has favored their diffusion, so much so that microplastic pollution has spread to the most remote corners of the planet and has led to the trophic transfer of micro- and nanoplastics in the marine food chain from plankton to fish [60,61].

4. Environmental Life-Cycle of Plastic Debris

MPs and NPs are considered emerging contaminants of great concern, with unique characteristics that pose a real risk to aquatic life [62,63]. Once in the marine environment, MPs and NPs undergo numerous transformations, which determine both their availability and toxicity to marine organisms [64,65]. Abiotic components (pH, temperature, ionic strength, light intensity, and organic matter concentration) influence these transformations [66]. However, these transformations are mainly driven by the surface properties of MPs/NPs, which play an important role in determining their rapid changes and ecological impacts [66]. The hydrophobic surface of MPs/NPs attracts substances from the surrounding environment, including natural organic matter (NOM), nutrients,

hydrophobic contaminants and heavy metals [67,68]. The interactions occurring with natural biomolecules and chemical substances present in the natural aquatic media lead to the formation of the so-called "eco-corona" [69]. Down the size scale, towards NPs, the absorption concept is translated to a finer level. NPs can adsorb the proteins present in biological fluids onto their surface, forming the "protein-corona", i.e., the bridge at the bio-nano interface [70].

The photodegradation of micro- and nanoplastics can increase the amount of dissolved organic carbon (DOC) in the marine environment by releasing (among others) polybrominated diphenyl ethers, nonylphenol, and triclosan, which are additives added to plastics during production to improve their properties [71]. According to Romera-Castillo et al. (2016), microbes absorb DOC released from plastic, potentially impacting the carbon cycle and microbial ecology as a whole. However, the possible effects of increased DOC on biodegradation and marine biota remain largely unknown [72]. Micro- and nanoplastics can lead to the aggregation of dissolved organic matter (DOM) in particulate organic matter (POM) [73].

Among other materials, plastic represents an ideal substrate for rapid colonization by microorganisms such as bacteria, fungi, diatoms, and algae, which together form a biofilm called "Plastisphere" [74]. This consortium may involve an active interaction in which MPs can be utilized as a carbon source by microorganisms; otherwise, in the case of a passive interaction, this is based on various biochemical and/or physical processes [75]. Among the prokaryotes and eukaryotes that live in the plastisphere, species that degrade hydrocarbons stand out; however, fish and human pathogens are also commonly present [76–78]. The plastisphere formation has led to severe implications for the marine environment because it allows for not only the transport and accumulation of potential pathogenic strains (i.e., genus *Arcobacter* and *Vibrio*), but also the breakdown of plastic fragments by microbial enzymes [75]. Furthermore, the presence of biofilms can alter the properties of virgin polymers by changing their environmental fate (density and buoyancy) and increase bioavailability, appeal, and toxicity to polymers. Laboratory studies demonstrate that befouled plastic debris is ingested more than the corresponding virgin polymers and has magnified acute toxicity in various marine organisms [79–81]. One of the key aspects of the surface interactions is related to the behavior of MPs/NPs along the water column. The layered components of the eco-corona can stabilize or aggregate particles in the water column, increasing their nominal size and density and resulting in the vertical transport of MPs/NPs to the seabed [82]. In this case, MP resuspension processes may occur following natural phenomena such as strong winds or tidal currents. In this respect, bioturbators can play an important role in determining the fate of MPs/NPs, promoting their long-term burial in deeper sedimentary layers or the upward transport of particles buried in sediments [83]. In this context, microbial activity in the plastisphere is considered one of the most important mechanisms of the loss of floating particles, which, due to biofilm formation, will present a consequent positive sedimentation velocity. For this reason, the plastisphere is considered a potential explanation of the conspicuous disparity between the quantities of plastic potentially introduced into the marine environment and those found on the surface, generally referred to as the "missing 99%" surface plastic [84–86].

5. Reactive Oxygen Species and Micro- and Nanoplastics

A literature review based on the Adverse Outcome Pathways (AOP) approach revealed that MP/NPs share the formation of reactive oxygen species as an initial molecular event [87]. Micro- and nanoplastics can generate ROS through a variety of mechanisms (Figure 2). One mechanism is the production of ROS by the particles themselves. The surface of micro- and nanoplastics can be chemically reactive and catalyze the formation of ROS, such as superoxide anion (O_2^-) and hydroxyl radical (•OH), through a process known as the Fenton reaction [88]. Fenton reaction is a chemical reaction that involves the reaction of hydrogen peroxide (H_2O_2) with a transition metal ion, such as iron or copper, to produce •OH and a hydroxyl anion (OH^-). Micro- and nanoplastics can act as

a source of metal ions that can facilitate the Fenton reaction and produce ROS. Another mechanism through which they generate ROS is the adsorption of other pollutants onto the surface of the particles. MPs and NPs can act as a sink for other pollutants, including heavy metals, organic pollutants, and pesticides. The adsorption of these pollutants onto the surface of micro- and nanoplastics can lead to the formation of ROS through various chemical reactions [89]. For example, the adsorption of polycyclic aromatic hydrocarbons (PAHs) onto micro- and nanoplastics surfaces can lead to the formation of ROS through photochemical reactions under UV radiation [88,90].

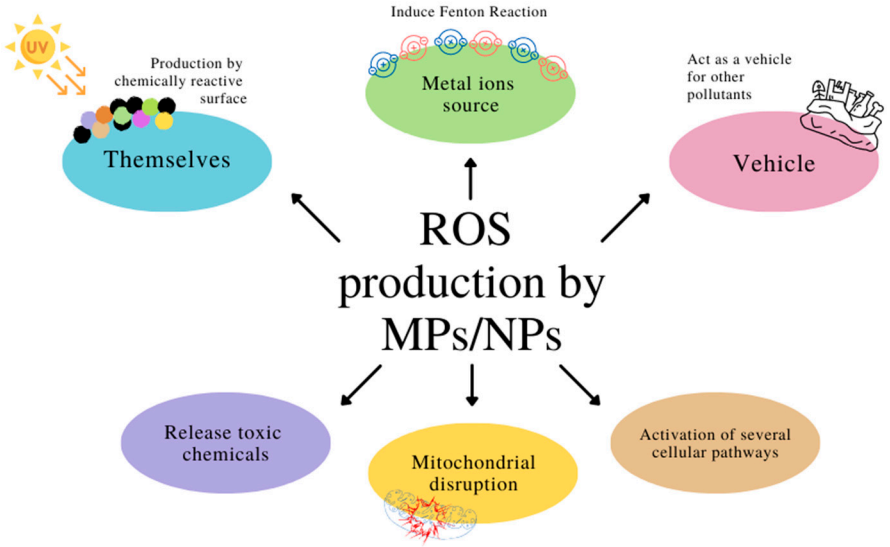

Figure 2. ROS production by MPs and NPs.

