~~A Comprehensive Review on the Pollutant Effects~~Toxicological Implications of Emerging Pollutants on Aquatic ~~Animals~~Organisms

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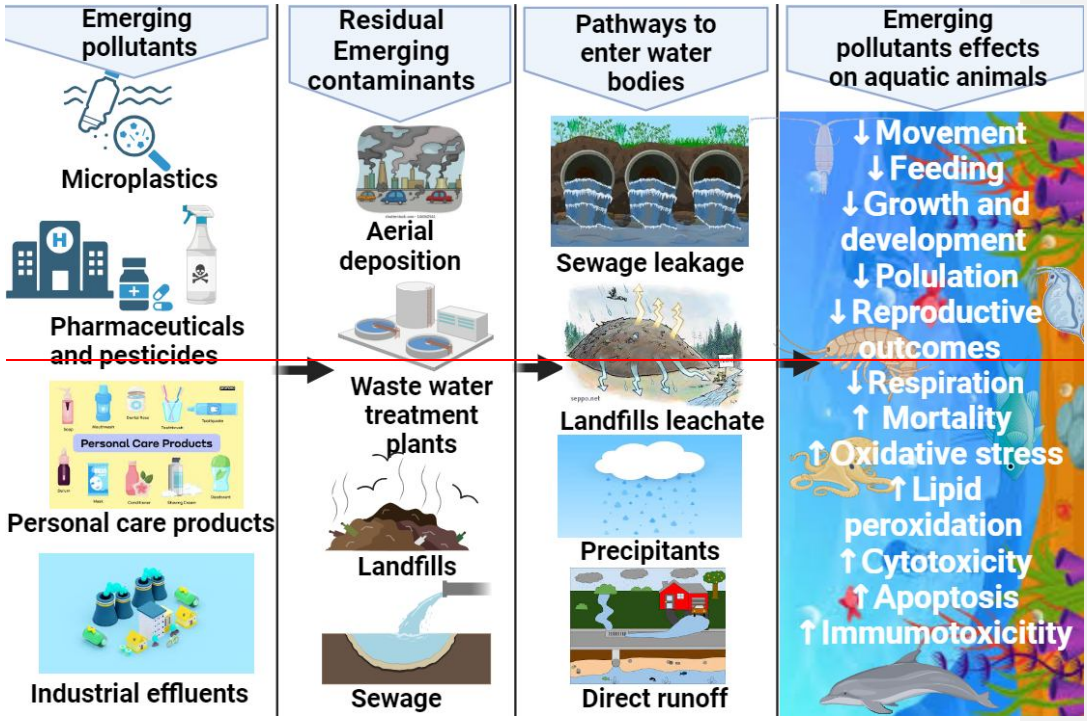
Abstract

Emerging pollutants (EPs), including pharmaceuticals, pesticides, personal care products, microplastics, ~~and~~ nanoparticles, and heavy metals have become significant environmental threats, ~~impacting both terrestrial and~~increasingly affecting aquatic organisms. This review ~~explores the pervasive influence of~~ consolidates recent findings on how EPs ~~on~~enter aquatic ~~species, emphasizing their entry~~systems through industrial discharges, agricultural runoff, wastewater, and landfill leachate~~ into the water bodies. The emerging pollutants persistence and accumulation in aquatic ecosystems pose serious risks to fish, amphibians, mollusks, and crustaceans. The~~, where they persist, bioaccumulate, and exert chronic ~~emerging pollutants exposure disrupts physiological and behavioral functions, causing~~ toxic effects. Evidence indicated that EPs induced oxidative stress, endocrine disruption, ~~immune suppression, oxidative stress,~~ genotoxicity, and immunotoxicity as primary mechanisms of toxicity. In fishes, exposure to microplastics, nanoparticles, and pesticides disrupted antioxidant enzyme activities, impaired reproduction, and altered behavior and metabolism. Amphibians exhibited delayed metamorphosis, endocrine and thyroid dysfunction, and neurotoxicity under exposure to pesticides and bisphenols, while molluscs showed impaired filtration capacity, oxidative DNA damage, and reduced gamete viability upon contact with pharmaceuticals and microplastics. These mechanistic disruptions collectively led to reduced growth, reproductive ~~success, and developmental abnormalities. These effects not only impair individual species health but also destabilize population dynamics, reduce~~ failure, and

biodiversity~~, and hinder the~~ loss, ultimately destabilizing aquatic food webs and ecosystem functionality. ~~Furthermore, the ability of EPs to bioaccumulate and biomagnify in food webs raises concerns for human health and ecosystem resilience that underscore the need for improved understanding of their toxicological mechanisms and long-term~~The review highlights critical research gaps, particularly concerning mixture toxicity, environmentally relevant concentrations, and chronic low-dose exposure effects~~ on aquatic life. This review emphasizes the need for regulatory measures~~. Addressing these gaps through pollutant-specific regulation, advanced ~~water~~wastewater treatment~~ technologies~~, and sustainable practices is essential to ~~minimize EP release into aquatic systems.~~mitigate EPs impacts. By ~~examining recent findings~~integrating mechanistic evidence across taxa, this study ~~underlines~~underscores the ~~importance of~~ urgent need for interdisciplinary ~~research to understand EP mechanisms and their impacts comprehensively. This study can underscore the importance of targeted research and policies~~approaches to safeguard aquatic ~~ecosystems and protect dependent human populations. Addressing this emerging pollutant contamination is essential for maintaining~~ biodiversity, ~~ecological~~ ecosystem balance, and public health~~, underscoring the interconnectedness of environmental and human wellbeing~~.

Keywords ~~Aquatic organisms •~~ Bioaccumulation • ~~Ecological impacts • Emerging pollutants • Toxicological effects~~Endocrine disruption • Ecotoxicological mechanisms • Oxidative stress • Wastewater treatment



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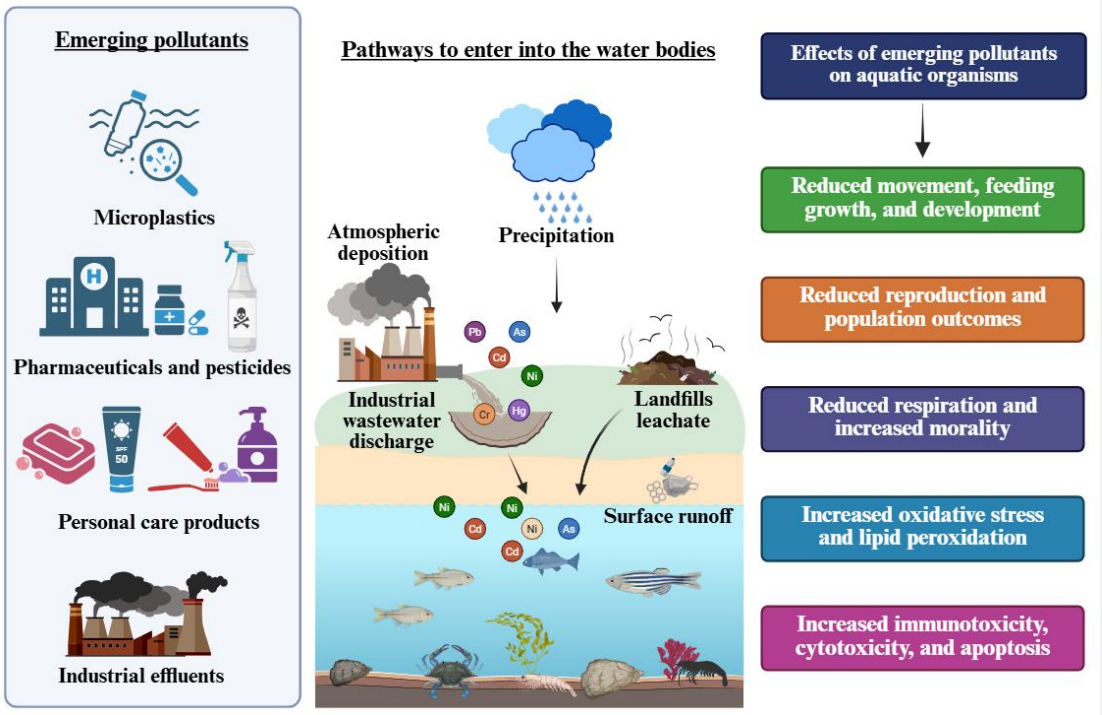

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127 **Graphical abstract**

1 Introduction

Owing to increasing urbanization and industrialization, it is well anticipated that the global population will continue to grow at the same pace, significantly increasing the number of emerging pollutants in the freshwater environment [1]. This uncontrolled discharge of emerging pollutants into water bodies represents a prominent threat to aquatic ecosystems. Emerging pollutants, also called contaminants of emerging concern [2] or micropollutants [3], are large groups of substances or chemicals generated by natural and anthropogenic activities. These include pharmaceuticals, personal care products, pesticides, industrial chemicals, and microplastics. Most are intended for utilization in industry, agriculture or as consumer products [4]. Although aquatic pollution has become a global issue in recent years, there is always a need to routinely monitor and execute plans to obtain solutions [5]. However, these pollutants are often left undetected or untreated by conventional wastewater treatment systems, which can pose a profound risk to aquatic organisms in the future because of their persistence, bioaccumulative properties, and toxicological impacts [6]. Unlike traditional pollutants, emerging pollutants have been identified only recently as environmental threats, yet their widespread use and environmental release have led to their accumulation across various aquatic environments globally [7]. In addition, these pollutants

~~further transform into their more toxic form upon exposure to different environmental conditions, such as temperature, salinity and pH, which leads to their accumulation in the food chain, causing severe effects on aquatic health even at low concentrations [8].~~

~~The impacts of EP on aquatic animals are far-reaching, contributing to bioaccumulation and biomagnification through the food chain. This phenomenon threatens biodiversity, ecosystem functionality, and human health, as many affected species are integral to human diets [9]. Various emerging pollutants, including endocrine disrupting compounds, pesticides, heavy metals, flame retardants, pharmaceuticals, personal care products, and micro- and nanoparticles, originate from natural events such as floods and weathering [10], as well as human-driven sources such as agriculture, wastewater, and industrial discharge [11]. These pollutants are introduced into the aqueous environment through multiple pathways, including direct discharge, runoff, atmospheric deposition, and precipitation [12]. Once in aquatic systems, they accumulate in the tissues of aquatic animals and travel through the food chain, posing risks to both ecosystems and human health [13]. Aquatic organisms also absorb pollutants through their gills, skin, and ingestion, affecting prey and predator species. The impact of emerging pollutants on aquatic life is extensive, affecting survival rates, gene expression, oxidative stress, and even DNA integrity [14]. Such exposure can also inhibit growth, disturb biochemical processes, alter behaviour and metabolism, and reduce reproductive success [10]. These consequences, including increased mortality rates, ultimately destabilize aquatic ecosystems [15]. Additionally, other environmental stressors, such as pollution, habitat loss and habitat degradation, as well as climate change, can also have significant negative impacts on aquatic ecosystems, impacting their overall health and biodiversity [10]. Therefore, understanding how these pollutants disrupt biological processes and address their toxicological and ecological impacts is essential for better regulation,~~

~~management, and practical strategies to mitigate their effects and safeguard aquatic ecosystems.~~

~~This comprehensive review aims to summarize and analyze the existing body of scientific knowledge on the potential toxicological impacts of several emerging pollutants on different aquatic organisms. By exploring the existing body of literature, this comprehensive review aims to achieve the following main objectives: 1) summarize current literature knowledge, firstly by providing an overview of different types of emerging pollutants existing in the environment, 2) evaluating their sources, fate of actions, and their mechanism of transport into the aquatic environment, 3) examining their toxicological implications on different aquatic organisms such as fish, amphibians, molluscs, crustaceans, mammals, reptiles, planktons, and small invertebrates highlighting their toxicity mechanism, 3) discussing ecological effects and 4) exploring how environmental change impacts EPs toxicity levels. This review not only provides a comprehensive resource for researchers, policymakers, and environmental advocates concerned with the EPs effects on aquatic ecosystems, especially aquatic organisms but also suggests some future perspectives for future research.~~

Aquatic ecosystems play a vital role in maintaining global biodiversity, food security, and ecological balance, yet they are increasingly threatened by contamination from a wide range of anthropogenic substances [1]. Among these contaminants, emerging pollutants (EPs), including pharmaceuticals and personal care products (PPCPs), pesticides, industrial chemicals, plastics, and heavy metals, have become a growing environmental concern due to their persistence, bioaccumulation potential, and toxicological impacts on aquatic life [2–5]. These compounds enter aquatic environments through various pathways such as municipal sewage, hospital discharges, agricultural runoff, and industrial effluents [6–8]. Once introduced, they can disrupt biochemical and physiological processes in aquatic organisms

[9], causing oxidative stress, endocrine disruption, immunotoxicity, reproductive impairments, and behavioral abnormalities, thereby affecting biodiversity and food web stability [6,10–12].

Despite extensive research on aquatic contamination, the complex nature of EPs poses significant challenges for environmental monitoring and assessment. Unlike conventional pollutants, EPs often occur at low concentrations yet produce chronic, multigenerational, and synergistic effects [13]. The coexistence of multiple contaminant classes amplifies their ecological risks, while their persistence and bioaccumulation tendencies make them difficult to eliminate through standard wastewater treatment processes [14]. These challenges underscore the urgent need for an integrated understanding of how diverse EPs collectively influence aquatic organisms at physiological, biochemical, and ecosystem levels.

Over the past decade, many studies have examined the presence and effects of EPs in aquatic environments, yet most remain limited in scope or mechanistic integration [15–21]. For example, Picinini-Zambelli [15] conducted a systematic review of the genotoxic potential of selected EPs, including pharmaceuticals, personal care products, and industrial chemicals. Their analysis demonstrated that many of these substances induce DNA damage in aquatic organisms, primarily detected through comet assays. However, their work was restricted to a small number of studies and focused solely on genotoxic endpoints, providing little insight into other mechanisms such as oxidative stress, endocrine disruption, or immunotoxicity. Similarly, other studies have explored specific pollutant categories, for instance, pharmaceuticals [22], pesticides [16,23], microplastics (MPs) [17], and heavy metals [24,25], but have largely analyzed them in isolation, without integrating their cumulative or synergistic effects across multiple contaminant groups and taxa.

This narrow, pollutant-specific approach has created a significant knowledge gap in understanding how multiple emerging pollutants interact and collectively affect aquatic organisms at physiological, biochemical, and molecular levels. The absence of an integrated synthesis across contaminant classes has limited the identification of shared toxicity mechanisms, cross-taxon biological responses, and potential biomarkers of exposure.

Recent investigations have expanded our understanding of EPs by addressing mixture toxicity, low-dose chronic effects, and cross-species responses [26,27]. These studies revealed that even trace levels of pharmaceuticals, pesticides, and microplastics can jointly trigger oxidative, endocrine, and genetic disturbances, challenging earlier assumptions of concentration-dependent toxicity [28,29]. Such advances reinforce the need for integrated assessments like the present review.

To address these limitations, the present study brings together and critically synthesizes studies that have investigated the toxicological effects of major groups of emerging pollutants, pharmaceuticals, pesticides, industrial chemicals, plastics, and heavy metals on a wide range of aquatic organisms. By compiling and interpreting findings from diverse experimental studies, this review provides a consolidated understanding of how these pollutants affect various aquatic species through key toxicity mechanisms, including oxidative stress, genotoxicity, immunotoxicity, and endocrine disruption. This integrated approach offers a clearer perspective on how multiple pollutant classes, often acting simultaneously in the environment, impair the physiology, reproduction, and survival of aquatic species.

The main objectives of this study are to (a) summarize the major types, sources, and environmental pathways of emerging pollutants; (b) evaluate and compare the available evidence on their toxicological effects across different aquatic taxa while understanding their

shared and distinct mechanisms of toxicity among pollutant categories; and (c) identify research gaps and emerging trends that can guide future ecotoxicological investigations. By doing so, the study serves as a valuable resource for guiding future studies, improving risk assessment, and informing strategies to protect aquatic ecosystems.

2 Overview of the types of emerging pollutants

~~2.1 Pharmaceuticals and personal care products (PPCPs)~~

~~Pharmaceuticals and personal care products (PPCPs) have become significant pollutants in aquatic ecosystems. Commonly available PPCPs include antibiotics, painkillers, anti-inflammatory drugs, hormones, and cosmetic ingredients such as fragrances and preservatives [16]. These chemicals enter soil and water bodies primarily through domestic household sewage, hospital waste, and effluents from wastewater treatment plants [17]. For example, ciprofloxacin, tetracycline, ampicillin, trimethoprim, erythromycin and trimethoprim/sulfamethoxazole are widely used in the healthcare and agriculture sector, and their residues often remain in treated sewage, contributing to the development of antibiotic resistance in aquatic microbial communities [18]. Painkillers and anti inflammatory drugs, such as ibuprofen, naproxen, and diclofenac, are frequently detected in rivers and lakes, where they have been shown to disrupt fish liver [19] and kidney functions [20].~~

~~Hormonal pollutants, both naturally and artificially derived estrogens, androgens, diethylstilbesterol, stigmasterol, and 17 β estradiol accumulate in water bodies by leaching, erosion and sewage discharge and act as pollutants for aquatic animals [21]. They pose a severe risk to aquatic animals by interfering with the endocrine systems of fish and amphibians, causing reproductive disruptions, including feminization of male fish, decreased fertility, and abnormal development [22]. In personal care products such as toothpaste, soaps, shampoo, hand sanitizers, and other cosmetic products, chemicals such as triclosan (an antibacterial agent) and parabens (preservatives) are frequently detected in sediments and~~

surface waters, where they contribute to oxidative stress and immune suppression in aquatic species [23]. The widespread use of these PPCPs in modern society makes them a continuous source of pollution, with complex and long term impacts on aquatic life.