Following the light irradiation of polystyrene microplastic suspensions, a significant correlation was observed between the production of organic EPFRs (Environmentally Persistent Free Radicals) and the production of ROS [91]. Micro- and nanoplastics can also release toxic chemicals such as phthalates, bisphenol A, and polycyclic aromatic hydrocarbons, which can induce ROS production [92]. It is possible that the effects induced by MPs and NPs, including ROS production, are size-dependent [54].

The generation of ROS in exposed organisms by micro- and nanoplastics involves several biomolecular mechanisms. One of the key mechanisms is the activation of NADPH oxidase (NOX), a complex enzyme responsible for the production of O_2^- [93]. Mice exposure to micro- and nanoplastics can activate NADPH oxidase through several pathways, including the activation of Toll-like receptor 4 (TLR4) and the aryl hydrocarbon receptor (AhR) [94]. Once activated, NADPH oxidase produces O_2^-, which can be converted to other ROS, such as H_2O_2 and •OH. As reported by Fasciolo et al. [95,96], in rats, the increase in NOX activity, which increases ROS production, could be underlying several diseases in which ROS take part. This is one of the mechanisms by which MPs and NPs can affect human wellness. MPs and NPs themselves can affect redox homeostasis [87]. This is due to the ability of UV radiation and photo-oxidative and thermos-oxidative actions to alter their surface. [97,98]. Photo oxidation or UV light radiation can lead to the formation of free radicals on MP/NPs surfaces as primary products via the subtraction of a hydrogen atom from the macromolecular chain or the addition of a hydrogen atom to an unsaturated carbon chain group (crosslinking reaction) [99]. Once the free radicals are generated along the polymer chain, they can react with atmospheric oxygen and produce polymer peroxyl radicals with the further generation of secondary polymer alkyl radicals [87]. These weathering-induced extracellular free radicals could be one possible explanation for why a significant increase in ROS was observed after the cell entrance of the aged MPs [100].

Another biomolecular mechanism by which micro- and nanoplastics promote ROS production is of the disruption of mitochondrial function [17]. Exposure to micro- and

nanoplastics can lead to mitochondrial dysfunction, which can result in the overproduction of ROS by the electron transport chain (ETC) [101]. The ETC is a series of protein complexes in the inner mitochondrial membrane that transfer electrons from electron donors to electron acceptors, generating a proton gradient that is used to produce ATP. ROS production by ETC is a normal physiological process that occurs as a byproduct of electron transfer. However, exposure to micro- and nanoplastics can disrupt the balance between ROS production and scavenging, leading to ROS overproduction. In addition, micro- and nanoplastics can also lead to the translocation of cytochrome c from mitochondria to the cytoplasm, which can trigger the activation of caspases and the induction of apoptosis [102,103]. The antioxidant defense system plays a critical role in protecting cells against oxidative stress induced by micro- and nanoplastics. Antioxidants scavenge ROS and prevent damage to cellular components, including lipids, proteins, and DNA. Exposure to micro- and nanoplastics can disrupt the antioxidant defense system by depleting antioxidants or impairing their function. For example, exposure to micro- and nanoplastics can lead to a decrease in the activity of antioxidant enzymes activity, such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) [104]. Micro- and nanoplastics can also deplete the levels of non-enzymatic antioxidants, such as glutathione and ascorbic acid [105]. The depletion of antioxidants can cause ROS buildup and consequent oxidative damage to cellular components.

6. The Impact of Micro- and Nanoplastics on the Aquatic Environment

Pollution by human activities (agriculture, industrialization and urbanization) can take many forms, including air, water and soil pollution, electromagnetic pollution (radiation waste), and even noise pollution. These may contribute to health issues and decline the quality of life [106]. Water pollution by agricultural, municipal, and industrial sources have become a major concern for the well-being of humanity and biodiversity [107]. Anthropogenic activities on the aquatic ecosystem have direct and indirect effects that negatively affect biodiversity both in freshwater and sea [108–110]. Among the main threats to aquatic ecosystems and biodiversity is climate change. Climate change can impact marine and freshwater ecosystems in several ways: ocean warming; sea level rise; the loss of sea ice; a decrease in pH due to increased ocean surface acidity; an increased risk of diseases in marine biota; the loss of habitats such as Coral Reef; pollution; nutrient enrichment; hydrological modifications; the spread of invasive species; and increasing levels of UV light [110]. In addition to climate change, the production of plastic and its release into the environment is contributing to the loss of biodiversity and is becoming a serious threat to animal redox homeostasis (Tables 1 and 2) and health (Table 3) and, subsequently, for human health [5].

6.1. MPs/NPs' Effects on Phytoplankton

Normally, micro- and nanoplastics, due to their small size and low density compared to water, tend to float in the water column. When microplastics enter the aquatic environment, they are rapidly colonized by microorganisms and algae, typically within hours [111]. It has been highlighted that a microbial biofilm instantly colonizes plastics when exposed to the environment. It is estimated that between 1000 and 15,000 metric tons of microbial biomass are harbored on plastic marine debris [112]. However, the biomass accumulation due to biofouling can increase its density and cause it to sink [113]. Microalgae also can adhere to particles such as MPs/NPs. Many microalgae secrete polysaccharides if stressed due to decreased nutrients and light. Exopolysaccharides may coagulate to form sticky particles named transparent exopolymer particles (TEPs). TEP favors microalgae aggregation; these aggregates are the primary vehicle for transporting phytoplankton and debris towards the seabed [114]. Another sedimentation pathway consists of small MPs ingested by zooplankton and deposited within fecal pellets [115].

MPs and NPs show effects on each trophic level. Microalgae are the first level in the food chain and are indispensable for the marine ecosystem equilibrium. MPs seem

to affect the well-being of microalgae. As has been reported, different sizes and concentrations of MPs and NPs can inhibit growth, reduce chlorophyll and photosynthesis, induce oxidative stress, cause changes in morphology, and promote the production of heteroaggregates [116,117].