2.2 Pesticides

Pesticides, which are widely used in agriculture to control pests, weeds, and fungi, constitute another major class of emerging pollutants [24]. The most common pesticides in aquatic ecosystems are herbicides, insecticides, and fungicides. Herbicides such as atrazine and glyphosate are used extensively to control weeds in agricultural fields but often end up in nearby rivers and lakes through processes such as surface runoff, drift, and vertical transport [25]. These chemicals disrupt the photosynthesis process in aquatic plants [26], leading to reduced oxygen production and growth [27], thereby harming aquatic organisms that are reliant on healthy plants for food and habitat.

Insecticides, such as organophosphates and neonicotinoids, are particularly harmful to nontarget species, including aquatic invertebrates and fish [28]. Neonicotinoids, for example, cause neurotoxicity in aquatic insects, leading to population declines due to insect paralysis and death that affect the entire food chain [29]. Fungicides such as chlorothalonil are widely used in agriculture to protect crops from fungal diseases, silviculture, and urban settings but can cause high toxicity in fishes, crustaceans, amphibians and aquatic invertebrates [30]. The runoff of these pesticides into aquatic systems often results in long term contamination, affecting biodiversity and ecosystem balance [5]. Even at low concentrations, they can bioaccumulate in the tissues of aquatic animals such as fishes, causing chronic health impacts and reproductive dysfunction [31].

2.3 Industrial chemicals

Industrial chemicals, such as flame retardants, solvents, and surfactants, frequently enter aquatic systems through industrial runoff, accidental spills, and improper waste disposal

[32]. Flame retardants, such as polybrominated diphenyl ethers (PBDEs), are used in many household materials, such as textiles, electronics, and furniture, to reduce fire risk. However, owing to their bioaccumulation tendencies, they are persistent pollutants in aquatic environments [33]. PBDEs accumulate in sediments, water, and dust and bioaccumulate in aquatic animals, especially in their internal organs instead of muscle tissue [34], leading to developmental, reproductive, and neurological problems in fish and other wildlife [35]. These compounds also exhibit endocrine disrupting properties, interfering with hormone regulation and reproductive health [36]. Solvents, which are used in industries ranging from manufacturing to cleaning, can dissolve in water and cause toxicity to aquatic organisms, especially fish and invertebrates [37]. Surfactants such as linear alkylbenzenesulfonate (LAS), commonly found in detergents and cleaning agents, reduce surface tension in water [38], can easily penetrate cell membranes, and interfere with the respiration of aquatic organisms, particularly by significantly damaging gill membranes and causing loss of sight in fishes [39].

2.4 Plastics and microplastics

Plastic pollution is a growing global environmental concern that significantly impacts aquatic environments. Plastics, especially microplastics (particles smaller than 5 mm) and nanoplastics, are now ubiquitous in oceans, rivers, and lakes [40]. These include polystyrene, polyethene, polyvinyl chloride, polypropylene, polyethene terephthalate, nylon and other small polymers [41]. These tiny plastic fragments originate from the breakdown of larger plastic debris [42], such as packaging, bottles, and fishing gear, as well as from the shedding of synthetic fibres during washing and microbeads used in personal care products [43]. Microplastics persist in aquatic environments for decades because of their resistance to degradation. Aquatic organisms, including fish, mussels, and zooplankton, ingest these particles, mistaking them for food, consequently representing the route through which the

~~microplastic can enter the food chain and transfer these particles to higher trophic levels [44]. Once ingested, microplastics can cause physical harm, including blockage in the digestive tract and reduced feeding efficiency [41]. More alarmingly, particulate plastics, including microplastics and nanoplastics, can adsorb toxic chemicals from the surrounding environment, turning them into vectors for transferring harmful substances into the food chain [45]. Recent studies have shown that microplastics can impair aquatic animal growth, reproduction, and immune function, contributing to population declines in sensitive or specialized species [46].~~

~~2.5 Metals~~

~~Heavy metals, such as mercury, cadmium, arsenic, nickel, cobalt, chromium, copper, and lead, are persistent pollutants that pose significant risks to aquatic organisms [47]. These metals enter water bodies primarily through mining [48], industrial effluents [49], and improper disposal of electronic waste [50]. Mercury, for example, is released into the aquatic environment during industrial processes such as coal burning and mining, where it is converted into a highly toxic form of methylmercury, organic thiols, or other reduced sulfur ligands that accumulate in fish and other aquatic organisms [51]. Methylmercury is known for its neurotoxic effects, which impair the nervous system of fish and can cause behavioural abnormalities, reproductive failure, and even death [52]. Cadmium and lead, often released from industrial effluents, also accumulate in the tissues of aquatic organisms, causing toxicity at various biological levels. Cadmium exposure is linked to kidney, gill, and liver damage in fish [53], whereas lead can interfere with calcium metabolism, leading to vertebral column, nuclear and cellular structural deformities or abnormalities [54]. Heavy metals are known to affect reproductive success, reduce growth rates, and weaken the immune systems of exposed species [55]. Because heavy metals do not breakdown in the environment, they remain in~~

~~aquatic ecosystems for extended periods, bioaccumulating and biomagnifying through the food chain [56], ultimately posing risks to human health as well [57].~~

EPs include pharmaceuticals and personal care products (PPCPs) [30], pesticides [31], industrial chemicals [32], plastics [33], and heavy metals [34] that persist in aquatic ecosystems and pose serious ecological and health threats. PPCPs such as antibiotics, anti-inflammatory drugs, hormones, and cosmetic ingredients enter aquatic environments through domestic sewage, hospital waste, and wastewater effluents [35]. Antibiotics are also extensively used in the agricultural sector (livestock and poultry), contributing to antibiotic resistance, oxidative stress, and endocrine disruption in aquatic fauna [22,36–40]. Pesticides, including herbicides like atrazine and glyphosate [41], insecticides such as organophosphates and neonicotinoids [42], and fungicides like chlorothalonil [43], reach water bodies through agricultural runoff and drift [5], impairing photosynthesis [44], causing neurotoxicity [45], and bioaccumulating through food webs [46]. Industrial chemicals such as flame retardants (PBDEs), solvents, and surfactants enter through industrial discharges and spills, persist in sediments, and disrupt hormonal and metabolic functions in aquatic organisms [47–53]. Plastic pollutants, particularly micro- and nanoplastics [33] composed of thermoplastics and thermosetting polymers [54], are pervasive and persistent. They act as vectors for adsorbed contaminants, host pathogenic and antibiotic-resistant biofilms on their hydrophobic surfaces [55–57], and release polymer additives such as plasticizers and flame retardants that can be carcinogenic or endocrine-disrupting [58]. Their hidden indirect effects, widespread detection across ecosystems and even human tissues, and poorly understood behavior make them one of the most alarming emerging pollutants [59]. Heavy metals such as mercury, cadmium, arsenic, nickel, chromium, copper, and lead originate from mining [60], industrial effluents [61], and e-waste [62], causing neurotoxicity [63], oxidative stress [64], reproductive failure

[65], and long-term bioaccumulation through the food chain [66], ultimately threatening both ecosystems and human health [67]

3 Pathways and mechanisms of EP entry in aquatic ecosystems

~~Emerging pollutants are a significant concern for environmental conditions [58], as they adversely affect the whole ecosystem [59]. These pollutants can enter the environment directly or indirectly through various pathways, including agricultural runoff [60] and household, domestic, sewage, industrial, and hospital effluent [61], leading them to enter aquatic systems [59]. Their routes to enter water bodies can be strongly influenced by the mechanism through which they can discharge [62] and their physio-chemical properties, such as volatility, polarity, adsorption properties, persistence, and properties of interacting compartments [3]. Agricultural runoff, for example, is a significant source of emerging pollutants such as pesticides, herbicides, and fertilizers. When water flows across the surface due to rain, irrigation or water released from another source onto the surface, the chemicals applied to crops are washed down into nearby rivers, lakes, and streams, carrying harmful substances that can disrupt aquatic ecosystems [63]. For example, pesticides in runoff can result in pesticide pollution in streams, lakes, ponds, wells, and rivers, which can harm aquatic animals [64], whereas fertilizers contribute to nutrient pollution, leading to harmful algal blooms and oxygen depletion in water bodies, which can kill fishes and reduce their essential habitats.~~

~~Owing to their discharge from single points, these emerging pollutants can also be called point source pollutants [65], whereas those that cannot be discharged from a single source are known as nonpoint source pollutants [66]. Agricultural runoff, deposition by air, landfill leachate [67], and vehicle emissions [68] are examples of nonpoint source pollutants [69], which are easy to maintain and monitor, whereas point source pollutants are easy to regulate, monitor, and mitigate. Wastewater treatment plants (WWTPs) are yet another~~

critical source of pollution. Although these facilities are designed to treat sewage and industrial waste, many emerging pollutants, particularly pharmaceuticals and personal care products (PPCPs), are only partly removed during the treatment process [2]. This results in the release of treated effluents containing residual antibiotics, hormones, and other chemicals into aquatic systems [70]. These pollutants can also be conveyed into water bodies due to overflow from treated sludge. They can cause endocrine disruption in aquatic animals [62], reduce reproductive success [71], and lead to the development of antibiotic resistant bacteria in the environment [17].

Industrial effluents and urban runoff also contribute to aquatic pollution. Industrial processes often involve the use of hazardous chemicals such as solvents, flame retardants, and heavy metals, which are discharged directly or indirectly into water bodies [72]. These pollutants can persist in the environment, bioaccumulate in aquatic organisms, and cause long-term ecological damage [7]. Similarly, during rainfall, stormwater washes away these pollutants from nearby urban environments (including roads, parking lots, lawns and other infrastructure elements) into rivers and lakes [73]. This urban runoff often carries oil, grease, microplastics, heavy metals, and chemicals from household products into the water, posing severe threats to aquatic life [29].

Pollutants can also reach water bodies through atmospheric deposition [74], a process by which airborne pollutants are deposited onto land or water from the atmosphere [75]. These pollutants can be discharged and deposited into water systems [76] through combustion [77], industrial gas, and pesticide spraying, which are all routes for aerial transporters [78]. Airborne pollutants such as heavy metals (e.g., mercury), pesticides, and industrial chemicals can travel long distances before settling on water bodies, especially in regions far from their source, making atmospheric deposition a significant global issue. Mercury, one of the most concerning pollutants, is released into the atmosphere primarily by

coal-burning power plants and industrial activities and can travel thousands of kilometres before being deposited into lakes, rivers, and oceans [79]. Once in aquatic systems, it transforms into a highly toxic substance, methylmercury, which bioaccumulates in fish and other aquatic organisms, posing severe health risks to wildlife and humans [46].

Similarly, persistent organic pollutants (POPs) can volatilize into the atmosphere at relatively high temperatures and be carried over large distances before being redeposited [80]. When these pollutants are deposited into aquatic systems, they can accumulate in sediments, bioaccumulate in organisms, and cause long-term environmental damage, even in remote and pristine environments such as Antarctica [80]. Atmospheric deposition thus represents a pathway through which pollutants not directly discharged into water still find their way into aquatic ecosystems [81].

3.1 Bioaccumulation and biomagnification

Bioaccumulation refers to the uptake of substances from food and water [82]. Simply put, the uptake of harmful particles or substances directly from the environment and the accumulation or accumulation of these pollutants in the tissues of organisms over time are complex processes [83]. Many emerging pollutants, including heavy metals, pharmaceuticals, and persistent organic chemicals, are not quickly metabolized or excreted by aquatic organisms [84]. As a result, these pollutants are not easy to digest and accumulate in the bodies of fish, molluscs, and other aquatic species, as they feed on or absorb contaminants from their surroundings [1]. For example, mercury bioaccumulates in fish [85], particularly in long-lived species such as tuna, where it reaches levels that can harm both the fish and the humans who consume it [86].

After bioaccumulation [87], the pollutant particles or substances are transferred from lower to higher trophic levels [83] because of their magnified nature. These EPs can be transferred through WWTPs, precipitation, sedimentation, or aerial deposition. POPs strongly

~~influence the structure of the aquatic food web. First, their size should be small. However, at a relatively high [88] of the trophic level, their size increases with increasing effects on those organisms [89]. The first organisms of the aquatic food chain are plankton, which can include phytoplankton, zooplankton, and bacterioplankton. Here, the first producers are phytoplankton, and the zooplankton are the first consumers [90] because they can feed on these phytoplankton [91]; the contaminants are then absorbed by the primary consumer (zooplankton), and these particles accumulate, are magnified, and are transferred to the following trophic levels. Zooplanktons are long lived baselines for the aquatic food web. These can be consumed by molluscs, mussels, clams, crustaceans, shrimp, and crabs [92]. They can be fed by small fishes or top predators such as common scoters, which are secondary consumers, and tertiary consumers are larger fish (cods) that can be fed by sea birds, aquatic mammals and seals, or octopuses (molluses). After that, the top predators, which are seals or sharks, consume large fish and then decompose, which can breakdown the plants and animals and recycle the nutrients back into the ecosystem. The food web plays a vital role in the ecosystem because of the energy flow from one trophic level to the next, such as in recycling nutrients, reducing the number of dead organisms, maintaining biodiversity, balancing ecology through population control and maintaining the health of the ecosystem. They decompose these organisms [36] and form organic material that bacteria can use to form nutrients [93]. These nutrients can be sediments or organic nutrients in the ecosystem [94].~~

~~Biomagnification occurs when the concentration of pollutants increases as they move up the food chain [95]. This generally occurs because predators consume multiple contaminated prey containing bioaccumulated pollutants, leading to relatively high concentrations of top predators. For example, tiny aquatic organisms (primary consumers) that absorb microplastics or other pollutants are eaten by fish (intermediate predators), which~~

~~are then consumed by larger fish, birds, or marine mammals (top predators) with a high tendency for bioaccumulation [96]. At each trophic level, the concentration of these pollutants increases, making the top predators particularly vulnerable to toxic effects [88]. This process threatens biodiversity and poses significant risks to human health, as humans are often at the top of aquatic food chains through the consumption of seafood [97]. Once pollutants enter aquatic environments, they are subject to bioaccumulation and biomagnification, even at low concentrations, two processes that significantly amplify their impact on ecosystems.~~

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Similarly, persistent organic pollutants (POPs) can volatilize into the atmosphere at relatively high temperatures and be carried over large distances before being redeposited [90]. When these pollutants are deposited into aquatic systems, they can accumulate in sediments, bioaccumulate in organisms, and cause long-term environmental damage, even in remote and pristine environments such as Antarctica [90]. Atmospheric deposition thus represents a pathway through which pollutants not directly discharged into water still find their way into aquatic ecosystems [91].

3.1 Bioaccumulation and biomagnification

Bioaccumulation refers to the uptake of substances from food and water [92]. Simply put, the uptake of harmful particles or substances directly from the environment and the accumulation or accumulation of these pollutants in the tissues of organisms over time are complex processes [93]. Many EPs, including heavy metals, pharmaceuticals, and persistent organic chemicals, are not quickly metabolized or excreted by aquatic organisms [94]. As a result, these pollutants are not easy to digest and accumulate in the bodies of fish, molluscs, and other aquatic species, as they feed on or absorb contaminants from their surroundings [1]. For example, mercury bioaccumulates in fish [95], particularly in long-lived species such as tuna, where it reaches levels that can harm both the fish and the humans who consume it [96].

After bioaccumulation [97], the pollutant particles or substances are transferred from lower to higher trophic levels [93] because of their magnified nature. These EPs can be transferred through WWTPs, precipitation, sedimentation, or aerial deposition. POPs strongly influence the structure of the aquatic food web. First, their size should be small. However, at a relatively high [98] of the trophic level, their size increases with increasing effects on those organisms [99]. The first organisms of the aquatic food chain are plankton, which can include phytoplankton, zooplankton, and bacterioplankton. Here, the first producers are phytoplankton, and the zooplankton are the first consumers [100] because they can feed on these phytoplankton [101]; the contaminants are then absorbed by the primary consumer (zooplankton), and these particles accumulate, are magnified, and are transferred to the following trophic levels. Zooplanktons are long-lived baselines for the aquatic food web. These can be consumed by molluscs, mussels, clams, crustaceans, shrimp, and crabs [102]. They can be fed by small fishes or top predators such as common scoters, which are secondary consumers, and tertiary consumers are larger fish (cods) that can be fed by sea birds, aquatic mammals and seals, or octopuses (molluscs). After that, the top predators, which are seals or sharks, consume large fish and then decompose, which can breakdown the plants and animals and recycle the nutrients back into the ecosystem. The food web plays a vital role in the ecosystem because of the energy flow from one trophic level to the next, such as in recycling nutrients, reducing the number of dead organisms, maintaining biodiversity, balancing ecology through population control and maintaining the health of the ecosystem. They decompose these organisms [50] and form organic material that bacteria can use to form nutrients [103]. These nutrients can be sediments or organic nutrients in the ecosystem [104].

Biomagnification occurs when the concentration of pollutants increases as they move up the food chain [105]. This generally occurs because predators consume multiple

contaminated prey containing bioaccumulated pollutants, leading to relatively high concentrations of top predators. For example, tiny aquatic organisms (primary consumers) that absorb MPs or other pollutants are eaten by fish (intermediate predators), which are then consumed by larger fish, birds, or marine mammals (top predators) with a high tendency for bioaccumulation [106]. At each trophic level, the concentration of these pollutants increases, making the top predators particularly vulnerable to toxic effects [98]. This process threatens biodiversity and poses significant risks to human health, as humans are often at the top of aquatic food chains through the consumption of seafood [107]. Once pollutants enter aquatic environments, they are subject to bioaccumulation and biomagnification, even at low concentrations, two processes that significantly amplify their impact on ecosystems.

The mechanisms by which different EPs induce ecotoxicity vary considerably depending on their physicochemical characteristics, size, and morphology. For example, MPs and NPs often cause mechanical stress such as intestinal blockage, tissue abrasion, or false satiation in aquatic organisms [108,109], whereas MPs can enter cells *via* endocytosis and trigger oxidative stress and inflammation [110–112]. In contrast, chemical pollutants like pharmaceuticals, pesticides, and heavy metals primarily act through molecular pathways involving receptor binding, enzyme inhibition, endocrine disruption, or oxidative damage [113–115]. The size and shape of particulate pollutants further influence their toxic potential, fibrous particles can entangle or damage tissues, while spherical particles are more easily ingested and transported across biological membranes [111,116]. Understanding these mechanistic differences is essential for accurately assessing ecological risks and designing pollutant-specific management strategies.

4 Toxicological impacts of emerging pollutants on different aquatic animals

4.1 Impacts on fishes

Emerging pollutant exposure can affect aquatic organisms in several ways, such as immune system suppression and reproductive and developmental abnormalities, which might even cause large-scale mortalities in aquatic species, mainly attributed to the reduced amount of dissolved oxygen, changes in the pH of the water and the presence of toxic chemicals [10]. Exposure to certain microplastics and nanoplastics (MPs and NPs) has been shown to affect liver function in *Danio rerio* [98], which plays a vital role in the digestive system of the fish and affects the transformation, accumulation, and elimination of various substances [99]. In some other cases, such exposure has also disrupted lipid and energy metabolism in *Sparus aurata* and *Dicentrarchus labrax* [98], as well as altered gene expression, lipid peroxidation, and enzyme activities in *Larimichthy crocea* [100] and *Carassius carassius* [98]. Microplastic exposure has also been shown to deteriorate fish muscle quality [101], induce tissue damage, affect the antioxidant response, alter AChE activity, and influence growth-related genes in fish muscle tissue [102]. Haematological impacts are similarly notable, with shifts in red and white blood cell counts, haemoglobin concentrations, and haematocrit levels in *Oncorhynchus mykiss* and *Oreochromis niloticus* [103]. Additionally, MPs and NPs have been linked to reduced toll-like receptor expression [104] and interferon-related gene activity [105], increasing disease susceptibility without necessarily increasing mortality rates [106]. Certain polystyrene microplastics alter the gastrointestinal tract, leading to a reduction in the reproductive rate, vitellogenesis gene expression, oxidative stress, sex hormone levels and apoptosis [107] (Table 1) .

Pesticide exposure negatively affects fish growth, physiology, reproduction, and immunity [108] (Fig. 1). These effects include altered hematobiochemical parameters [109]; histopathological changes in organs such as the gills, liver, brain, kidney, and gonads [110]; and behavioral and oxidative stress responses, hormonal changes, and genotoxic impacts [111]. The entry of specific pesticides through the gills, skin, and digestive system leads to

apoptosis, neurophysiological disruptions, changes in swimming behaviour, morphological shifts, and increased reactive oxygen species (ROS) that damage DNA, cellular transport mechanisms, and biochemical pathways [112]. Certain pesticides, such as thiamethoxam, chlorpyrifos and cypermethrin, cause specific cellular and organ damage, such as necrosis and degeneration of tissues in organs such as the liver, kidney, and ovary [113]. Other chemical exposures, such as imidacloprid and propoxur mixtures, have been shown to inhibit AChE activity in the brain, causing oxidative stress in proteins and lipids and increasing reactive oxygen species production as well as CAT activity in fish muscles [108]. In addition, dieldrin exposure can change the expression of neurotransmitter receptors such as GABA [114], which is a luteinizing hormone stimulator often used for dopamine signalling to inhibit LH secretion [115].

On the other hand, heavy metals, including arsenic, cadmium, copper, and zinc, induce significant toxic effects, including organ damage, genotoxicity, oxidative stress, and decreased immune parameters. For example, exposure to silver nanoparticles alters swimming behaviour and appetite in *Clarias gariepinus* [116], whereas copper exposure in *Danio rerio* reduces swimming ability and prompts DNA damage through lipid oxidation and decreased antioxidant enzyme activity [117]. Pharmaceutical and personal product (PPCP) contaminants, such as diclofenac, carbamazepine, and triclosan, at various concentrations in various fish species induce spine malformations, pericardial edema, yolk abnormalities, and oxidative damage in fish [16] (Table 2). PPCP exposure also inhibits essential signalling pathways, impairing hormonal and immune system functions [118]. For example, diclofenac exposure increases AChE activity in *Carassius auratus*, whereas carbamazepine decreases AChE activity, affecting neurotoxicity markers and promoting oxidative stress [119]. Additionally, in *Pseudobatos horkelii*, PPCPs are transferred from mothers to offspring during vitellogenesis, and their exposure disrupts redox balance and reduces growth [120].

EPs such as MPs, pesticides, pharmaceuticals, nanoparticles (NPs), and heavy metals exert multifaceted toxicological impacts on fishes, affecting biochemical, physiological, and ecological functions. Exposure can disrupt immune homeostasis, reproduction, and development, potentially leading to population declines, especially under compounded stressors such as hypoxia, pH fluctuations, and chemical toxicity [6]. MPs and NPs interfere with hepatic metabolism and energy balance, as shown in *Danio rerio* [117,118], *Sparus aurata*, and *Dicentrarchus labrax* [119], by impairing lipid turnover and antioxidant defenses [120–122]. They also altered gene expression and induced oxidative damage and tissue degeneration [123,124]. Hematological disturbances, including changes in erythrocyte count, hemoglobin content, and immune cell ratios have been reported in *Oncorhynchus mykiss* [125], *Etroplus suratensis* [126], and *Oreochromis niloticus* species [120]. Pesticides elicited oxidative stress, histopathological lesions, and behavioural impairments in multiple species [127,128]. Commonly used compounds like thiamethoxam, chlorpyrifos [129], and cypermethrin induced apoptosis and neurotoxicity through AChE inhibition and ROS accumulation, damaging liver, kidney, and gonadal tissues [130]. Mixtures of neonicotinoids and carbamates (*e.g.,* imidacloprid + propoxur) further amplify oxidative injury and disrupt neurotransmission [131].

The key mechanistic pathways underlying these toxic responses are summarized in Fig. 1, which integrates findings from multiple studies (Table 1) on oxidative, endocrine, and immune disruption mechanisms. As illustrated, EPs activated oxidative stress responses *via* elevated antioxidant enzyme activities such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) and excessive reactive oxygen species (ROS) generation [132,133]. These molecular alterations cascade into hormonal and immunological dysregulation, manifested as reduced thyroid [134] and reproductive hormones (estradiol, testosterone) [135], increased inflammatory cytokines (TNF-α, IL-6) [125], and disrupted

metabolic homeostasis. The resulting tissue and behavioural effects such as necrosis, altered swimming [136,137], and reduced feeding, impair growth [138,139] and reproductive capacity [135,140], demonstrating how pollutant-specific mechanisms converge on shared oxidative and endocrine pathways linking biochemical disruption to ecological decline (Fig. 1).

Heavy metals and NPs cause organ damage, genotoxicity, and reduced immune competence in fishes such as *Clarias gariepinus* [141] and *Danio rerio* [142]. For instance, silver nanoparticles impair swimming and feeding [141], while copper exposure elevated lipid peroxidation and DNA damage [142]. PPCPs such as diclofenac, carbamazepine , and triclosan induce skeletal malformations, pericardial edema [143,144], and oxidative stress [145] across multiple fish species [30]. Neurotoxicity, *via* modulation of AChE activity [146], and transgenerational transfer of PPCPs during vitellogenesis further underline their long-term ecological risks. However, many experimental doses ($\geq$1 mg L^{-1}) remain orders of magnitude higher than those detected in aquatic environments, indicating that field-level exposures are more likely to cause chronic, rather than acute, physiological disruptions [147,148]. Overall, the toxicological evidences showed that EPs disrupt fish physiology through oxidative and endocrine mechanisms, yet the ecological relevance of many experimental exposures remains limited. Future studies should compare effect thresholds with measured environmental concentrations, highlight mixture toxicity, and adopt chronic, low-dose exposure designs to bridge the lab–field gap.

690 **Table 1** Overview of the toxicological effects of different emerging pollutants on different fish species

Compound	Size/ Concentration ~~(mg kg^{-1})~~	Nominal vs Measured	Exposure duration	Exposure Type (Experimental/Field)	Biomarker	Tissue	Species name	~~Effects~~ Effects
Florfenicol ~~(antibiotics)~~ and Oxytetracycline	5 ~~mg kg^{-1}~~ and 100	Nominal	12 weeks	Experimental	AST, ALT	Liver, kidney, blood	~~Fish—O.~~ *Oncorhynchus niloticus*	• Phagocytic activity ↓ • Creatinine ↓
Zinc oxide NPs	1000 ~~mg kg^{-1}~~	Nominal	10 days	Experimental	GST, CAT, EROD	Intestine, gill, liver	*Oncorhynchus mykiss* (~~*Salmonid*~~ freshwater fish)	• CYP1A-mediated ethoxy resoru O-deethylase activity in the l tissue ↑
~~AgNP~~ AgNPs	~~25.~~0 ~~μg L^{-1}~~ .025	Nominal	15 days	Experimental	CAT, SOD, GSH-Px, AMS, TPS, and LPS	Liver, intestine and brain	~~Fish—~~ *Prochilodus lineatus* (freshwater fish)	• Hematological and plas biochemical parameters ↑ • Glycogen in liver and mus muscle protein concentration ↑ • All antioxidant enzyme activitie • ACAP in the liver ↓ • CFU (colony forming units) in mucus ↓

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Estradiol
4 ng L-1 0.000004
Nominal
28 days
Experimental
VTG, CYP450, SOD
Brain, liver, ovotestis
Fish
Induced delayed effects in a
brain, liver and ovotestis proteo
Insulin-like growth factor
binding-protein 3 down regulate
Diverse functions of prot
involved in reproduction and
metabolism
Antidepressant fluoxetine Fluoxetine
0.3 µg L-1 0003 and 0.003
Nominal
15 days
Experimental
SOD, CAT, LDH, AChE, ERDO
Muscles Muscle, liver
Fish
Growth ↓
Biotransformation ↓
Caused oxidative stress
AgNPs
20 and 40 nm / 10 (0.01 and 100 µg L- 0.1)
Nominal
15 days
Experimental
AST, ALT
Liver
Catfish
Concentrations of liver enzymes
Aspartic aminotransferase, Ala
aminotransferase, alka
phosphatase and Total lipids
Glucose total proteins, albu
and globulin concentrations
Polyethylene MPs
<5 µm
Nominal
14 days
Experimental
GST, GPx, MDA
Blood, Liver, Gills, kidney
Oncorhynchus mykiss
Impaired innate immunity
Increased presence of mucin
cells
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PVC	34.89 ~~mg L^{-1}~~	Nominal	10 days	Experimental	AChE, MDA, SOD, CAT	Blood, brain, gills	*Etroplus suratensis*	• Leukocytes ↓ • Disruption of balance antioxidant enzymes capacity ↑ • Expression of pro-inflamma genes ↑ • Kidney damage ↑ • Lipid peroxidation ↑ • Fin flickering, burst swimming, jerking movement ↑ • RBC and WBC ↓
Chlorpyrifos (CPF)	~~5 µg L^{-1}~~ ~~0.005 and 0.08~~	Nominal	96 hours	Experimental	AChE	Muscles, brain, liver	*Solea senegalensis* (marine fish)	• Muscle tissue cholineste activity ↓
Polyethylene MPs	0.3 mm	Nominal	9 months	Experimental	VTG1, Hydroxysteroid dehydrogenase	Gonads, liver, brain, pituitary	*Gadus morhua* (marine fish)	• Expression of the estrogen rece (*esr1*) in the pituitary ↓ • Luteinizing hormone rece (LHR) showed an increase relative expression level • No effect on feed intake nutrient digestibility • Upregulation of *vtg1* liver