Janak Raj Khatiwada (2023), evaluated the effect of PET microplastic (25 mgL^{-1}, 50 mgL^{-1}, 100 mgL^{-1} and 200 mgL^{-1}) of *Scenedesmus* sp. for 24 days. PET showed the highest growth inhibition effect at 200 mg/L. Moreover, compared to the control group, microalgae exposed to microplastics showed a lower chlorophyll content, possibly due to blocking the pores of MPs for cellular respiration [118]. Cai Zhang (2017) carried out tests on *Skeletonema costatum*, demonstrating that microplastic (Micro-PVC, average diameter 1 μm) significantly inhibited the growth of microalgae and chlorophyll at 96 h under 50 mg/L treatment [119]. Microplastics may also affect microalgae lipid and fatty acid composition. Guschina et al. (2020) assessed the exposure of *Chlorella sorokiniana* to polystyrene microplastics (<70 μm, 60 mgL^{-1}) for four weeks. The study showed that microplastic could alter the concentrations of essential fatty acids, which are necessary for algae's lipid synthesis. Such changes could also have repercussions for food quality, growth, and stress resistance in primary consumers, and could affect potential propagation through trophic transfer [120]. On the other hand, microalgae can also produce effects on microplastics. Among these, it is possible to enumerate the different alterations of MP properties; in particular, their adsorption seems to be enhanced [111]. Wang et al., (2020) showed that the development of a biofilm altered the adsorption of copper Cu(II) and tetracycline (TC) by PE microplastic [121]. The biofilm can also camouflage the plastic particles and promote their ingestion by grazers, such as zooplanktonic organisms [111].

6.2. MPs/NPs in Sediments and Benthos

When micro- and nano plastics lose their ability to float, they can fall and settle on the sea bottom. This severe issue can be observed primarily in coastal shallow-water regions. However, these pollutants are not limited to the coastline; they have also been identified in deep-sea sediments with concentrations of up to 2000 particles m^{-2} at a depth of 5000 m [113,122]. Finally, these small-sized materials will find their pathway to the benthos, affecting some marine organisms. Several studies in the laboratory have investigated the ingestion of micro- and nanoplastics by benthic organisms.

Cole et al. (2020) have compared the toxicity of microplastics and nanoplastics on *Mytilus edulis* for 24 h or 7 days. Mussels were exposed to 500 ng mL^{-1} of 20 μm polystyrene microplastics, 10 × 30 μm polyamide microfibers, or 50 nm polystyrene nanoplastics. After 24 hours of exposure, there was a rise in SOD activity, but after 7 days, it returned to normal levels without negatively impacting health. Particle size, however, can influence sublethal toxicity because exposure to nanoscopic plastic raises the proportion of phagocytic hemocytes (indicating a heightened immune response) and leads to a significant increase in micronuclei formation [123]. The mussels are very important in these studies because they represent the prey of many intertidal species and serve as a food source for humans [124].

Sussarelli et al. (2016) assessed the impact of the polystyrene microspheres (micro-PS) on the physiology of the Pacific oyster (*Crassostrea gigas*). The organisms were exposed to virgin micro-PS (2 and 6 μm in diameter; 0.023 mgL^{-1}) for 2 months during a reproductive cycle. Ingestion preference was shown for the 6 μm particles over 2 μm particles. After 2 months, the histological analysis only detected micro-PS particles in the stomach and intestine. Hyalinocytes and granulocytes in exposed oysters were larger than in controls. The total number of oocytes and the oocyte diameter were significantly lower in exposed females than in controls, while exposed males had a considerably lower sperm velocity. Finally, in progeny issued from exposed genitors than in progeny issued from control genitors, the larval growth and size were significantly slower [125].

Murray and Cowie (2011) assessed the capability of the *Nephrops norvegicus*, an ecologically and commercially important crustacean, to consume plastics. They evaluated the stomach microplastic content of shrimp through trawls in the north Clyde Sea area.

Four tanks were set up, and the organisms were fed fish seeded with approximately 5 mm strands of blue polypropylene rope (ten filaments for one cm^3 of fish). Fish without plastic seeds were provided to the animals in the control group. *Nephrops* were killed after 24 h, and new animals were placed in each tank. This experiment was repeated five times over a 2-week period. *Nephrops* with no plastic in their stomachs had a larger median carapace length than those with plastic. This is presumably due to the capacity of the larger animals to sort plastics from food or excreting plastic once ingested. This study demonstrates that *N. norvegicus* can passively accumulate plastic in the stomach via during feeding. There could be potential implications for human health because the *Nephrops* is a commercially fished species [126].

6.3. Trophic Levels: From Zooplankton to Main Fishes Consumed by Humans

Considering the enormous variety of fish and the environments in which they live [127], humans make use of fish in many ecosystem services [128,129]. Precisely in this regard, it must be considered that human activities are impacting fish biology and their redox homeostasis, on whose alteration micro- and nanoplastics play an increasingly decisive role.

6.3.1. MPs/NPs' Effects on Zooplankton

Zooplankton is the second food chain level and represents a key trophic link in pelagic food webs. The role of zooplankton is vital in the aquatic environment, as they are primary consumers and include the juvenile stages of numerous commercially important species (e.g., the meroplankton) [130]. Cole et al. (2013) reported that the ingestion of polystyrene beads, already starts with the zooplankton, namely the copepod *Centropages typicus*, showing the relevance of the MPs in significantly reducing the consumption of algae. This study implies that marine microplastic debris can negatively impact health and zooplankton function [130]. Ziajahromi et al. (2017) investigated the effects of microplastics on the freshwater zooplankton *Ceriodaphnia dubia*. The acute (48 h) and chronic (8 d) effects of microplastic polyester fibers and 1–4 µm PE beads on zooplankton have been examined. This study demonstrated the microplastic fibers caused a 50% reduction in reproductive capacity at concentrations approximately six times higher than environmental concentrations. Unlike other studies, this study did not observe any ingested fibers. However, malformations have been observed in the carapace of organisms exposed to polyester fibers. However, malformations have been observed in the carapace of organisms exposed to polyester fibers. This demonstrates that the negative effects of microplastic fibers on exposed aquatic organisms can also include external physical damage and not only those resulting from ingestion [131]. Rehse et al. (2016) investigated the effects of 96 h exposure of PE-particles (size 1 µm) on *Daphnia magna*. The study showed that the particles are ingested and cause immobilization. The immobilization of daphnids increases with dose and time. EC_{50} was 57.43 mg L^{-1} [132].

Another species utilized to study the effects of MPs/NPs is the marine copepod *Calanus helgolandicus*. This organism is a keystone species that can constitute up to 90% of mesozooplankton biomass within marine waters throughout Europe and the northeast Atlantic. *Calanus helgolandicus* has a large size and high lipid content; its abundance makes it a vital prey species for the larvae of a number of commercially important fish. Cole et al. (2015) exposed *Calanus helgolandicus* to 20 µm polystyrene spheres and cultured algae for 24 h to evaluate ingestion rate and for 9 days to evaluate reproductive function. The results demonstrate that microplastics can alter the ingestion of algae and reduce the size of the eggs, probably due to a reduction in the ingested carbon biomass. Moreover, reducing ingested carbon could cause an energy depletion and the consequent death of the organism [133].