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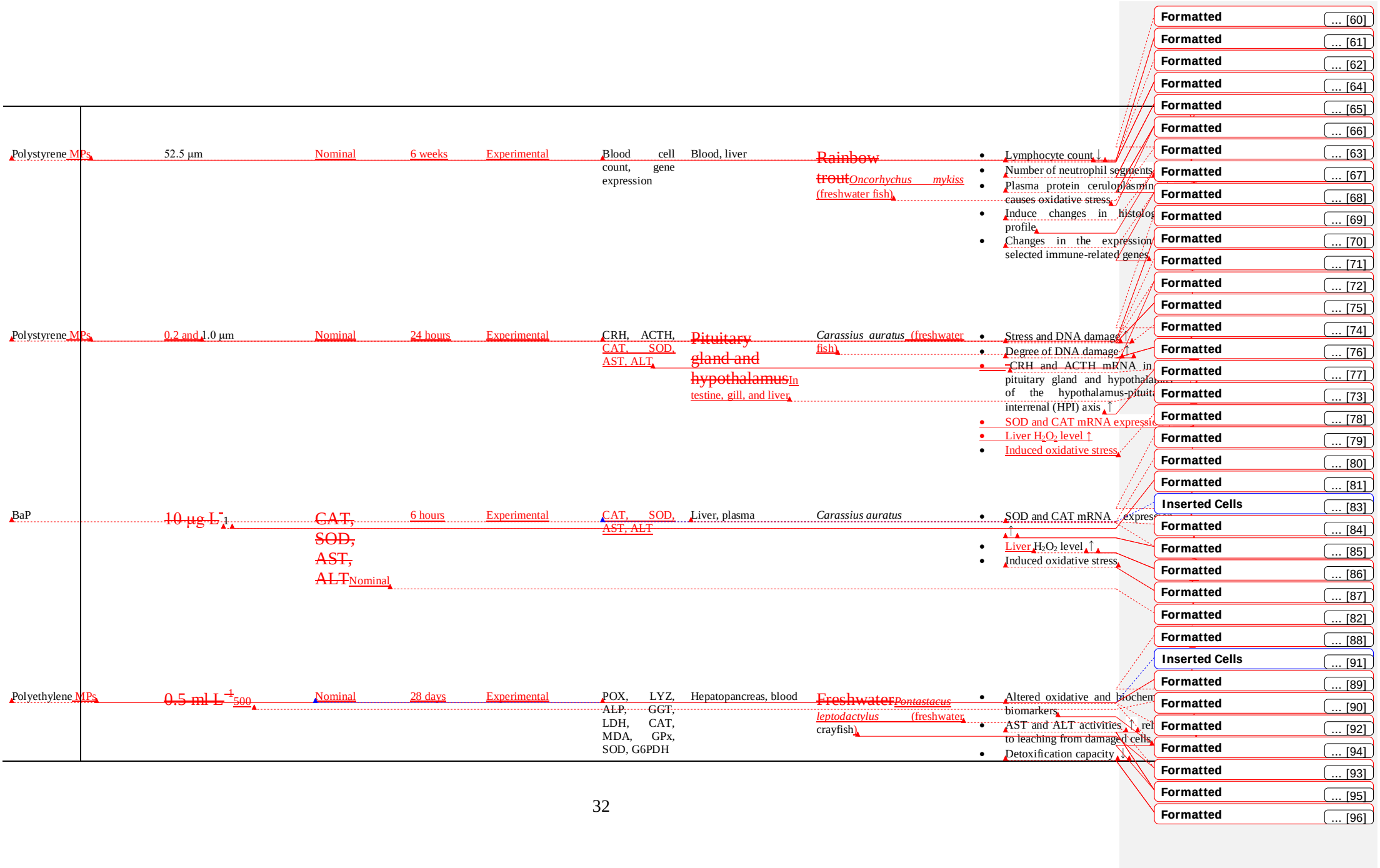
Polystyrene MPs
52.5 µm
Nominal
6 weeks
Experimental
Blood cell count, gene expression
Blood, liver
Rainbow trout Oncorhychus mykiss (freshwater fish)
Lymphocyte count ↓
Number of neutrophil segments
Plasma protein ceruloplasmin causes oxidative stress
Induce changes in histolog profile
Changes in the expression selected immune-related genes
Polystyrene MPs
0.2 and 1.0 µm
Nominal
24 hours
Experimental
CRH, ACTH, CAT, SOD, AST, ALT
Pituitary gland and hypothalamus In testine, gill, and liver
Carassius auratus (freshwater fish)
Stress and DNA damage ↑
Degree of DNA damage ↑
CRH and ACTH mRNA in pituitary gland and hypothala
of the hypothalamus-pituit
interrenal (HPI) axis ↑
SOD and CAT mRNA expressi
Liver H2O2 level ↑
Induced oxidative stress
BaP
10 µg L-1
CAT, SOD, AST, ALT Nominal
6 hours
Experimental
CAT, SOD, AST, ALT
Liver, plasma
Carassius auratus
SOD and CAT mRNA expres
↑
Liver H2O2 level ↑
Induced oxidative stress
Polyethylene MPs
0.5 ml L-1 500
Nominal
28 days
Experimental
POX, LYZ, ALP, GGT, LDH, CAT, MDA, GPx, SOD, G6PDH
Hepatopancreas, blood
Freshwater Pontastacus leptodactylus (freshwater crayfish)
Altered oxidative and biochem
biomarkers
AST and ALT activities ↑ rel
to leaching from damaged cells
Detoxification capacity ↓
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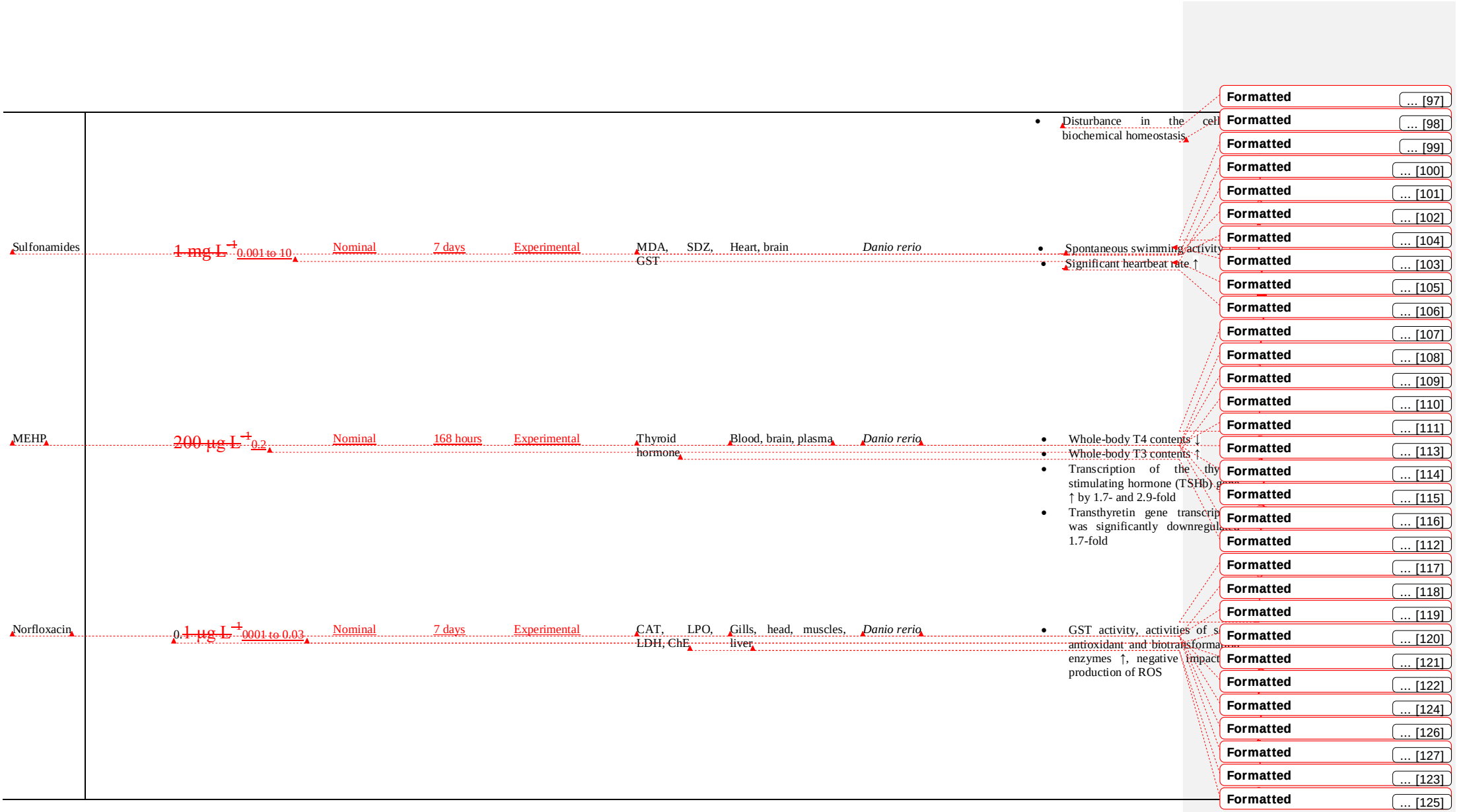
Sulfonamides
1 mg L−1
0.001 to 10
Nominal
7 days
Experimental
MDA, SDZ, GST
Heart, brain
Danio rerio
Disturbance in the cell biochemical homeostasis
Spontaneous swimming activity
Significant heartbeat rate ↑
MEHP
200 µg L−1
0.2
Nominal
168 hours
Experimental
Thyroid hormone
Blood, brain, plasma
Danio rerio
Whole-body T4 contents ↓
Whole-body T3 contents ↑
Transcription of the thy stimulating hormone (TSHb) g ↑ by 1.7- and 2.9-fold
Transthyretin gene transcrip was significantly downregul 1.7-fold
Norfloxacin
0.1 µg L−1
0001 to 0.03
Nominal
7 days
Experimental
CAT, LPO, LDH, ChE
Gills, head, muscles, liver
Danio rerio
GST activity, activities of s antioxidant and biotransforma enzymes ↑, negative impact production of ROS
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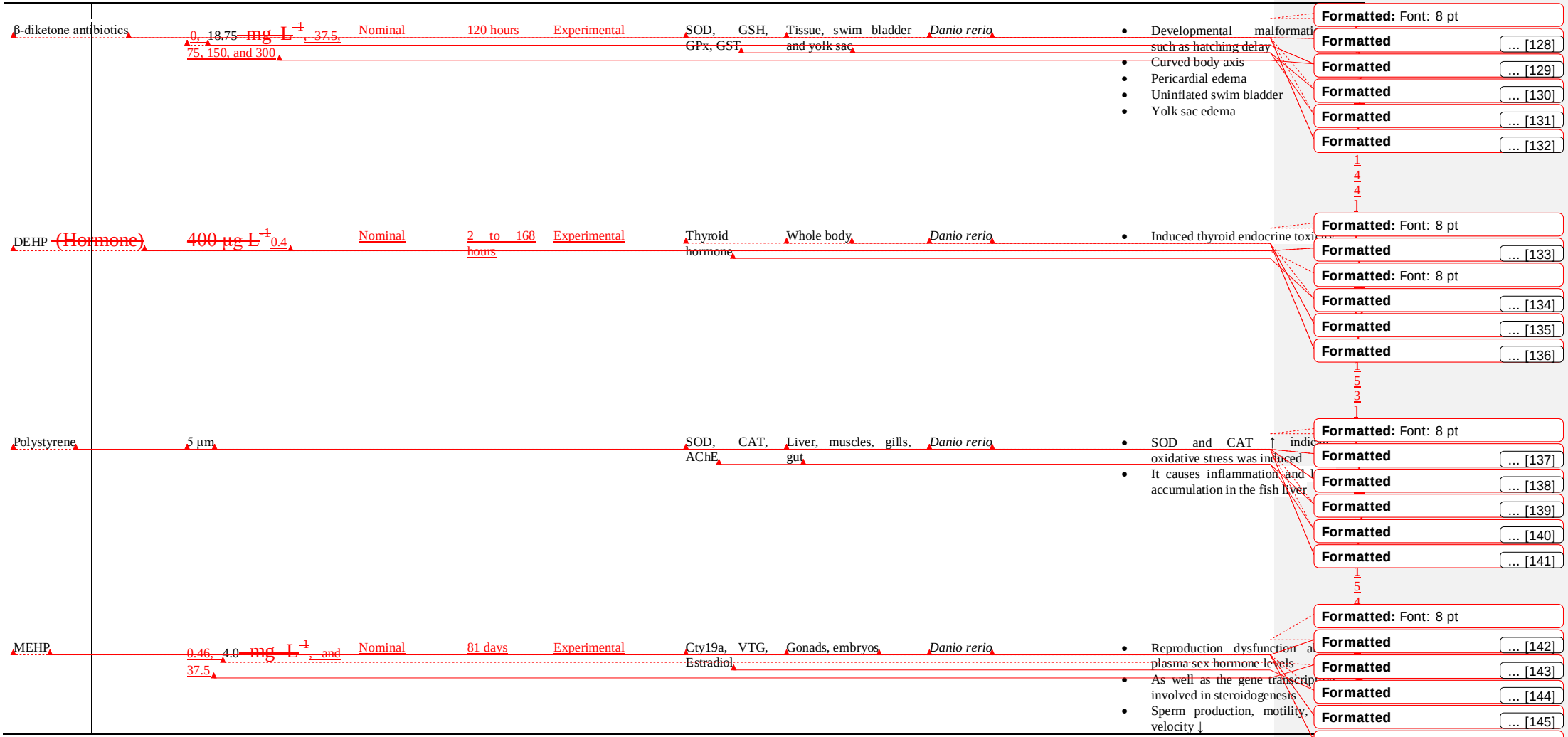
β-diketone antibiotics
0, 18.75 mg L-1, 37.5, 75, 150, and 300
Nominal
120 hours
Experimental
SOD, GSH, GPx, GST
Tissue, swim bladder and yolk sac
Danio rerio
Developmental malformati
such as hatching delay
Curved body axis
Pericardial edema
Uninflated swim bladder
Yolk sac edema
DEHP (Hormone)
400 µg L-1 0.4
Nominal
2 to 168 hours
Experimental
Thyroid hormone
Whole body
Danio rerio
Induced thyroid endocrine toxi
Polystyrene
5 µm
SOD, CAT, AChE
Liver, muscles, gills, gut
Danio rerio
SOD and CAT ↑ indic
oxidative stress was induced
It causes inflammation and
accumulation in the fish liver
MEHP
0.46, 4.0 mg L-1, and 37.5
Nominal
81 days
Experimental
Cty19a, VTG, Estradiol
Gonads, embryos
Danio rerio
Reproduction dysfunction a
plasma sex hormone levels
As well as the gene transcrip
involved in steroidogenesis
Sperm production, motility, velocity ↓
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TBBPA
0.1, 0.4 mg L-1, 0.7, and 1
Nominal
8 days
Experimental
SOD, CAT, GPx
Heart, brain, blood
Danio rerio
Egg diameter and eggshell ↓
Egg protein content ↓, worse hatching rates
Malformation rates ↑
Induces developmental toxicity, oxidative stress, and apoptosis in embryos
Growth rate ↓
Induce malformation
PROSPFOS
0.4 mg L-1, 0.8, and 1.6
Nominal
96 hours
Experimental
CAT, GST, MDA, GSH, GPx
Liver, thyroid, embryo
Danio rerio
Oxidative stress and apoptosis
PROSPFOS
200 µg L-1 0.2
Nominal
96 hours
Experimental
ROS, SOD, CAT, and AChE
Liver, intestine, gills, skin, brain
Danio rerio
Induced more reactive oxygen species
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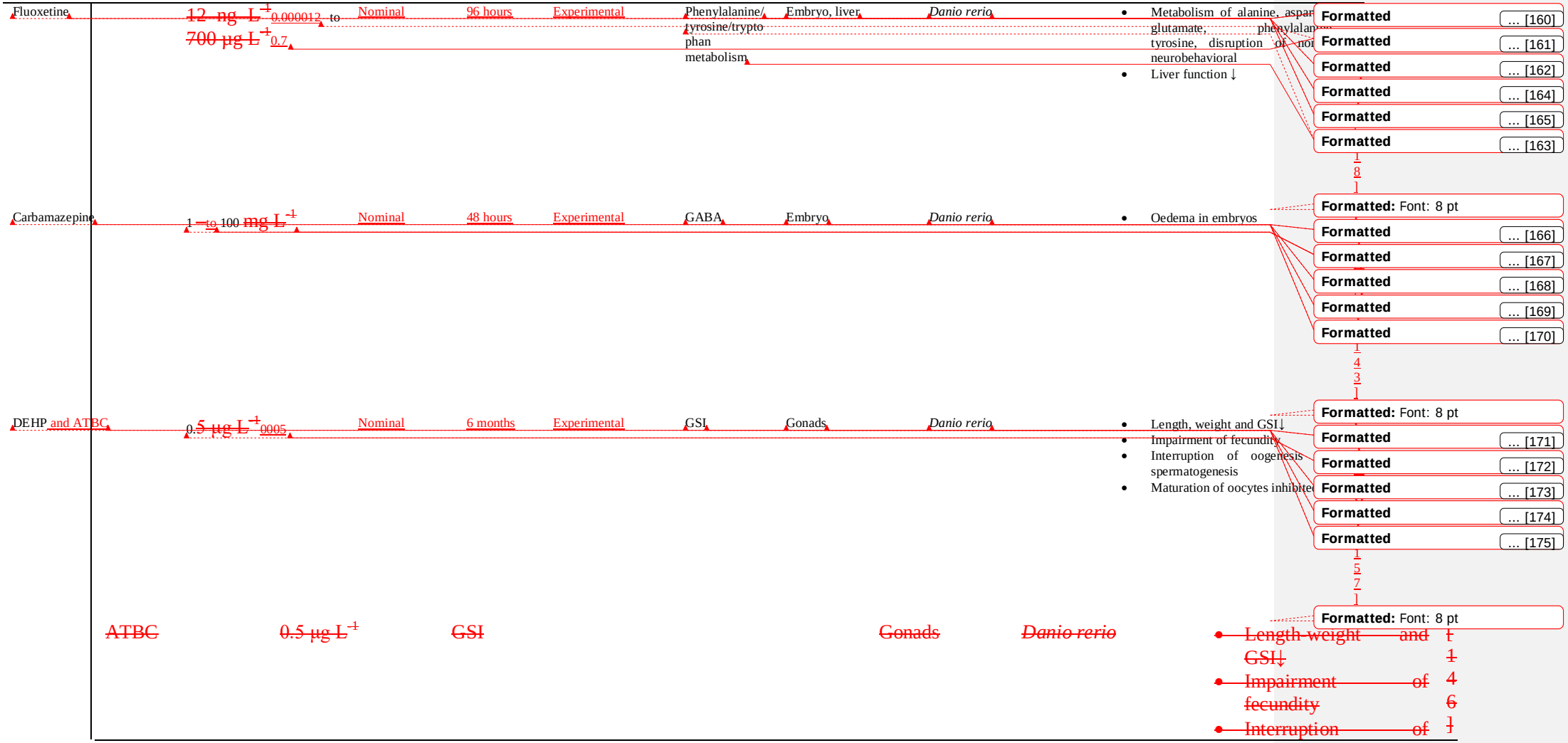
Fluoxetine
12 ng L-1 0.000012 to 700 µg L-1 0.7
Nominal
96 hours
Experimental
Phenylalanine/ tyrosine/tryptophan metabolism
Embryo, liver
Danio rerio
Metabolism of alanine, aspar glutamate, phenylalan tyrosine, disruption of nor neurobehavioral
Liver function ↓
Carbamazepine
1 to 100 mg L-1
Nominal
48 hours
Experimental
GABA
Embryo
Danio rerio
Oedema in embryos
DEHP and ATBC
0.5 µg L-1 0005
Nominal
6 months
Experimental
GSI
Gonads
Danio rerio
Length, weight and GSI↓
Impairment of fecundity
Interruption of oogenesis spermatogenesis
Maturation of oocytes inhibite
ATBC
0.5 µg L-1
GSI
Gonads
Danio rerio
Length weight and GSI↓
Impairment of fecundity
Interruption of
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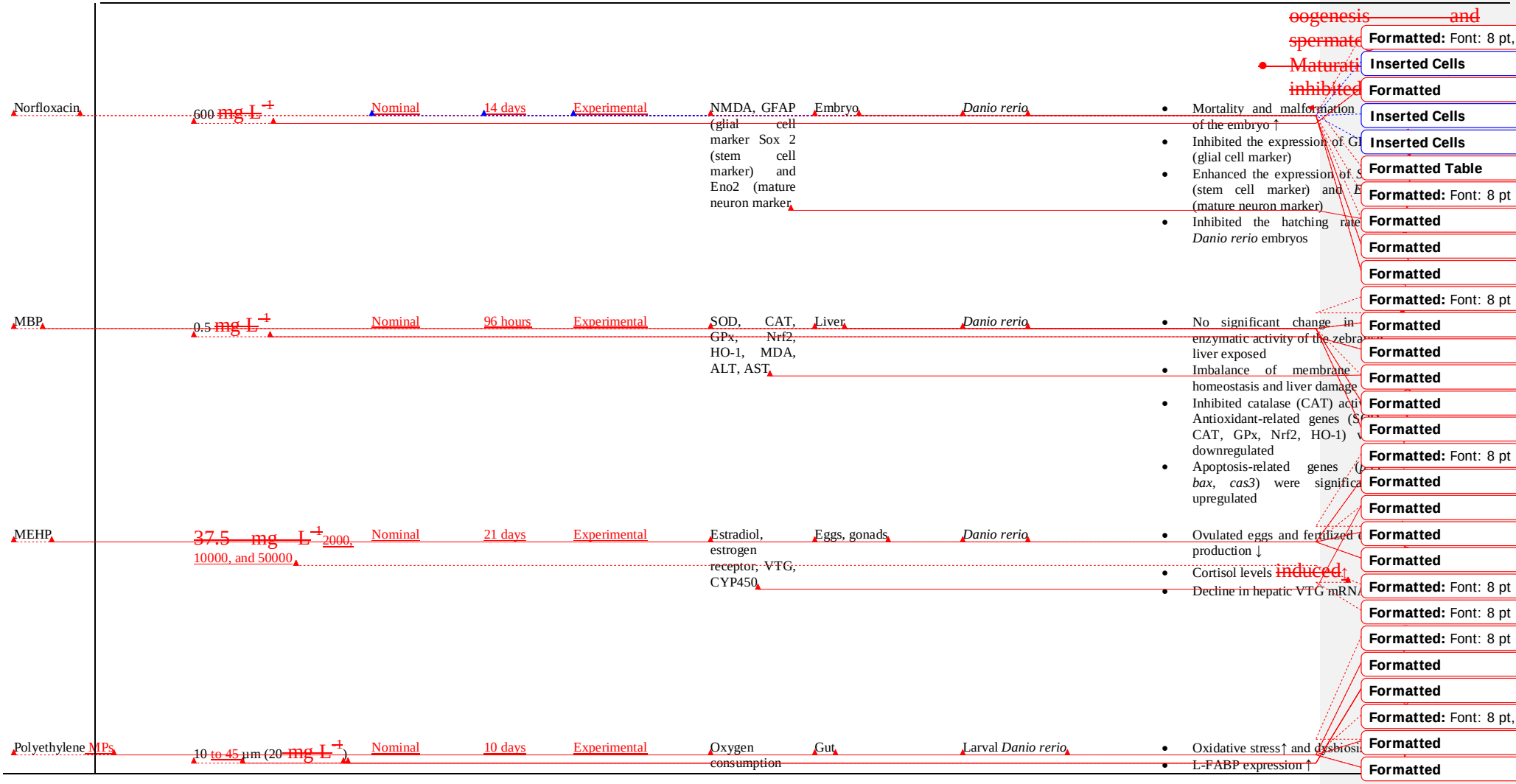
Norfloxacin
600 mg L-1
Nominal
14 days
Experimental
NMDA, GFAP (glial cell marker Sox 2 (stem cell marker) and Eno2 (mature neuron marker
Embryo
Danio rerio
Mortality and malformation of the embryo ↑
Inhibited the expression of GF (glial cell marker)
Enhanced the expression of S (stem cell marker) and E (mature neuron marker)
Inhibited the hatching rate Danio rerio embryos
MBP
0.5 mg L-1
Nominal
96 hours
Experimental
SOD, CAT, GPx, Nrf2, HO-1, MDA, ALT, AST
Liver
Danio rerio
No significant change in enzymatic activity of the zebra liver exposed
Imbalance of membrane homeostasis and liver damage
Inhibited catalase (CAT) acti
Antioxidant-related genes (SO CAT, GPx, Nrf2, HO-1) downregulated
Apoptosis-related genes (bax, cas3) were significa upregulated
MEHP
37.5 mg L-1 2000, 10000, and 50000
Nominal
21 days
Experimental
Estradiol, estrogen receptor, VTG, CYP450
Eggs, gonads
Danio rerio
Ovulated eggs and fertilized e production ↓
Cortisol levels induced ↑
Decline in hepatic VTG mRNA
Polyethylene MPs
10 to 45 µm (20 mg L-1)
Nominal
10 days
Experimental
Oxygen consumption
Gut
Larval Danio rerio
Oxidative stress↑ and dysbiosis
L-FABP expression ↑
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					rates (OCRs), SOD, ROS				
Polyethylene MPs	1000 μg L^{-1}, 1 to 4 μm (0.01 to 1)	Nominal	7 days	Experimental	Metabolism-related genes, LPL	Embryo	*Danio rerio*	• Transcriptional levels of gluc… and lipid metabolism-rel… genes ↓ • mRNA levels of phospholi… metabolism-related genes … changed	
AgNP, AgNPs	<10 μg L^{-1}, 0.001, 0.003, and 0.005	Nominal	96 hours	Experimental	CAT, AChE	Brain, muscles, gills, liver	*Danio rerio*	• Brain and mu… acetylcholinesterase activity ↓ • Liver and gill catalase activity	
AgNPs	3.31 mg L^{-1}, 6.63, 13.25, and 26.50	Nominal	28 days	Experimental	SOD, CAT, CYP1A and Hsp70	Blood, liver, kidney	Nile tilapia *Oreochromis niloticus*	• Expression of antioxidant ge… (SOD and CAT) was suppressed • Red blood cell, haemoglo… hematocrit, and white blood … values ↓ • Hematotoxicity, liver and kid… functions, growth, and surv… disruption	

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Polystyrene MPs	3 to 12 µm (1000, 25000, and 50000)	Nominal	21 days	Experimental	AChE, MDA	Heart	*Danio rerio*	• Heart frequency of ventricular contraction ↓ • Swimming velocity ↓ • Oxidative stress indices metabolic adjustments ↑
~~Gold nanomaterial~~ AuNPs	~~40~~ 10–100 nm (~~0.8 µg L^{-1}~~ 25, 0.5, 1 and 2)	Nominal	72 hours	Experimental	ROS, SOD	Heart, brain, foregut, swim bladder, GIT	*Danio rerio*	• No AuNP-related renal injury cellular oxidative stress • Prevent ROS production • Downregulation of *sod2* in muscle and gill
Sulfamethoxazole	3, 6, 12 ~~mg L^{-1}~~ and 24	Nominal	8 weeks	Experimental	SOD, CAT, GSH Px activity and MDA	Liver and gill, gut	*Danio rerio*	• Oxidative damage and immune system disorders were caused in the liver and gill • No apparent inhibitory effect on growth stress
Zinc oxide NPs	1 ~~mg L^{-1}~~ and 2	Nominal	15 days	Experimental	SOD, CAT, GR, GPx, GST mRNA, MDA, GSH	Muscles	*Oreochromis niloticus* ~~Nile tilapia~~	• Induce oxidative stress in muscle • Glutathione levels, superoxide dismutase, catalase ↓ • Glutathione, glutathione peroxidase, and glutathione transferase activities ↓

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Polyethylene MPs	2 mg L^{-1}	Nominal	96 hours	Experimental	VTG, CYP1a, CYP450	Liver, gills, intestine	Adult zebrafish	• Voids inside the intestine for [illegible] ↓ • The expression of *cyp1a* in intestine and *vtg1* in the [illegible] upregulated • Detoxification-related [illegible] (*cyp1a1*) and CYP450 induced	[16,51]
~~Polyethylene~~	~~38.26 µm~~	~~ZET~~				~~Embryo~~	~~Zebrafish~~	~~• Larval [illegible] after egg~~ ~~• Negative [illegible] development, growth, and metabolism~~	~~8~~ ~~1~~
~~Polyethylene~~	~~60 mg L^{-1}~~	~~ChE~~				~~Gills, liver and brain~~	~~Zebrafish~~	~~• Induced mutagenic and cytotoxic effects~~	~~[1,59,1]~~

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Arrow (↑)- Signifies increase; Arrow (↓)- Signifies decrease/reduction. AST - Aspartate aminotransferase ALT - Alanine aminotransferase; GST - Glutathione S-transferase; CAT - Catalase; EROD - Ethoxyresorufin-o-deethylase; SOD - Superoxide dismutase; GSH-Px - Glutathione peroxidases; AMS - Amylase; TPS - Tissue polypeptide-specific antigen; LPS - Lipid peroxidation; CYP450 - Cytochrome P450; VTG - Vitellogenin; AChE - Acetylcholinesterase; ACTH - Adrenocorticotropic hormone; CRH - Corticotropin-releasing hormone; POX - Peroxidase; LYZ - Lysozyme; GGT - Gamma- glutamyl transferase; G6PDH - Glucose-6-phosphate dehydrogenase; PVC - Polyvinyl chloride; BaP -

Bisphenol A; AgNPs - Silver nanoparticles; MDA – Malonidiadehyde; NPs – Nanoparticles/plastics; MPs – Microplastics; PFOS - Perfluorooctane sulfonate

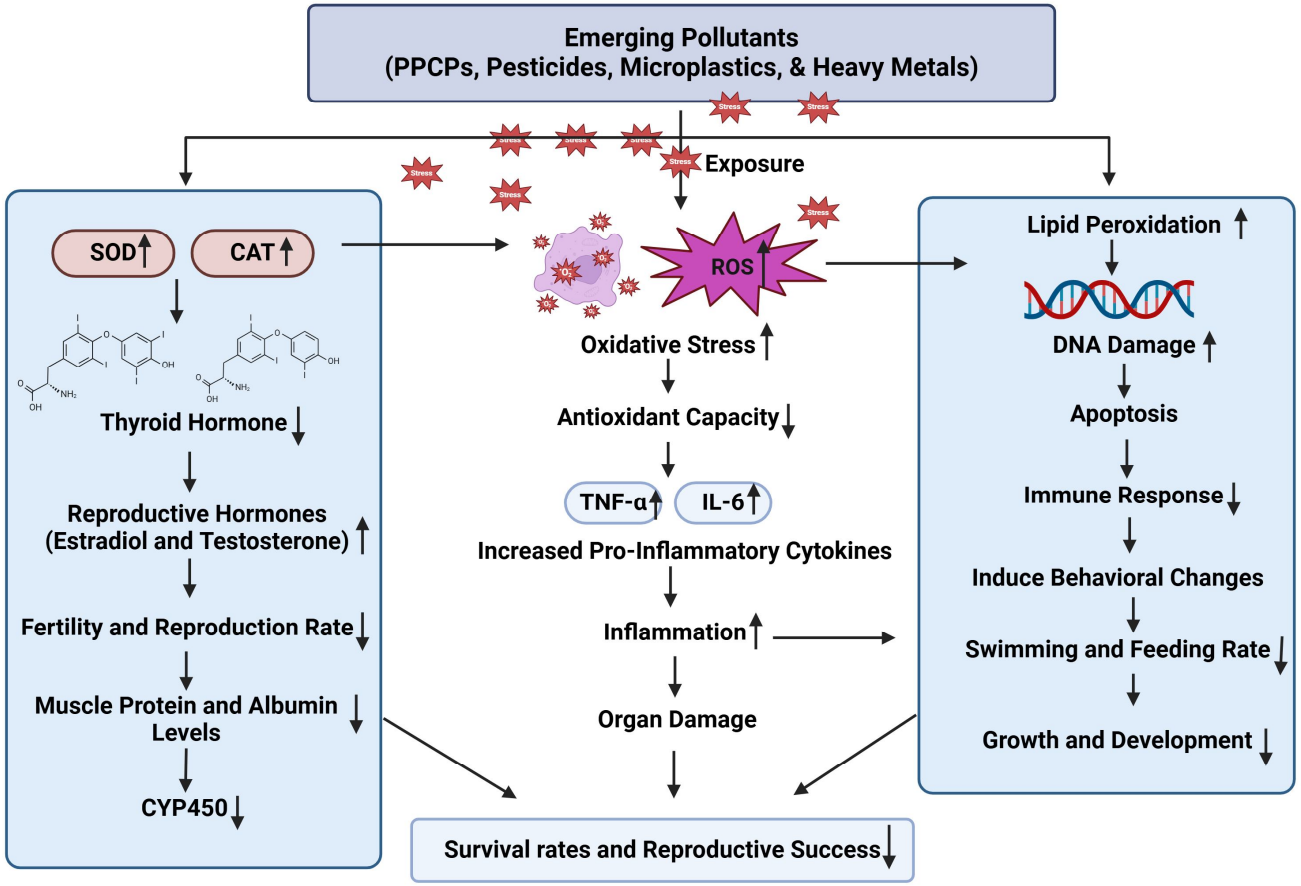

Fig. 1 ~~Toxicological Effects~~Mechanistic pathways of ~~Emerging Pollutants~~ toxicological effects of emerging pollutants on ~~Fishes. The~~ fishes. This figure ~~illustrates~~summarizes the ~~complex mechanisms by~~key mechanistic pathways through which emerging pollutants ~~impact different aquatic fishes. Elevated oxidative enzyme activity,~~ such as microplastics, pesticides, pharmaceuticals, nanoparticles, and endocrine disruptors, affect fish physiology and ecology. These pollutants trigger oxidative stress (*via* elevated superoxide dismutase (SOD~~) and~~), catalase (CAT), and glutathione peroxidase (GPx) activity), hormonal ~~disruptions resulted in decreased thyroid hormones~~imbalances (reduced triiodothyronine (T3~~ and~~), thyroxine (T4~~) and reproductive hormones levels~~ (), estradiol (E2), and testosterone), ~~which lead to increased oxidative stress and inflammatory responses such as~~ and immune modulation (increased tumor necrosis factor-alpha (TNF-α) and ~~interleukins~~interleukin-6 (IL-6~~),~~) expression). The resulting ~~in~~ cellular damage~~, including~~ manifests as lipid peroxidation~~ and DNA damage. Physiological changes manifest as decreased immune response and altered behaviors, results in reduced swimming and feeding rate~~, DNA fragmentation, and histopathological lesions in liver, gill, and kidney tissues. These biochemical and physiological disturbances lead to behavioural impairments and reduced growth and reproductive output, ultimately ~~affecting growth and development,~~ contributing to population-level declines~~ and reduced reproductive success, highlighting the interconnectedness of biochemical, physiological, and ecological responses to environmental stressors~~.

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4.2 Impacts on amphibians

Exposure to pesticides and fertilizers significantly affects amphibians across all life stages, including adults, embryos, and larvae, leading to deformities, developmental delays, increased mortality rates, and altered feeding behaviours [160] (Fig. 2). These contaminants can also increase disease susceptibility, disrupt physiological processes, inhibit cholinesterase production [24] (Fig. 2), reduce weight, and, in severe cases, result in death [161]. Many studies have shown that exposure to a mixture of pesticides, such as atrazine, 2,4-D, and metolachlor, alters neurotransmitter functions and increases AChE activity in *Lithobates catesbeianus* while decreasing plasma AChE in *Hoplobatrachus tigrina* [29]. However, exposure of *Xenopus laevis* to DDE and vinclozolin impairs vocalization, reducing their calling ability and increasing their rasping behavior [162]. Herbicides, such as glyphosate, have genotoxic effects on amphibians, damaging their genetic material and leading to micronuclei formation as well as erythrocyte abnormalities [163]. This genotoxicity, which is evident in species such as *Physalaemus gracilis* and *P. cuvieri*, serves as an indicator of DNA damage and chromosomal instability, potentially causing both structural and numerical chromosomal mutations [164]. Pesticide exposure can strongly disrupt amphibian health, affecting population dynamics by increasing predation risk, reducing reproductive success, altering metabolism and behaviour, causing gonadal abnormalities, and increasing larval mortality [165]. Exposure to pharmaceutical pollutants such as ibuprofen and fluoxetine similarly impacts amphibians. Exposure to these substances in *Oreochromis niloticus* has been shown to reduce body length and weight [166], whereas in the case of bullfrogs, exposure to these substances seems to decrease their feeding rates and increase serotonin levels, affecting behaviour controlled by brain-regulated corticotropin-releasing

factors [167]. Similarly, exposure to antibiotics, such as enrofloxacin and ciprofloxacin, results in growth disruption, the induction of antioxidant activity and neurotoxicity in *Rhinella* arenas, which further leads to impaired swimming and feeding behaviours [168].