The effects of microplastics are not always so evident, as in the case of exposing the MPs to Pacific oyster larvae (*Crassostrea gigas*). The exposure has not shown any significant effects on the development or feeding capacity of the larvae. This could be because of the oyster's more simplistic intestinal tract [134].

Zheng et al. (2020) suggested an interesting point of view. MPs ingested by copepods, the crustacean zooplankton that is the main prey of fish, are likely to be carried up the food chain, while those ingested by jellyfish, which have fewer predators than copepods, are more likely to be discharged into the marine environment and circulate in seawater or seabed sediments. In this regard, the distribution of MPs, in addition to depending on the chemical-physical characteristics already discussed, also depends on the different groups of zooplankton [135].

6.3.2. MPs/NPs' Effects on Others Aquatic Trophic Levels

The effects of these pollutants on fish vary depending on the dose, the target organisms, and the interactions between the pollutants [136,137]. Nanoplastics have been reported to have a higher impact than microplastics [50,138]. However, this is not always true. As reported by Jiang et al. (2020), there are conditions in which microplastics can have a greater impact than nanoplastics [45]. These differences can be attributed to multiple factors, including the capacity of ingestion, the possibility of entering tissues and cells, and the type and shape of the polymer that can affect the particles' interaction with other pollutants and chemical compounds [139–141].

Microplastics may not always have "negative" effects. As already stated, based on the shape and size of the polymer, microplastics can interact with other chemical pollutants. This capacity, which also depends on the dose, could mitigate the effect of chemical pollutants (antagonistic effect). If, on the one hand, MPs can reduce pollutants' effects, on the other, they can act synergistically by enhancing its effects (agonist effect), which in the latter case is potentially more toxic than nanoplastics [142–144]. Environmental factors can also dictate the dose-dependent impacts of microplastics. For example, phenomena such as storms and cyclones can increase their concentration and thus induce an increment in dose in specific areas, leading to an increase in the absorption by aquatic organisms [50,145].

Neves et al. (2015) reported the presence of microplastics in 63.5% of benthic fish and 36.5% of pelagic fish species, with a total of 73 microplastics identified from fish stomach contents [146]. A study on rainbowfish (*Melanotaenia fluviatilis*) exposed to microbeads adsorbed with polybrominated diphenyl ethers (PBDEs) was monitored for 0, 21, 42, and 63 days. Exposed fish accumulated high concentrations of PBDEs (ca.115 pg g^{-1} ww per day) in tissue after ingestion [147]. Redondo-Hasselerharm et al. (2018) report that in *Gammarus pulex* exposed to sediment containing MPs (Micro-PS 20–500 µm for 28 d), the growth is reduced [148], as well as in *Gammarus fossarum* [149]. A study conducted on juvenile European perch (*Perca fluviatilis*) exposed to polystyrene microplastic particles (90–150 µm) over six months showed that the animals ingested and accumulated the polystyrene microplastics which resulted in reduced growth; delayed hatching; and impaired performance, nutrition, and behavior [150].

MPs and NPs also influence the olfactory senses, which increases susceptibility to being killed by predators [151,152]. The predator–prey relationship gradually increases the toxicity of these substances as they are transferred from organism to organism, accumulating [62,64,109,153]. About 18% of the main predators of the central Mediterranean, Swordfish (*Xiphias gladius*), bluefin tuna (*Thunnus thymus*), and albacore (*Thunnus alalunga*) ingested micro-, meso-, and macroplastic debris ranging in size from <5 mm to 5–25 mm to 25 mm, respectively, [154,155] found microplastics in 36.5% of the gastrointestinal tracts of pelagic and demersal fish. Tuna is the main consumed fish; in fact, much attention is being paid to the sustainability of its fishing by FAO and the UN [156]. Dias-Basantes et al. (2022) showed that canned tuna can provide an average of 692 ± 120 MPs/100 g in brine-soaked tuna and 442 ± 84 MPs/100 g in oil-soaked tuna, values that significantly exceed those reported in research on canned fish [157]. Di Giacinto et al. (2023) showed the presence of MPs (size < 10 µm), polymers (PET, polycarbonate (PC)), and additives (Bisphenol A (BPA) and p-phthalic acid (PTA)) in the muscle *Thunnus Thynnus* and *Xiphias Gladius* fished in the Mediterranean Sea [158]. Abihssira-García et al. (2020) report that MPs were also found in Atlantic Salmon (*Salmo salar*), the second main fish consumed by humans. They

show that Atlantic salmon immune cells from blood, distal intestine, and head kidney can phagocytose MPs (1–5 μm) even at relatively low concentrations (low 0.05 mgL^{-1}; medium 5 mgL^{-1}; high 50 mgL^{-1} for 1, 24, 48 and 72 h) and that their mortality is affected by the time exposure and the microplastic type [159].

Another aspect concerning the trophic levels that should be considered is that the ingestion and accumulation of microplastics take place before maturity, impacting the number of available organisms. A study conducted on *Artemia* nauplii (the early stage of development that occurs after the cysts hatch), which were subjected to a high concentrations of microplastics (1.2×10^6 particles per 20,000 nauplii), showed that they had ingested and accumulated microplastic particles ranging in size from 1 to 20 μm, in high concentrations; these particles were subsequently transferred to the zebrafish that fed on the nauplii [160]. Although not all particles would have been transferred from the nauplii to the zebra, as they were partially excreted, some were retained within the epithelial cells and intestinal villi. Another study on the absorption and effect of microplastics on zebrafish showed that most of the plastic particles (5 μm in diameter) had accumulated in the gills, intestines, and liver. Those with a larger diameter (20 μm) could accumulate only in the intestine and gills. Thus, the smaller particles (5 μm) are more toxic than the bigger ones (20 μm), causing inflammation and the accumulation of lipids in the fish liver, inducing oxidative stress, and altering the metabolic profiles of the fish liver, disturbing lipid and energy metabolism [161]. Accordingly, Jeong et al. (2016) showed that exposure to plastic beads (0.05, 0.5, 6 μm) in *Brachionus koreanus* was associated with increased oxidative stress and decreased growth rate, fecundity, lifespan, reproduction time, and body size, suggesting that the toxicities of MPs and NPs are size-dependent and that smaller plastics are more toxic than bigger ones [162]. This size dependency is probably correlated with the ability of smaller molecules to enter cellular compartments. Manabe et al. (2011) reported that smaller NPs were more easily ingested by medaka (*Oryzias latipes*) than the bigger ones and that NPs were excreted more slowly than the microplastics [163]. NPs negatively affect the liver health of medaka fish, leading to significant alterations in digestion, innate immune, and antioxidant-enzyme-based liver tissue damage [164] and inflammation [165]. However, microplastics are capable of damaging cellular organelles. In line with that, Felix et al. (2023) find that MPs in zebrafish can affect the center of the energy balance: mitochondria. After 21 days of exposure to a toxicological concentration of MPs, anxiety-like behavior arose, while 1 mg L^{-1} treatment showed a decrease in hepatic mitochondrial respiration and membrane potential ($\Delta\Psi$), both indices of suppression of mitochondrial respiratory chain [166]. Trevisan et al. (2019) also reported that NPs can cause mitochondrial energy disruption, a decline in energy efficiency, and differential mitochondrial uptake in developing zebrafish [167]. Mitochondria can also be a target for PAHs toxicity as its high lipid content facilitates PAH uptake [168]. Several studies reported that PAHs, such as benzo[a]pyrene (BaP), phenanthrene, and fluoranthene [169] or complex PAHs mixtures, can impair mitochondrial bioenergetics in embryonic or larval fish [170,171]. Gu et al. (2023) found that in sea cucumber (*Apostichopus japonicus*), exposure to polystyrene nanoplastics (PS-NPs) significantly inhibited the complex activities in the mitochondrial respiratory chain and affected the relative expression levels of mitochondrial apoptosis-related genes [172].