Many studies have also demonstrated varying impacts of exposure to microplastics on amphibians, depending on their shape, size, density, and chemical composition. Exposure to such plasticizers results in impaired growth, pigmentation changes, neurotoxicity, and altered immune responses [169]. In the case of *Xenopus laevis*, exposure to microplastics results in increased malformations, reduced feeding, and elevated mortality rates [170], whereas in *X. tropicalis*, pigmentation increases, and body size decreases. Polyethene microplastics alter erythrocyte morphology; liver function; social and antipredator behaviours; and the activities of enzymes such as SOD, CAT, MDA, and AChE while increasing the oxygen demand and impacting feeding habits due to a decrease in the buccal area [171]. Microplastics also reduce the erythrocyte count, haemoglobin level, white blood cell count and platelet count in *Oreochromis niloticus* [172]. Silver and gold nanoparticles have been demonstrated to cause gut toxicity, damage microvilli, and reduce energy metabolism in a study involving *Xenopus* embryos, impacting heart and craniofacial development by altering the expression of genes such as *fgf8*, *bmp4*, and *sox9* [173]. This exposure increases ROS production and cortisol levels (Fig. 2), affecting pigmentation, eye size, and overall organism development [173]. Additionally, endocrine-disrupting compounds, such as BPA, BDE-47, PBDE, PFOS, and PFOA, adversely impact amphibian development, altering liver function, movement, predator response, and ingestion behavior [169]. For example, studies involving PFOS and PFOA exposure in northern leopard frogs revealed impaired neurotransmitter functions; decreased dopamine; and altered serotonin, norepinephrine, GABA, and glutamate levels [174].

~~However, in black-spotted frogs, PFOS exposure disrupted cholesterol metabolism, influencing lipid metabolism and immune function, which led to increased toxicological effects [175] (Table 2).~~

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In amphibians, exposure induces multiple adverse effects, including oxidative stress, genotoxicity, metabolic impairment, hormonal dysregulation, and immune disruption. Within the immune system, altered gene expression triggers endoplasmic reticulum (ER) stress and oxidative DNA damage, which interfere with key metabolic pathways. These disruptions lead to hormonal imbalances characterized by decreased levels of dopamine, hepatic growth hormone (HGH), luteinizing hormone (LH), and thyroid hormone (TH). Collectively, these molecular and endocrine disturbances compromise overall amphibian health, immune competence, and survival (Fig. 2).

800 **Table 2** Summary of the toxicological effects of emerging pollutants on amphibians

Compound	Size/ Concentration (mg kg^{-1})	Nominal vs Measured	Exposure duration	Exposure type (Experimental/ field)	Biomarker	Tissue	Species name	Effects	Referen
Glyphosate-based herbicide	2.9 mg L^{-1}	Nominal	21 days	Experimental	mRNA expressions	Brain, tail	Tadpoles- *Lithobates sylvaticus*	• Effects on the metamorphosis by decreasing their development	[176]
DEHP	0.1 mg L^{-1}	Nominal	6 months	Experimental	17β-estradiol liver vitellogenin, gonad histology	Plasma	Marine medaka	• Egg production of exposed females ↓ • Testosterone levels ↓ • Fertilization rate of oocytes ↓	[177]
Nitrate	100 mg L^{-1}	Nominal	24 hours	Experimental	Thyroid hormone	Whole body at larval stage	*Bufo gargarizans*	• Delayed metamorphosis • Reduced circulating T4 and T3 levels • Downregulation of *Dio2*, upregulation of *Dio3* mRNA, and partial colloid depletion in thyroid gland follicles • Total body length, tail length and hid-limb length ↑ • Mortality ↑ and significant metamorphic retardation	[178]
Polystyrene	50 nm	Nominal	-	Experimental	mRNA, gene expression change	Digestive gut cells, cytoplasm	*Xenopus laevis*	• Mortality rate ↑ • Expressions of the mesoderm markers, bra (T-box Brachyury gene), *myod1* (myogenic differentiation1), and of neural crest marker *sox9* sex SRY determining region Y-box 9 transcription factor *sox9*) are modified	[179]
Zn nanoparticles	50 μg L^{-1}; 100 μg L^{-1} 0.05, 0.1	Nominal	14 days	Experimental	Thyroid activity, vitellogenesis, cytochrome P450	Blood, serum, brain, liver	Marsh frog- *Pelophylax Ridibundus*	• Hepatic EROD activity↓ • Decrease in serum cortisol and TSH concentrations • Capacity for organic xenobiotic detoxification ↓	[180]
Dimethyl phthalate (DMP) and dicyclohexylphthalate (DCHP)	0.03–924.0 mg L^{-1}	Nominal	72 hours	Experimental	mRNA expression	Embryo and tadpole	Frog- *S. tropicalis*	• Malformations, such as edema • Improperly developed heart and tail abnormalities • Improperly coiled guts • Absent gills • Upregulated cellular stress-related messenger-RNA	[181]
Azocyclotin	0.1 μg L^{-1} 0001	Nominal	21 days	Experimental	Thyroid hormone receptor, ST3, MMP2, BTEB	Whole body tadpoles	*Xenopus laevis*	• Downregulated the expression of *trβ*, *dio2*, basic transcription element binding protein (bteb), matrix metalloproteinase 2, and stromelysin-3	[182]
Copper	32 μg L^{-1} 0.032	Nominal	64 days	Experimental	Thyroid hormone receptors, mRNA levels	Whole body	*Bufo gargarizans*	• Induced an upregulation of Dio3 expression but downregulated trα and trβ • Mortality ↑ • Inhibited growth • Downregulated mRNA expression of TR α and TR β mRNA level	[183]
BDE -47	50 μg g^{-1}	Nominal	21 days	Experimental	Thyroid hormone	Brain, tail	*Xenopus laevis*	• Metamorphic rate↓	[184]

PFOA	0.3 mg L^{-1}	Nominal	35 weeks	Experimental	VTG	Liver, gonads	*Oryzias latipes*	• Male myo-inositol (m-Ino) ↓ • Female trimethylamine N-oxide ↑ • Increase of male dimethylamine (DMA) • Alterations to fecundity, hatching rate, histological change • VTG expression, metabolism disturbances, glucose and oxaloacetate expression increased.
A-cypermethrin	0.5 µg/L0005	Nominal	14 days	Experimental	SOD, CAT, MDA, GST,	Tadpole	*Rana nigromaculata*	• Increased tadpole mortality • Growth of tadpoles↓ • Induce oxidative stress
Triclosan	500 ng L^{-1} 0.0005	Nominal	14 days	Experimental	MT, CYP450, GSH	Brain, liver, blood plasma	Frog	• Endocrine disruption caused oxyradicals and glutathione (GSH and GSSG) levels and lysosomal membrane instability
Fipronil	35 µg kg^{-1} 0.035	Nominal	7 days	Experimental	CAT, GST, MDA, G6PDH	Tadpole	Frog- *Eupemphix nattereri*	• Cause oxidative stress • Decrease in CAT activity • Increase in lipid peroxidation levels
Estrone	100 ng L^{-1} 0.0001	Nominal	14 days	Experimental	MT, CYP450, GSH	Brain, liver, blood plasma	Frog	• Decrease of deiodinase activity in the liver • Decreased lactate dehydrogenase activity • Increased level of pyruvate in the liver • Activation of CYP450 and caspase-3 and efflux of cathepsin D from lysosomes
Zinc oxide NPs	3.1 µM	Nominal	14 days	Experimental	SOD, MT, CYP450	Liver, serum, brain	Frog	• Endoplasmic reticulum stress, oxidative stress caused • Increase in the total and oxidized glutathione concentrations • Increase in DNA fragmentation
CPX and ENR	10 µg L^{-1} 0.01	Nominal	96 hours	Experimental	GST, CAT,	Eggs, embryo	*Rhinella arenarum*	• Affected larval development, size, shape and growth alter enzyme activities related to oxidative stress
BPA	0.05 mM	Nominal	96 hours	Experimental	Estrogen-receptor antagonist	Plasma membrane and nucleus	Frog	• Inhibits nerve conduction independent of estrogen-receptor activation
Cyproconazole	13.462 mg L^{-1}	Nominal	28 days	Experimental	SOD, CAT, GSH, GST, MDA, LDH	Liver, gut, tail, brain, stomach, and heart of a tadpole	*Rana nigromaculata*	• Lactic dehydrogenase (LDH) and oxidative stress induced • Bioaccumulation, tissue distribution ↑
Atrazine	0.5 µg L^{-1} 0005	Nominal	28 days	Experimental	VTG, estrogen	Reproductive organs	Northern leopard frogs, fathead minnows	• Testicular size↓, down regulations of genes involved in gonadal steroidogenesis and luteinizing hormone signalling reduced egg production
4-MBC	0.013 mg L^{-1}	Nominal	144 hours	Experimental	CAT, LPO, LDH, ChE, Vtg	Embryo	*Pelophylax perezi*	• It affects GST activity and delays hatching time. • Reduced survival.
Chlorpyrifos	0.01 - 0.7 mg L^{-1}	Nominal	336 hours	Experimental	Sound production	Tadpole	*Ceratophrys ornata*	• Mortality, behaviour, abnormalities, growth inhibition, swimming alterations, and abnormalities increase • The duration and number of pulses of sounds ↓
BPF	100 nM	Nominal	24 hours	Experimental	Morphologic	Muscles,	*Xenopus laevis*	• TH signalling and postembryonic development ↓

						al biomarkers (BW, SVL, HLL, HA and BL), TH	brain		• Inhibited T3-induced transcription TH-response genes	
Cadmium		50 µg L^{-1} 0.05	Nominal	40 days	Experimental	T3, T4	Bone, tadpole	*Bufo gargarizans*	• Retarded metamorphosis, follicular cell hyperplasia of the thyroid • Down regulated mRNA expression *dio2* and *trβ* transcripts • mRNA expression of TRα up regulated • Bone formation delayed • Body size ↓	[195]
DEHP		10 µM	Nominal	72 hours	Experimental	Testosterone, mRNA, HSP50,	Gonads	Japanese medaka-*Silurana tropicalis*	• Mortality rate↑ • Expression of androgen-related genes ↑ • Malformations in eyes (reduction in size, asymmetric formation • Incomplete separation from the brain and cyclops), tails (shortening and flexure), hearts (failure to coil in an 'S' shape), guts (failure to coil), gills (shredded appearance), and head and face (reduction in size and unusual shape)	[196]
DBP		200 µM	Nominal	72 hours	Experimental	Gene stability,	Embryo	*Xenopus laevis*	• Head, eye and mouth irregularities. • Induced cell cleavage irregularities. • Slowed cytokinesis • Fewer numbers of cells per embryo, cellular dissociation, neural abnormalities • Embryo survival ↓	[197]
Atrazine		5 µg L^{-1} 0.005	Nominal	25 days	Experimental	Total sperm count, estradiol	Gonads	*Oryzias latipes*	• Effect reproduction, fertility and expression of genes related to steroidogenesis • DNA methylation in gonads ↑ • Males: Fertilization rate, sperm count and total motile sperm ↓ • Expression of star and fish genes in testis ↑ • Females: Expression of the methyltransferase *dnmt1* gene in ovaries ↓	[198]
PFOA		1000 ppb_1	Nominal	30 days	Experimental	GABA, Dopamine	Brain	*Lithobates pipiens*	• Dopamine significantly ↓	[199]
17 β-estradiol		0.1 µg L^{-1} 0001	Nominal	21 days	Experimental	GHR mRNA, VTG, estrogen receptor	Yolk sac, adult	*Mozambique tilapia*	• Hepatosomatic index ↓ • Induce hepatic growth hormone receptor (*ghr*) mRNA expression • Erα levels ↑	[200]
Estradiol (cyproterone acetate)		0.5 mg L^{-1}	Nominal	40 hours	Experimental	Mate recognition protein (MRP), steroid receptor	Neonates	*Brachionus calyciflorus*	• Positive impact of CPA on sexual reproduction • Regulatory hormonal pathways of mixis induction and sexual reproduction (e.g. fertilization, resting egg production) are different	[201]
Organotin		1 µg L^{-1} 0.001	Nominal	21 days	Experimental	T3, RNA expression	Tadpoles	*Xenopus laevis*	• Induce TH disruption by decreasing T3 level • Inhibitory effects on metamorphic development of tadpoles • TH responsive genes *trβ*, *bteb* and *dio2* were down regulated • While *tshβ* and *slc5a5* were up regulated	[202]
Cyproconazole		10 mg L^{-1}	Nominal	90 days	Experimental	CAT, SOD, GSH	Muscles	*Rana nigromaculatus*	• Affected the rate of metamorphosis • Significantly altered transcriptional regulation	[203]
DEHP		0.1 µg L^{-1}	Nominal	21 days	Experimental	Vitellogenic oocytes	Ovaries	Japanese medaka	• Mortality ↑ • Decline in mature sperm count	[204]

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	0001							• Interstitial space ↑	
BPAF	100 nM	Nominal	8 weeks	Experimental	Estradiol	Gonads	*Xenopus laevis*	• Inhibit testicular differentiation and subsequent development • Alterations in hormone levels • Sperm quantity and quality ↓	[205]
Polyethylene	60 mg L^{-1}	Nominal	7 days	Experimental	Oxidative stress reactions	Gills, GIT, liver, muscle tissues of the tail and Blood	*Physalaemus cuvieri*	• Frequency of erythrocyte nuclear abnormalities↑ • Different histopathological changes (blood vessel dilation, infiltration, congestion, hydropic degeneration, hypertrophy and hyperplasia	[159]
Zinc oxide	10 mg L^{-1}	Nominal	7 days	Experimental	DNA stability	Blood	*Lithobates catesbeianus*	• Genotoxic and mutagenic effect, DNA damage	[206]
Triclosan	10 µm	Nominal	-	Experimental	Respiratory rate	Liver, blood, cytoplasm	Marsh frog	• Liver mitochondria function ↓ • Rate of oxygen consumption ↓ • Enhanced the production of hydrogen peroxide by mitochondria • Induce permeabilization of mitochondrial membranes and cytoplasmic membrane of the erythrocytes	[207]
Chlorpyrifos	10 µg L^{-1} 0.01	Nominal	24 hours	Experimental	Cholinesterase, Acetylcholine	Tadpole	*Proechimys cuvieri* and *Leptodactylus latrans*	• Swimming ability ↓ may indirectly lead to death	[208]
Lambda-Cyhalothrin	8 ppb0.008	Nominal	5 weeks	Experimental	Complete blood count (CBC), and liver function test (LFT) and reproductive hormones	Testis, ovaries, blood	*Rana tigrina*	• Fertility ↓ • Kidney, liver, and intestine function is inhibited • Breaking of cyst walls, mutilation • Deformation in the epididymis, high disruption in seminiferous tubules • Degeneration of cells and oocytes, discolouration • Edema in the stroma in male gonads • Female gonads (ovaries) showed mild changes, a contraction in follicles, and deformation • Disruption of granulosa cells and oocytes	[209]
BPA	100 nM	Nominal	4 weeks	Experimental	T3-induced TH-response gene expression	Brain	*Xenopus laevis*	• Affected TH-dependent brain development • Body size ↓ • Head shrinkage, mouth sharpening, brain remodelling, and hind limb growth	[210]
Carbaryl and dimethoate	2.9 – 4.8 mg L^{-1}	-	-	-	AChE, CbE, CAT, GPx, SOD	Tadpoles, Adult	*Xenopus laevis*, *Oreochromis mossambicus*	• Caused oxidative stress in the aquatic organisms • Inhibition of AChE and CbE affected the standard transmission of nerve impulses	[211]
Actellic 50 EC	0.1 mg L^{-1}	Nominal	5 days	Field	c	Liver, muscles	*Pelophylax ridibundus*	• 50% ↓ in the survival rate • Body volume and length ↓ • Respiratory rate and oxygen consumption ↓ • Number of erythrocytes and leukocytes ↑ • Blood glucose levels ↑	[212]

BPA		~~100 µmol L^{-1}~~ 0.0001	Nominal	60 days	Experimental	SOD, CAT, ROS	Blood	*Xenopus tropicalis*	• Reactive oxygen species ROS level ↑ • DNA damage in embryos	[102]
BPA		10 µM - 20 µM	Nominal	5 days	Experimental	Oestrogen-like and anti-androgenic activity	Early pregnancy	*Xenopus laevis*	• Swimming performances↓ • Induces apoptosis via oxidative stress	[213]
Polyethylene MPs		60	Nominal	7 days	Experimental	ChE	Gills, liver and brain	*Physalaemus cuvieri*	• Induced mutagenic and cytotoxic effects	[213]

Arrow ~~(↑)~~ (↑) - Signifies increase; Arrow ~~(↓)~~ (↓) - Signifies decrease/reduction. SOD - Superoxide dismutase; CAT - Catalase; MDA - Malondialdehyde; GST - Glutathione S-transferase; CYP450 - Cytochrome P450; GSH - Glutathione; G6PDH - Glucose-6-phosphate dehydrogenase; GPx - Glutathione peroxidase; VTG - Vitellogenin; TH - Thyroid hormone; HA - Hemagglitinin; GHR - Growth hormone receptor; GIT - Gastrointestinal tract; ROS - Reactive oxygen species; GABA - Gamma-aminobutyric acid; GR - Growth hormone; T3 - Triiodothyronine; T4 - Thyroxine; MT - Metallothione; ChE - Cholangiinesterase; TNF - Tumor necrosis factor; MMP2 - Matrix metalloproteinase; BDE - Brominated diphenyl ethers; 4-MBC - 4-methylbenzylidene; BPF - Bisphenol F; DEHP - Di-2-ethylhexyl phthalate; DBP - Dibutyl phthalate; PFOA - Perfluorooctanoic acid; CPX – Ciprofloxacin; MPs – Microplastics; LFT - Liver function test; CBC - Complete blood count.