6.4. MPs/NPs' Effects on the Redox Homeostasis of the Aquatic Organisms

A previously mentioned, MPs and NPs are able to affect the redox homeostasis. However, the effects reported in literature sometimes are conflicting, even in the same species. Probably, the differences found are related to age [173].

6.4.1. NPs

In zebrafish embryos, at 96 h post-fertilization (hpf), while exposed 50 nm (1 mgL^{-1}) to PS-NPs, Bhagat et al. (2022) found that ROS production was increased without a significant change in the malondialdehyde (MDA) concentration. Instead, the antioxidant system

was affected with decreased SOD and GR activity, increased CAT activity, and reduced glutathione content without significant change in GPX activity [174]. In 96 hpf zebrafish embryos exposed for 24 h to 50 nm and 1 µm PS-NPs (which are considered NPs by the authors) (10 mg/L), an increase in ROS content with both sizes was found [175].

In adult zebrafish exposed to 70 nm PS-NPs (0.5 ppm and 1.5 ppm) for 7 days, a significant increase in muscle (but not liver) ROS content was found only at the highest concentration [176]. According to these results, in adult zebrafish (F0) exposed to 42 nm PS-NPs added to the diet (10% of the food by mass) for one week, no difference in either genders was found in the liver GR, GPX, and CAT antioxidant enzymes [177]. Instead, a significant decrease was found in the GR activity of male muscle. In F1 96 hpf larvae from exposed maternal and co-parental groups displayed significantly lower total thiol levels (reduced forms of protein and non-protein thiols). GR activity was significantly reduced in these larvae, while the activities of GPX and CAT were not changed [177]. In another study, Aliakbarzadeh et al. (2023) exposed adult zebrafish for 45 days to PS-NPs of different sizes, 20–80 nm, at different concentrations (0.1 μgL^{-1}, 1 μgL^{-1}, 10 μgL^{-1}, and 100 μgL^{-1}). They found an NP-concentration-dependent decrease in CAT activity and total GSH content [178]. In another study conducted on the adult zebrafish exposed to PS-NPs (103–113 nm) for 14 days at 10 μgL^{-1} and 100 μgL^{-1}, a reduction in gills' antioxidant enzymes activity (SOD and total antioxidant capacity (TAC)) was found [179]. Although it may appear that in adult zebrafish, exposure to the different concentrations of PS-NPs reduces antioxidant capacity by promoting oxidative stress, in another study, it was shown that after 3 weeks of exposure to 70 nm PS-NPs at different concentrations (20 µg/L, 200 µg/L, and 2 mg/L), there is an increase in the activity of CAT and SOD only at the higher concentration (2 mg/L) [180].

Table 1. Effects of PS-NPs on redox homeostasis of zebrafish. ROS: reactive oxygen species; CAT: catalase; SOD: superoxide dismutase; GR: glutathione reductase; NS: No significative difference compared to control; t-GSH: total glutathione content; TAC: total antioxidant capacity; hpf: hours post-fertilization (↑ increase; ↓ decrease).

Redox Homeostasis in *Danio Rerio* Exposed to PS-NPs						
Author	**Age**	**Exposition**			**Tissue**	**Results**
		Size	**Concentration**	**Time**		
Bhagat et al. [174]	96 hpf	50 nm	1 mgL^{-1}	~2–96 hpf	/	↑ROS ↑CAT ↓SOD ↓GR
Sendra et al. [175]	96 hpf	50 nm, 1 µm	10 mgL^{-1}	24 h	/	↑ROS
Pitt et al. [177]	96 hpf	42 nm	10% add. to diet	1 week	Co-parental	↓Thiols ↓GR
Sarasamma et al. [176]	Adult	70 nm	0.5 ppm, 1.5 ppm	1 week	Muscle Liver	↑ROS NS
Pitt et al. [177]	Adult	42 nm	10% add. to diet	1 week	Male muscle	↓GR
Aliakbarzadeh et al. [178]	Adult	20–80 nm	0.1 μgL^{-1}, 1 gL^{-1}, 10 gL^{-1}, 100 gL^{-1}	45 days	Gut	↓CAT ↓t-GSH
Umamaheswari et al. [179]	Adult	103–113 nm	5 mgL^{-1}, 10 mgL^{-1}	14 days	Gills	↓SOD (5 mgL^{-1}) ↓TAC (10 mgL^{-1})
Lu et al. [180]	Adult	70 nm	20 μgL^{-1}, 200 μgL^{-1}, 2 mgL^{-1}	3 weeks	Liver	↑SOD (2 mgL^{-1}) ↑CAT (2 mgL^{-1})

6.4.2. MPs

In 96 hpf zebrafish embryos exposed to PS-MPs 1–5 µm (2 mgL^{-1}), the redox homeostasis results unaffected [181]. The lack of effects of PS-MPs on the redox homeostasis of zebrafish embryos was also highlighted in another work, in which, following seven days of

exposure of 6dpf (days post fertilization) zebrafish to 5 µm PS-MPs (50 ngmL^{-1}), no changes were found in the concentration of MDA and the activity of SOD, CAT and GPX [182]. Similar results were found in 30dpf zebrafish exposed to 5 µm PS-MPs (500 µgL^{-1}) for 25 days [183]. However, while in 96hpf zebrafish, the activity of antioxidant enzymes was not affected by MPs exposition, in 30 dpf zebrafish, CAT activity was decreased [183], reinforcing the hypothesis that the MP-induced impairment of redox homeostasis is age-related. On the other hand, Guimarães et al. (2021) found that in juvenile zebrafish exposed to two different concentrations of PS-MPs (4×10^4 and 4×10^6 particles/m^3) for five days, an increase in thiobarbituric acid reactive species (TBARS) following both treatments. However, they found no increase in the hydrogen peroxide concentration but verified an increase in the SOD and CAT activity enzyme and in the total glutathione concentration, without difference in the reduced glutathione concentration [173].

CAT and SOD were also increased in the gut tissue of adult zebrafish exposed to PS-MPs (5 µm) for 21 days at two different concentrations (50 µgL^{-1} and 500 µgL^{-1}) [184]. Another work conducted on adult zebrafish exposed to PS-MPs 9–10 µm (10 mg/L) for 4 or 8 days reports an interesting view of the effect exerted by MPs on the liver [185]. They found that after four days of exposition to MPs, the activity of the SOD, CAT, and GPX enzymes was increased, while MDA concentration was unaffected. After eight days of exposition, SOD activity returned to the control level, while CAT and GPX activity remained higher, and MDA content decreased compared with the control. Accordingly, Lu et al. (2016) found that after three weeks of exposure of adult zebrafish to PS-MPs (5 µm) at different concentrations (20 µgL^{-1}, 200 µgL^{-1}, and 2 mgL^{-1}), SOD activity increased at all concentrations, while CAT activity increased with 200 µgL^{-1} and 2 mgL^{-1} treatment [180].

Table 2. Effects of PS-NPs on redox homeostasis of zebrafish. CAT: catalase; NS: no significative difference compared to control; TBARS: thiobarbituric acid reactive substances; SOD: superoxide dismutase; t-GSH: total glutathione content; GPX: glutathione peroxidase; MDA: malondialdehyde; hpf: hours post-fertilization; dpf: days post-fertilization (↑ increase; ↓ decrease).

Redox Homeostasis in *Danio Rerio* Exposed to PS-MPs						
Author	**Age**	**Exposition**			**Tissue**	**Results**
		Size	**Concentration**	**Time**		
Santos et al. [181]	96 hpf	1,5 µm	2 mgL^{-1}	/	/	NS
Yang et al. [182]	6 dpf	5 µm	50 ngmL^{-1}	1 week	/	NS
Chen et al. [183]	30 dpf	5 µm	500 µgL^{-1}	25 days	/	↓CAT
Guimarães et al. [173]	Juvenile	~17 µm	4×10^4, 4×10^6 particles/m^3	5 days	/	↑TBARS ↑CAT ↑SOD ↑t-GSH
Qiao et al. [184]	Adult	5 µm	50 µgL^{-1}, 500 µgL^{-1}	21 days	Gut	↑CAT ↑SOD
Li et al. [185]	Adult	9, 10 µm	10 mgL^{-1}	4 days 8 days	Liver	4 d ↑SOD ↑CAT ↑GPX NS MDA 8 d NS SOD ↑CAT ↑GPX ↓MDA
Lu et al. [180]	Adult	5 µm	20 µgL^{-1}, 200 µgL^{-1}, 2 mgL^{-1}	3 weeks	Liver	↑SOD ↑CAT (200 µgL^{-1}, 2 mgL^{-1})

Table 3. PET: polyethylene terephthalate; PVC: polyvinyl chloride; PS: polystyrene; PA: polyamide; PP: polypropylene; PE: polyethylene PHB: polyhydroxybutyrate; PMMA: poly(methyl methacrylate); PLA: poly(l-lactide).

Authors	Species	Type of MPs/NPs	Exposition Time	Effects
Khatiwada et al. [118]	*Scenedesmus* sp.	Micro-PET	24 days	Negative influence on the growth of microalgae and eventually reducing chlorophyll.
Zhang et al. [119]	*Skeletonema costatum*	Micro-PVC	96 h	Growth inhibition and chlorophyll.
Guschina et al. [120]	*Chlorella sorokiniana*	Micro-PS < 70 µm	4 weeks	Alteration of the concentrations of fatty acid molecules and lipid synthesis.
Cole et al. [123]	*Mytilus edulis*	Micro-PS 20 µm/Microfibers-PA 10 × 30 µm/Nano-PS 50 nm	24 h 7 days	Higher SOD activity after 24 h exposure. SOD activity returned to normal values after 7 days. Nanoplastics increased the proportion of phagocytic hemocytes and resulted in a marked increase in micronuclei formation.
Sussarellu et al. [125]	*Crassostrea gigas*	Micro-PS 2–6 µm	2 months	Exposed oysters, hyalinocytes, and granulocytes > control. Exposed oocytes and oocyte diameter < control. Exposed sperm velocity < control. Exposed Progeny size and growth < control.
Murray and Cowie et al. [126]	*Nephrops norvegicus*	PP	24 h each tank five times 2 weeks total	Plastic in stomachs; potential implications for human health.
Cole et al. [130]	*Centropages typicus*	Micro-PS 7.3 µm	24 h	Decreased algal feeding and negative impact upon health and zooplankton function.
Ziajahromi et al. [131]	*Ceriodaphnia dubia*	Micro-PS/PE 1–4 µm	48 h 8 days	50% reduction in reproductive and malformations in the carapace.
Rehse et al. [132]	*Daphnia magna*	Micro-PE 1 µm	96 h	Immobilization and EC50 of 1-µm was 57.43 mgL^{-1} after 96 h.
Cole et al. [133]	*Calanus helgolandicus*	Micro-PS 20 µm	24 h 9 days	Decrease in microalgae ingestion and reduction in egg size.
Redondo-Hasselerharm et al. [148]	*Gammarus Pulex*	Micro-PS 20–500 µm	28 days	Growth decreased.
Straub et al. [149]	*Gammarus Fossarum*	Micro-PHB PMMA 32–250 µm	28 days	Growth decreased.
Kardgar et al. [150]	*Perca fluviatilis*	PLA 90–150 µm	Over 6 months	Reduced growth; hindered hatching; and impaired their performance, nutrition, and behavior.