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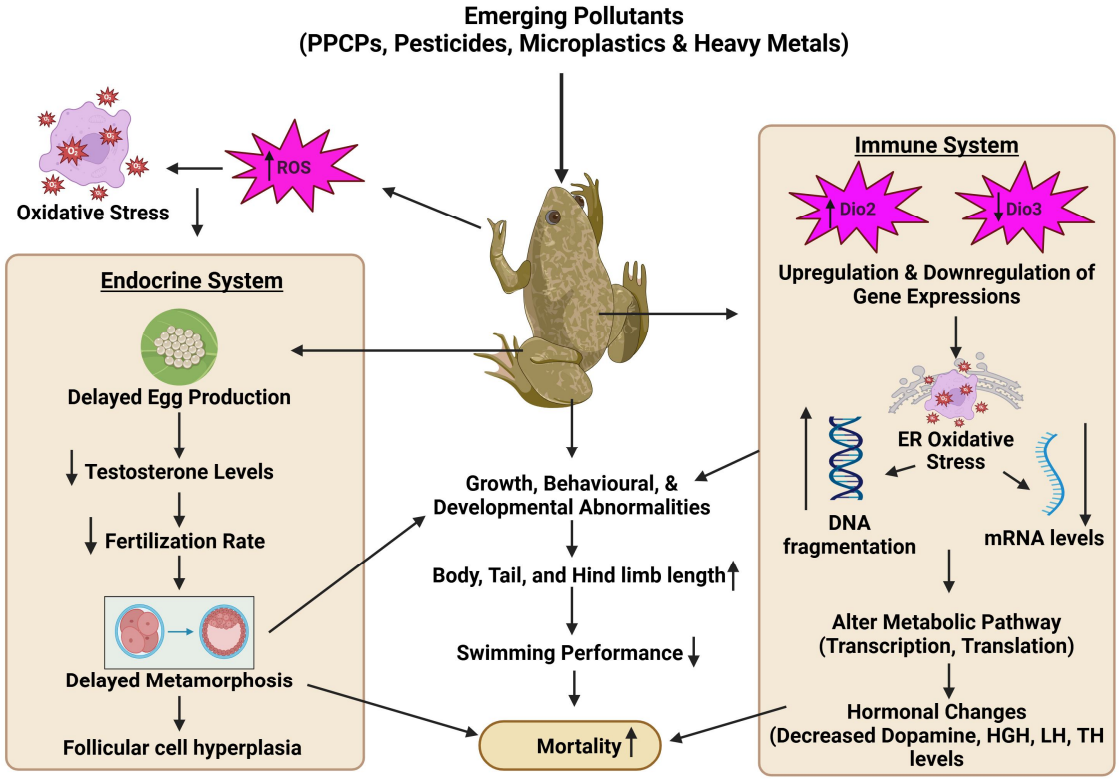

Fig. 2 ~~Toxicological effects~~Schematic diagram showing the mechanistic pathways underlying the toxicological impacts of emerging pollutants on ~~Amphibians. The figure illustrates the mechanism by which exposure to emerging pollutants affects~~ amphibians. The exposure to such

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pollutants ~~causes increased~~induces oxidative stress, disrupting the endocrine system, resulting in delaying egg production, reducing testosterone, and slowing metamorphosis, which further leads to growth and developmental abnormalities like increased body and limb length and decreased swimming performance, increasing mortality risk. In the immune system, gene expression changes cause endoplasmic reticulum (ER) oxidative stress and DNA damage, altering metabolic pathways, leading to hormonal imbalances, such as reduced dopamine, hepatic growth hormone (HGH), luteinizing hormone (LH), and thyroid hormone (TH) levels, which collectively harm amphibian health and survival.

4.3 Impacts on molluscs

Molluscs such as clams, oysters, mussels, and scallops are filter feeders, processing up to 90% of seawater particles [214]. This behaviour makes them highly susceptible to contaminants directly consumed from their environment [215]. Molluscs attach to solid surfaces such as rocks and wood (e.g., mussels and oysters) or burrow into sediments (e.g., clams). Owing to these habits, they often serve as bioindicators, as contaminant accumulation in their tissues reflects environmental conditions [216]. For example, the filtration rate of *Dreissena polymorpha* is temperature sensitive and varies with size, as more giant mussels filter more slowly than smaller mussels do [217]. Other molluscs, such as *Corbicula fluminea*, undergo strong filtration through suspension feeding and bioaccumulation. Bioaccumulation depends on environmental factors and varies across different body parts in molluscs, helping researchers assess specific pollutants [87]. Molluscs are sensitive to microplastics, and exposure to microplastics can reduce sperm velocity and oocyte size and lead to decreased fertilization rates, shell malformations, and increased ROS production [218] (Fig. 3). In the case of *Mytilus edulis*, exposure to particular microplastics, such as PLA and HDPE, results in reduced filtration ability and changes in hemolymph composition and can also trigger inflammatory responses [219], whereas polystyrene exposure can increase respiration and reduce filtration activity, highlighting the potential impact of microplastics on energy distribution [220]. Additionally, exposure to these plastic particles has led to genotoxic effects, including DNA damage and oxidative stress (Fig. 3), with altered antioxidant enzyme levels, as recorded in a study involving *M. galloprovincialis* [221].

Heavy metals also impact molluscs. Cadmium, for example, affects hemocytes in *M. edulis*, altering gene expression related to immunity and cell viability [222], whereas in the clam *R. philippinarum*, exposure to such heavy metals results in altered total hemocyte counts and immune stress and can lead to the production of oxyradicals, thereby disturbing

~~energy metabolism [223]. However, arsenic exposure can induce oxidative stress in the digestive glands and destabilize lysosomes in *R. philippinarum*, disrupting metabolic processes and gill structure [223]. When exposed to titanium dioxide, *M. coruscus* shows impaired hemolysis, reduced lysosomal content, and lower growth rates [224]. Similarly, cadmium and arsenic exposure in *M. galloprovincialis* and *Pinctada martensi*tic further disrupts reproductive functions and energy metabolism, compromising immune responses and altering nucleic acid metabolism [225]. Pharmaceuticals, personal care products, and pesticides can also harm molluscs, even when they are exposed for shorter periods of time, which can affect their metabolism, growth and development (Table 3). For example, caffeine exposure can damage the digestive glands, gills, and hemolymph of molluscs [226]. Exposure to pesticides such as atrazine, indaziflam, bifenthrin, and hexazinone can trigger oxidative stress in clams, reducing AChE activity [227] and negatively impacting growth and development by disrupting gills, digestive system formation, and the digestive system [228].~~

Molluscs such as clams, oysters, mussels, and scallops are efficient filter feeders, processing up to 90 % of suspended particles in seawater [223]. This behaviour makes them highly susceptible to contaminants ingested directly from their surroundings [224]. Most species either attach to solid substrates (e.g., mussels, oysters) or burrow into sediments (e.g., clams), leading to differential contaminant exposure and accumulation. Consequently, molluscs serve as reliable bioindicators, as pollutant concentrations in their tissues often reflect ambient environmental levels [225]. For example, the filtration rate of *Dreissena polymorpha* varied with temperature and body size, with larger mussels exhibiting slower filtration than smaller ones [226]. Likewise, *Corbicula fluminea* showed strong suspension feeding and bioaccumulation tendencies, where pollutant partitioning across tissues helps identify site-specific contamination [97].

MPs and NPs are among the most studied emerging pollutants affecting molluscs. Exposure to MPs have shown to reduce sperm velocity, oocyte size, and fertilization success while inducing shell malformations and ROS production [227]. In *Mytilus edulis*, exposure to specific polymers such as polylactic acid and high-density polyethylene results in reduced filtration capacity, altered hemolymph composition, and activation of inflammatory responses [228]. Polystyrene MPs and NPs have shown to increase respiration and reduce filtration activity, redistributing energy away from growth and reproduction [229,230]. Moreover, chronic MP exposure led to genotoxicity, including DNA strand breaks, oxidative stress, and modulation of antioxidant enzymes such as SOD and CAT, as reported in *M. galloprovincialis* [231,232].

Heavy metals such as cadmium and arsenic also exert toxic effects in molluscs. Cadmium exposure have shown to alter hemocyte viability and immune-related gene expression in *Mytilus edulis* [233]. While *Ruditapes philippinarum* exhibited disturbed hemocyte counts, oxidative stress, and impaired energy metabolism under arsenic exposure [234]. Arsenic also lead to induced oxidative stress in the digestive gland and destabilizes lysosomal membranes, compromising metabolic integrity [234]. Exposure to titanium dioxide NPs resulted in hemolysis inhibition, reduced lysosomal content, and growth impairment in *Mytilus coruscus* [235]. Similarly, studies related to cadmium and arsenic exposures in *M. galloprovincialis* and *Pinctada martensii* have demonstrated disruption in reproductive function and nucleic acid metabolism, leading to immune suppression [236].

Exposure to PPCPs compounds such as caffeine, diclofenac, and ibuprofen have resulted in impaired gill and digestive gland function, altered antioxidant enzyme activities, and reduced cellular viability [237–239]. For example, ibuprofen exposure decreased glutathione reductase activity and increased oxidative imbalance in *Crassostrea gigas* [238]. Endocrine-active substances such as drospirenone and estradiol modified glutathione-S-

transferase (GST) and GPx activities in *Lampsilis siliquoidea* and *Ruditapes philippinarum*, respectively, leading to oxidative stress and altered metabolism [240,241]. Pesticides such as atrazine, indaziflam, bifenthrin, and hexazinone trigger AChE inhibition and oxidative stress in clams and gastropods, disrupting gill structure and digestive development [242,243].

Overall, molluscs display a range of sublethal and lethal effects, including oxidative stress, genotoxicity, metabolic impairment, neurotoxicity, and immune disruption across diverse taxa (Table 3). Although vary few studies have directly compared environmentally measured concentrations with nominal exposure levels, available evidence indicates that sublethal and lethal effects in freshwater mussels generally occur at concentrations exceeding those expected under natural aqueous conditions [240]. For instance, a study demonstrated that such effects manifest only at exposure levels substantially higher than environmentally realistic concentrations, suggesting a relatively low ecological risk from current PPCP contamination [240].

The key mechanistic pathways through which these pollutants act, including oxidative imbalance, lipid peroxidation, AChE inhibition, and transcriptional impairment are summarized in Fig. 3, which integrates findings across multiple molluscan species and pollutant classes.

Table 3 Overview of the toxicological effects of different kinds of emerging pollutants on mollusc species

Compound	Size/Concentration (mg kg^{-1})	Nominal vs Measured	Exposure duration	Exposure Type (Experimental /Field)	Biomarker	Tissue	Species	Effects	Referen
Drospirenone	0 to 3 mg L^{-1}	Nominal	21 days	Experimental	GST	Mussels	*Lampsilis siliquoidea* (freshwater)	• Filtering activity ↓ • Survival and algal clearance rates have no effects	[229]
Diclofenac and cyclosporine A	0.001 to 1 10 µg/ L	Nominal	3 days	Experimental	ROS	Adult body	*Lymnaea stagnalis* (freshwater)	• Metabolism pathways differ significantly • Induced immune responses	[230]
Ibuprofen	1 µg L^{-1} 0.001 and 0.1	Nominal	1, 4, and 7 days	Experimental	MT, CYP450, GSH, GR, CAT, GPx	Gills	*Crassostrea gigas* (marine)	• This caused the upregulation of GST and cell viability of hemocytes • GPx activity ↓ • Inhibition of GR activity suggests an imbalance in the gill redox system	[231]
Estradiol	5-625 ng L^{-1} 0.000005 to 0.000625	Nominal	28 days	Experimental	GR, GPx, GSTs, GLY	Calms	*Ruditapes philippinarum* (marine)	• Metabolic and oxidative status alterations induced • GSTs activity ↑	[232]
Polystyrene NPs	0.1 to 100 mg L^{-1}	Nominal	1, 3, and 5 hours	Experimental	ROS	Gametes	*Crassostrea gigas* (marine)	• Oxidized glutathione ↑ • Changes in the antioxidant defence system • Incline in reactive oxygen species • Effect immune system • Malformation of the nervous system • Changes in growth and development	[233]
Polystyrene MPs	3 µm (50 to 10,000)	Nominal	48 hours, 3, 6 and 9 days	Experimental	ROS	Larvae	*Mytilus galloprovincialis* (marine)	• Feeding efficiency↓ • Physical and transcriptional impairments ↑	[234]
Paracetamol	50 0.05 and 500 µg L^{-1} 0.5	Nominal	96 hours	Experimental	CAT, GSTs, ChEs	Snails	*Gibbula umbilicalis* (marine)	• The levels of lipid peroxidation ↓ • CAT ↓	[235]
Paracetamol	0 500 µg L^{-1} to 0.5	Nominal	96 hours	Experimental	CAT, GSTs, LPO	Mussels	*Mytilus spp* (marine)	• Alteration of antioxidant defenses and damage	[236]
Polyethylene MPs	10 mg MP L^{-1}	Nominal	24 hours	Experimental	-	Larvae	*Crassostrea gigas* (marine)	• No significant alterations • Maximum swimming speed ↓of larva • No difference in the swimming behaviour of adults	[237]
Microplastics	0.0 to 0.8 g L^{-1} 800	Nominal	24 hours	Experimental	-	Mussels	*Dreissena bugensis* (freshwater)	• Affect survival • Mortality ↑ • Filtration rate ↓ • Reproduction rate ↓ • Overall fitness ↓	[238]
PE and PET	10 0.01 and 1000 µg L^{-1}	Nominal	21 days	Experimental	HK, PK, GPT	Adult oysters	*Crassostrea gigas* (marine)	• Induced alterations in metabolic profiles • Histopathological damage • Alternation in inflammatory responses	[239]

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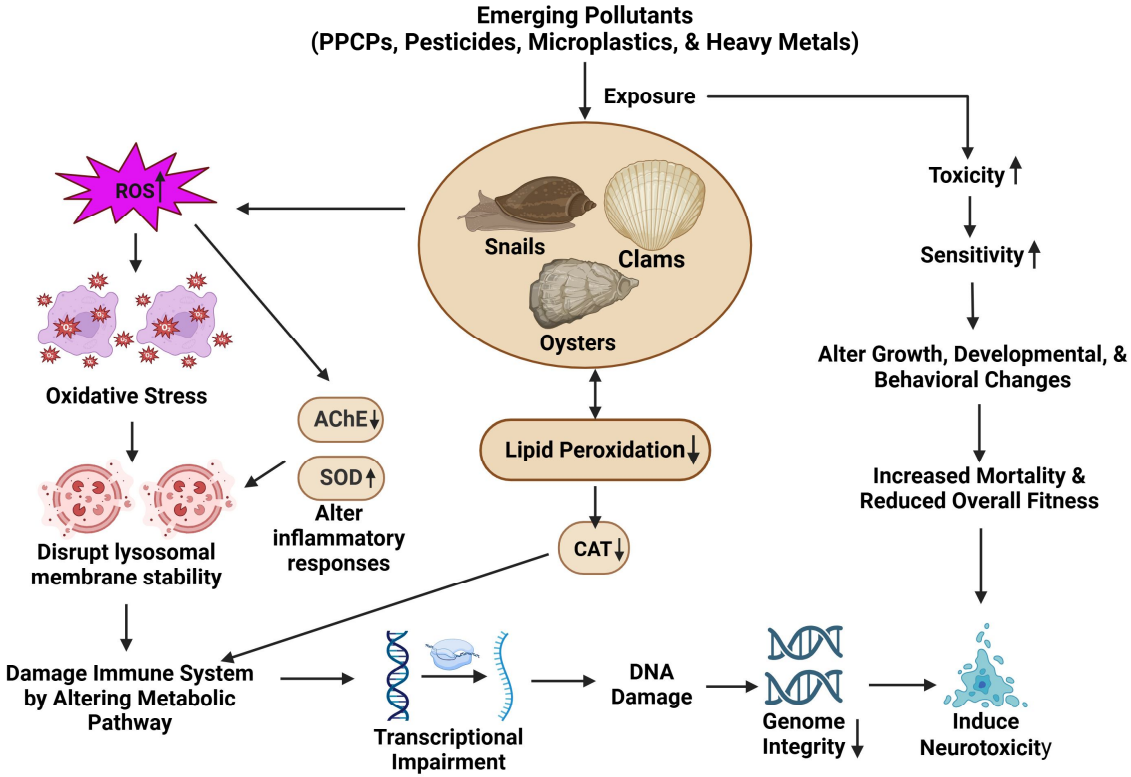
Polystyrene MPs	0.01 to 514 µm (100 to 4000[1])	Nominal	120 hours	Experimental	-	Adult snails	*Potamopyrgus antipodarum* (freshwater)	• Altered the behavior • Mortality ↑
Microplastics	1.0 mg L kg^{-1}	Nominal	14 days	Experimental	SOD, CAT, AChE	Mussels	*Unio tumidus* (freshwater)	• SOD ↑ • AChE ↓ • Lysosomal integrity ↑ • Genome integrity of cells of different tissues ↓
Polystyrene MPs	0.9 µm (10^6)	Nominal	3 days	Experimental	-	Digestive glans, gills	*Mytilus trossulus* (marine)	• Digestive gland cells damage • DNA damage • Neurotoxicity induced • Changes in digestive enzymes
Polystyrene MPs	9.57 items/individual	Nominal	7 days	Experimental	AChE	Edible molluscs	*Mactra veneriformis*, *Bullacta exarate*, *Cyclina sinensis* (marine)	
Polyethylene MPs	10 µg L^{-1} 0.01	Nominal	14 days	Experimental	-	Clams	*Ruditapes decussatus* (marine)	• Immune system disruption • Nutrient filtration function↓ • Destabilization of their lysosomal membranes • Viability of haemocytes ↓ • Hemolymph ↓ • Ca^{2+} ATPase activity increased in the mantle edge
Polyethylene MPs	0.02 20 µg L^{-1}	Nominal	24, 72 and 120 hours	Experimental	CA	Gastropod	*Pomacea canaliculata* (freshwater)	
Heavy metals	0.137 mg kg^{-1}	Nominal		Experimental	-	Whole body	*Bivalves* (marine)	• Cancer risk ↑