Table 3. *Cont.*

Authors	Species	Type of MPs/NPs	Exposition Time	Effects
Abihssira-García et al. [159]	*Salmo salar*	Micro PS/PE 1–5 µm	1, 24, 48, and 72 h	Immune cells can phagocytose MPs and the mortality of the blood cells, distal intestine, and head kidney is affected by the time exposure, and the impact is dependent on the microplastic type.
Lu et al. [161]	*Danio Rerio*	Micro-PS 5 µm–70 nm	1 week	Increased inflammation, oxidative stress, altered metabolic liver profile.
Jeong et al. [162]	*Brachionus koreanus*	Micro-PS 0.05–0.5–6 µm	12 days	Increased oxidative stress and decreased growth rate, fecundity, lifespan, reproduction time, and body size.
Zhou et al. [164]	*Oryzias latipes*	Nano-PS 100 nm	3 months	Liver health impaired.
Felix et al. [166]	*Danio Rerio*	Micro-PP 200 µm	21 days	Decreased mitochondrial health, anxiety-like behavior, and increased oxidative stress.
Trevisan et al. [167]	*Danio Rerio*	Nano-PS 44 nm	24–48–96 h post-fertilization	Mitochondrial energy disruption, a decline in energy efficiency, and differential mitochondrial uptake.
Gu et al. [172]	*Apostichopus japonicus*	Nano-PS 100–200 nm	20 days	Inhibits the complex activities in the mitochondrial respiratory chain and affect the relative expression levels of mitochondrial apoptosis-related genes.
Li et al. [186]	*Chlamydomonas reinhardtii*	Micro-PS	10 days	Micro-PS had negative effects on growth and algal photosynthesis.
Besseling et al. [187]	*Arenicola marina*	Micro-PS 400–1300 µm	28 days	As plastic is ingested by *A. marina*, its predators will be exposed to plastic as well. Weight loss.
Wang X et al. [188]	*Mytilus coruscus*	Micro-PS 2 µm	14 days plus 7-day recovery acclimation	Inhibition of digestive enzymes.

7. Ingestion of MPs/NPs from Aquatic Food by Human

Total aquaculture and fisheries production reached a record value of 214 million tonnes in 2020, 178 million tonnes of which were accounted for by aquatic animals and 36 million tonnes by algae. These activities generated about USD 424 billion in revenue. Aquatic food furnishes about 17% of animal proteins globally; this figure rises to over 50% in several countries, mainly from Asia and Africa. Primary production alone employs 58.5 million people, 21% of whom are women. In 2019, global aquatic food available for human consumption was estimated at 158 million tonnes [189]. Asia accounted for 72% of the total, while its population represented 60% of the world population. As aquatic food consumption expands worldwide, humans are inevitably exposed to microplastics at some level. Van Cawenberghe and Janssen (2014), estimated that in Europe, mollusks consumers ingest up to 11,000 microplastics per year [190]. In the case of the annual consumption of shrimps, the amount is 175 microplastic particles per person per year, assuming a scenario in which 90% of microplastics are removed [191]. Similarly, the consumption of sea urchins *Paracentrotus lividus*, assuming an average consumption by humans of approximately 1 kg of sea urchin gonads, could deliver 166–207 items/per year/per person [42]. The evidence of genetic alterations in mollusks associated with the presence of micro- and nanoplastics in the food web indicates that potential genetic alterations could also occur in humans due to the consumption of seafood contaminated with plastic particles [192,193]. The problem increases where it is customary to consume raw or dried fish, since the transfer of microplastics is greater, as in the transfer of MPs accumulated in the intestine and the lymphatic system [194]. The transport mechanism of MPs and NPs across physiological barriers is influenced by the para-physiological condition. In fact, it has been observed that there is an increased intestinal permeability in patients with inflammatory diseases of the colon related to an increased transport of MPs [194]. High concentrations of MPs/NPs are usually highly cytotoxic, usually via ROS production and pro-inflammatory processes. MPs/NPs' toxicity is assessed by the response of intestinal lipid peroxidation biomarkers as an indication of the feedback leading to oxidative stress, inflammation, epithelial barrier integrity, and changes in the intestinal microbiota [195,196]. In the immortalized cell line of human colorectal adenocarcinoma cells (Caco-2), it has been shown that PS-MPs (0.1 µm and 5 µm, 1–200 µg/mL) can disrupt the mitochondrial membrane potential and inhibit ATP-binding cassette (ABC) transporter activity [197]. In addition to local damage in the intestine, it has been reported, in mammalian systems models, that microplastics that have specific characteristics (resistance to chemical degradation, resistance to mechanical clearance, and biopersistence) can translocate across living cells, such as M cells or dendritic cells, to the lymphatic and/or circulatory system, accumulate in secondary organs, and impact the immune system and cell health [198]. Buyan (2022) also reports that MPs can be transferred in distal tissues via the circulatory system, causing inflammation reactions, cell blood cytotoxicity, vascular swelling, obstruction, and respiratory high blood pressure [199]. The close link between MPs and NPs, mitochondria and ROS has also been demonstrated in human lung epithelial BEAS-2B cells, on which they have a cytotoxic and inflammatory effect related to an increase in reactive oxygen species [200]. Another study has demonstrated that the accumulation of NPs in lysosomes played a central role in the observed cell death, leading to the swelling of the lysosomes and the release of cathepsins into the cytosol, which ultimately propagated the damage to the mitochondria with the subsequent activation of apoptosis. This process was accompanied and sustained by other events, such as increasing ROS levels and autophagy [201]. These effects are due to the ability of internalized NPs to impair the function of mitochondria and induce the monomerization of BAX on the mitochondrial surface, leading to the influx of calcium and the outflux of mitochondrial cytochrome c, which would result in a reduction in the membrane potential, insufficient ATP production, and the activation of downstream caspase-9 and caspase-3, eventually initiating the mitochondrial apoptotic pathway [202].

In summary, the micro- and nanoplastics released into the environment, through the consumption of food return to humans (Figure 3), and once internalized, MPs and NPs

can act in multiple districts and levels, representing a key factor in human health. In fact, Wick et al. (2010) report that nanoparticles can pass the placental barrier in a perfusion model of the human placenta [203], while Lehner et al. (2019) report that NPs can permeate the blood–brain barrier [204], making it critical and urgent to deepen studies. Following ingestion, MPs/NPs access the bloodstream and, following the translocation process, reach different organs and tissues, triggering toxicity pathways, thereby including inflammation and oxidative stress.

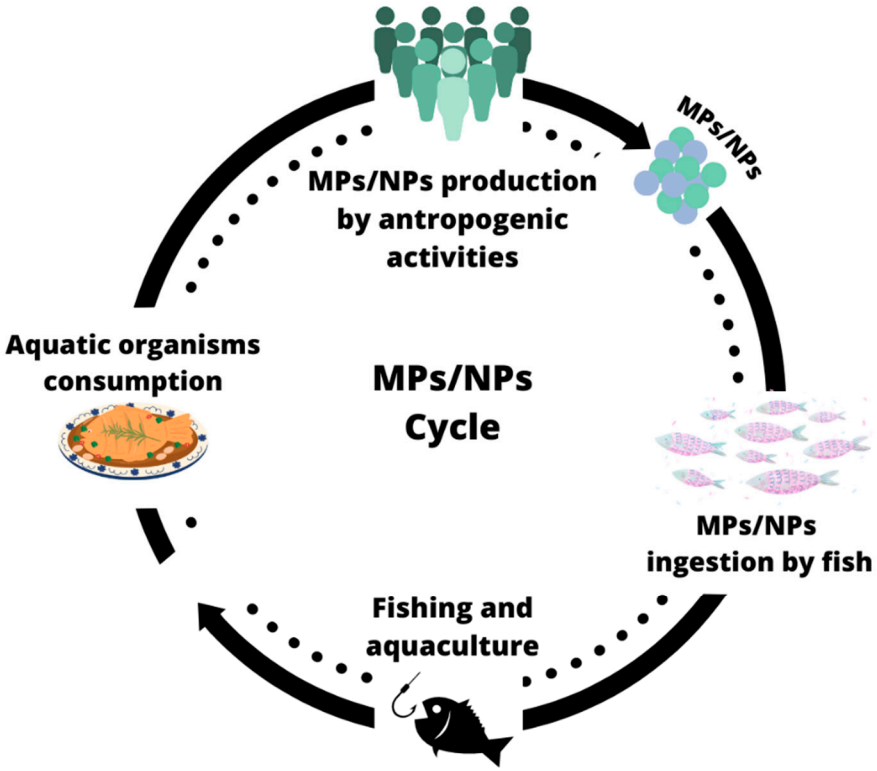

Figure 3. MPs/NPs cycle. Micro- and nanoplastics produced by anthropogenic activities end up in the water and, by the consumption of aquatic organisms as food, return to human.

8. Future Studies and Perspectives

The production and accumulation of plastic debris in aquatic environments has become a major concern, with microplastics and nanoplastics posing a significant risk to the ecosystem. Studies on the ecotoxicities and effects of microplastics and nanoplastics on aquatic organisms have increased since the 1990s, and many questions about their fate and invisible effects remain incomplete. The association of metal ions with plastic particles in aquatic systems alters their potential bioavailability and may trigger toxic effects. It is crucial to conduct more realistic experiments to better assess the risk of micro- and nanoplastics in aquatic environments and to establish new standardized methods to isolate, identify, and quantify nanoplastics. The trophic transfer of micro- and nanoplastics in complex food chains, particularly in brackish water environments, must be further investigated. It is also essential to thoroughly assess the toxicity of biodegradable plastics, such as polyhydroxybutyrate (PHB), to ensure the necessary health and safety assessment before substituting them for non-biodegradable plastics [205,206]. Future studies on micro- and nanoplastics should focus on urgent topics, such as the interactions between the surfaces of micro- and nanoplastics and the environment, trophic transfer, generational effects, and the long-term effects of these small plastics on aquatic organisms. To achieve this goal, it would be helpful to use plastic particles sampled in natural environments, characterize their composition, pollutants, and additive content, and analyze the effects of plastic particle contamination at the ecosystem level. As indicated by Prüst et al. (2020), it should be considered that there are significant differences between the most-used model species (such as fish and bivalves)

and mammals, including humans with respect to physiology and routes of exposure (e.g., exposure via water (gills) vs. oral uptake (gut) or inhalation (lungs)). Such differences in exposure pathways may affect uptake and/or distribution, explaining the diversity in the observed effects [193]. One solution could be to start using alternative materials. As mentioned in the previous chapters, ALDFGs are a constantly increasing problem in aquatic environments. Using bio-based and/or biodegradable polymers may reduce plastic waste and MPs entering aquatic environments. The bio-products include different materials, like polylactic acid (PLA) or polybutylene succinate (PBS) (biodegradable in industrial composting conditions at temperatures higher than 50 °C), polystarch or cellulose composites, and polyhydroxyalkanoate (PHA) polyesters of natural origin, accumulated in the form of intracellular granules by a wide variety of bacterial strains during unstable growth circumstances [207,208]. PHB belongs to the family of PHA, and its accumulation has been demonstrated in several cyanobacteria [1,2,209]. PHB is a biodegradable polymer that is renewable and can be an alternative to petroleum-based plastics [210]. The main issue in producing these polymers is the high cost of the nutrient source. The price of the raw material, mainly the carbon source and the biopolymer recovery process, contributes to the high PHA production cost [211]. The cultivation of source organisms on wastewater can help overcome this limit [1,2].

Aquaculture wastewater could be a viable alternative for low-cost PHB production. *Arthrospira platensis* (Spirulina) is an important microorganism that accumulates PHB [210] and, in addition to growing on aquaculture wastewater for bioremediation, can also be a viable alternative in the partial replacement of fishmeal in aquafeed, where overfishing of fish stock and impacts of aquaculture are becoming another issue for our oceans [212]. Furthermore, Harding et al. (2007) evaluated through life cycle assessment (LCA) the cradle-to-gate impact of PHB production compared to PP and PE production. The results showed that PHB production is more beneficial than PP, while PE production has lower environmental costs than PHB in acidification and eutrophication. For the impacts in all other categories, PHB performs better than PE. The effects of polymer disposal were not included in this study. The disposing of polymers different from PHB is expected to have additional negative environmental impacts on their life cycles, giving PHB further environmental benefit [213]. However, the utilization of these materials should be limited in aquatic environments due to the little data available on their functionality or durability [207].

9. Conclusions

In this review, we wanted to collect information about the impact of micro- and nanoplastics on the food chain, particularly on redox homeostasis. In this regard, we have focused on oxidative stress regarding the effect of a single pollutant, polystyrene, at different exposure times, concentrations, tissues, and evolutionary stages. The effects of micro- and nanoplastics reported in the literature on redox homeostasis appear varied and sometimes conflicting. NP effects are quite contrasting. Contrarily, MP effects appear to be somewhat more consistent. This difference is probably due to the ability of NPs to enter cells more than MPs and to be ejected more slowly. The only certainty is the ability of nanoplastics and microplastics to affect redox homeostasis and mitochondrial well-being, acting at multiple levels and in several tissues directly or as vectors to other pollutants. The ability of MPs/NPs to cross almost any barrier, such as the blood–brain barrier, or the ability to reach the placenta, makes it urgent to find solutions that can be implemented in the shortest possible time. A start could be represented by the use of bio-based and/or biodegradable polymers in the perspective of the circular economy to minimize the impact of MPs and NPs on the environment and the health of both aquatic organisms and humans.

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