910 Arrow (↑) (↑) - Signifies increase; Arrow (↓) (↓) - Signifies decrease/reduction. PE - Polyethylene; PET - Polyethylene terephthalates; GSTs-
911 Glutathione S-transferase; ROS- Reactive oxygen species; CAT- Catalase; ChEs- Cholinesterases; LPO- Lipoperoxidation; HK- Hexokinase;
912 PK- Pyruvate kinase; GPT- Glutamic pyruvic transaminase; AChE- Acetylcholinesterase; CA- Carbonic anhydrase; CYP450 - Cytochrome
913 P450; MT- Metallothione; GSH- Glutathione; GR- Glutathione reductase; GPx- Glutathione peroxidases; GLY- Glycogen; NPs-
914 Nanoparticles/plastics; MPs- Microplastics



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916 **Fig. 3** ~~Toxicological Effects~~Mechanistic pathways of ~~Emerging Pollutants~~toxicological effects of emerging pollutants (EPs) on ~~Mollusks. The~~

917 molluscs. This figure illustrates the ~~mechanism by~~ mechanistic pathways through which emerging pollutants such as pharmaceuticals and

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personal care products (PPCPs), pesticides, microplastics, and heavy metals, affect molluscan physiology. Upon exposure ~~to emerging pollutants affects mollusks. Pollutant exposure triggers~~ , these contaminants induce oxidative stress~~, leading to increased~~ characterized by the overproduction of reactive oxygen species (ROS~~) and disrupting~~), increased superoxide dismutase (SOD) activity, and decreased catalase (CAT) levels, leading to lipid peroxidation and disruption of lysosomal membrane stability. ~~This oxidative damage reduces~~Altered acetylcholinesterase (AChE) activity~~, increases superoxide dismutase (SOD) levels, and impairs~~ and inflammatory responses~~, affecting~~ contribute to neurotoxicity and immune ~~function. Additionally, lipid peroxidation decreases catalase (CAT) levels, causing DNA damage and system impairment. These biochemical disturbances further cause~~ transcriptional impairment, ~~which undermines~~DNA damage, and loss of genome integrity ~~and may induce neurotoxicity. These~~ , resulting in growth, developmental, and behavioral changes ~~increase~~. Collectively, these effects elevate organismal toxicity and sensitivity ~~to toxicity, alter growth, behavior, and development, and reduce~~, ultimately increasing mortality and reducing overall fitness~~, potentially raising mortality rates~~ in molluscan species such as snails, clams, and oysters.

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4.4 Impacts on crustaceans

Owing to their open circulatory system, crustaceans are particularly susceptible to toxic substances that readily enter their bodies, leading to potential poisoning [246]. Emerging pollutants, such as pesticides, micro- and nanoplastics [247], pharmaceuticals, personal care products [62], and heavy metals, cause various harmful effects. These effects include acute toxicity, increased oxidative stress, reduced antioxidant capacity, immune system alterations [248], inflammatory bowel diseases, and behavioural changes such as impaired swimming, feeding, and reproduction. These pollutants can also decrease growth, lipid metabolism, and survival rates [249] (Table 4). When exposed to prometryn, *Eriocheir sinensis* undergoes apoptosis *via* PI3k–Akt signalling pathway inhibition linked to mitochondrial pathways, thus triggering immune responses, oxidative stress, and reduced cell viability, leading to apoptosis [250]. Similar results can also be observed when exposed to other pesticides, such as glyphosate and imidacloprid, which resulted in elevated levels of GOT and GPT, indicating hepatopancreatic cell damage [251], enriched fatty acids and biosynthesis pathway disruption due to the downregulation of genes such as LipT1, ACC, and acyl-CoA 9D, which are involved in fatty acid synthesis [252]. Moreover, the entry of ammonia into the gills and mixing with the blood can affect the oxygen-carrying capacity and damage organ structures [253]. In one study, atrazine exposure in *Faxonius virilis* increased oxidative stress and toxic effects by impairing detoxification, altering gene expression, and impacting transcription factors such as nuclear factor erythroid 2-related factor 2, which regulate cellular defense against toxicity and oxidative stress [254]. The expression of genes such as heme oxygenase-1 and glutathione S-transferase, which are crucial for inflammation prevention and detoxification, can also change [255].

~~The accumulation of microplastic fibres in the stomach can lead to false satiation, reduced body mass, and decreased food intake in *Nephrops norvegicus* [256], whereas in the case of E. *sinensis*, the accumulation of microplastic fibres, such as SOD, GOT, GSH, GPx, CAT, and alanine aminotransferase, is disrupted in response to microplastic accumulation in the hepatopancreas [257]. However, high-density polyethylene microplastic exposure in shrimp induces nodule formation in the hepatopancreas, chronic inflammation, tissue damage, and reduced immunity-related gene expression, leading to cellular dysfunction and mortality [258]. Excessive exposure to manganese in *N. norvegicus* has neurotoxic effects on the neuromuscular system, specifically through calcium-regulated ion channel inhibition, which reduces muscle performance and affects the brain, optic nerve, and ganglia, potentially leading to blindness [259]. Additionally, exposure to *Carcinus maenas*to copper causes lysosomal stability alterations, neurotoxicity, increased hemolymph, and changes in AChE and MT expression levels [260]. In contrast, *Pachygrapsus marmoratus* showed no significant AChE change but experienced increased SOD and CAT activity with reduced glutathione reductase [261]. When exposed to mercury, such an effect was also observed in the crab *C.* maenas, which resulted in decreased AChE activity, affecting neurotransmitter activity [262]. Additionally, when *P. serratus* is exposed to fenitrothion, a pesticide [263], its behaviour changes, and it inhibits AChE activity [259]. Azamethi-treated lobsters also presented increased mortality due to altered stress biomarkers and oxidative damage and inhibited ChE activity, affecting muscle regulation, hepatosomatic and gonadosomatic indices, and behaviour, leading to apoptosis without morphological changes [264] (Fig. 4).~~

4.4 Impacts on crustaceans

Owing to their open circulatory system, crustaceans are particularly susceptible to toxic substances that readily enter their bodies, leading to potential poisoning [254]. EPs, such as pesticides, MPs and NPs [255], PPCPs [72], and heavy metals, cause various harmful effects, including acute toxicity, oxidative stress, reduced antioxidant capacity, immune alterations [256], inflammatory bowel diseases, and behavioural impairments affecting swimming, feeding, and reproduction. These pollutants also contributed to decreased growth, disrupted lipid metabolism, and lowered survival rates [257] (Table 4).

For instance, exposure to the herbicide prometryn in *Eriocheir sinensis* have shown to induce apoptosis through inhibition of the PI3K–Akt signalling pathway linked to mitochondrial dysfunction, triggering oxidative stress, immune dysregulation, and cell death [258]. Similar outcomes were observed following exposure to pesticides such as glyphosate and imidacloprid, which elevated glutamate oxaloacetate transaminase (GOT) and glutamate pyruvate transaminase (GPT) levels, indicating hepatopancreatic cell damage [259]. These compounds also resulted in disruption of fatty acid biosynthesis by downregulating genes such as *LipT1*, *ACC*, and *acyl-CoA-9D*, key regulators of lipid metabolism [260].

Ammonia entry through the gills can impair oxygen transport and damage organ structure [261], while atrazine exposure in *Faxonius virilis* can increase oxidative stress and toxicity by altering detoxification mechanisms, gene expression, and transcription factors such as nuclear factor erythroid 2–related factor 2 (*Nrf2*), which mediates cellular antioxidant responses [262]. Additionally, changes in the expression of *heme oxygenase-1* and *glutathione S-transferase* affected detoxification and inflammation regulation [263].

The ingestion and accumulation of MPs fibres also induced substantial physiological stress. For example, in *Nephrops norvegicus*, accumulated fibres in the stomach caused false satiation, reduced body mass, and lowered food intake [264]. In *E. sinensis*, MPs accumulation disrupted antioxidant enzyme activities such as SOD, GOT, GSH, GPx, CAT, and alanine aminotransferase in the hepatopancreas [265]. High-density polyethylene MPs further induced nodule formation, chronic inflammation, and tissue damage in shrimp hepatopancreas, impairing immune gene expression and leading to cellular dysfunction and mortality [266].

Heavy metals similarly exert neurotoxic and metabolic effects. Excessive manganese exposure in *N. norvegicus* have shown to impair the neuromuscular system by inhibiting calcium-regulated ion channels, reducing muscle performance, and damaging the brain, optic nerve, and ganglia, potentially causing blindness [267]. Copper exposure in *Carcinus maenas* resulted in lysosomal stability alteration, increased neurotoxicity, thereby affecting hemolymph composition, and modifying AChE and MT expression levels [268]. In contrast, *Pachygrapsus marmoratus* exhibited no significant AChE inhibition but demonstrated increased SOD and CAT activity alongside reduced glutathione reductase levels [269].

Mercury exposure in *C. maenas* similarly decreased AChE activity, disrupting neurotransmitter regulation [270], while exposure of *Palaemon serratus* to the pesticide fenitrothion resulted in behavioural alterations and inhibition of AChE [271]. Likewise, azamethi-treated lobsters exhibited increased mortality due to oxidative damage, altered stress biomarkers, and inhibited ChE activity, affecting muscle regulation, hepatosomatic and gonadosomatic indices, and behaviour, ultimately leading to apoptosis without morphological changes [272].

Although most available studies rely on nominal laboratory concentrations, a few have incorporated environmentally measured levels. For instance, sulfamethoxazole and erythromycin exposures in freshwater shrimp demonstrated oxidative stress, membrane hyperpolarization, and growth inhibition even at environmentally relevant concentrations [273]. Similarly, deltamethrin exposure caused acute neurotoxic responses at both nominal and measured concentrations, with lethality observed after prolonged exposure [274]. While laboratory studies showed clear physiological impairments, the actual ecological risk under current environmental conditions may appear relatively low, though chronic impacts remain possible.

Overall, these findings demonstrated that EPs induced oxidative stress, disrupted antioxidant and endocrine functions, altered behavioural and metabolic responses, leading to apoptosis and mortality across multiple crustacean taxa (Fig. 4). The underlying mechanisms of these effects, including oxidative imbalance, immune suppression, enzymatic modulation (AChE, SOD, CAT, GST, MDA), and endocrine disruption are summarized in Fig. 4, which integrates evidence from experimental-based studies, while detailed toxicological endpoints are compiled in Table 4.

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1018 **Table 4** Summary of the toxicological effects of emerging pollutants on crustacean species

Compound	Size/ Concentration (mg kg⁻¹)	Nominal vs Measured	Exposure duration	Exposure Type (Experimental/Field)	Biomarker	Tissue	Species name	Effects Effect
Carbamates	0.1 μg L⁻¹ 0001	Nominal	7 days	Experimental	AChE, GABA	Whole body	Shrimps (freshwater)	• Sensitivity to acetylcholinesterase • Inhibiting insecticides and fungi ↓ • Growth, feeding, reproduction ↓ • Mortality, immobilization ↑
Methylparaben	0.017 mg L⁻¹	Nominal	9 days	Experimental	CAT, GST, ChE	Larva, embryo	Shrimps (marine)	• Antioxidant defences (cat (CAT) and glutathione-S-transfe (GST), neural activity cholineste (che Che), xenob biotransformation, survival ↓
Diazinon	0.0120 mg L⁻¹	Catecholamine, glycogen	Muscle, blood	Shrimps				• This is due to the metabolic compensation mechanism of the shrimp in reaction to the stress produced • Triglyceride level ↑ [267]
Diazinon, Azinphos-methyl	0.0120 0.0101 mg	Nominal Catecholamine, glycogen	Muscles,	Shrimps Experimental	Catecholamine, glycogen	Muscle, blood	Shrimps(marine)	• Triglyceride level ↑ • This is due to the meta

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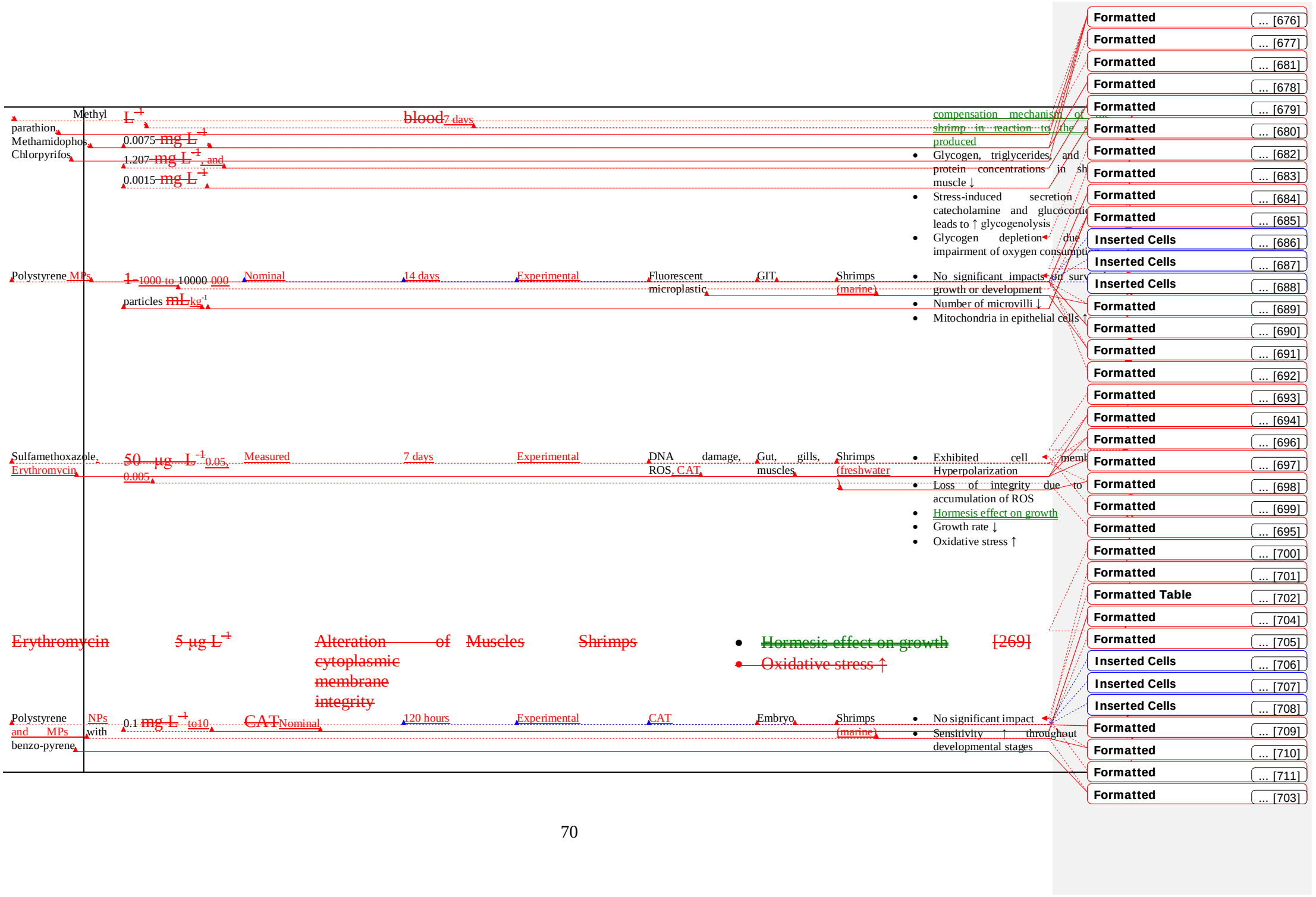
Methyl parathion
Methamidophos
Chlorpyrifos
L⁻¹
blood 7 days
0.0075 mg L⁻¹
1.207 mg L⁻¹, and
0.0015 mg L⁻¹
compensation mechanism of shrimp in reaction to the s produced
• Glycogen, triglycerides, and protein concentrations in sh muscle ↓
• Stress-induced secretion catecholamine and glucocorti leads to ↑ glycogenolysis
• Glycogen depletion due impairment of oxygen consumpti
Polystyrene MPs
1 1000 to 10000 000 particles mL kg⁻¹
Nominal
14 days
Experimental
Fluorescent microplastic
GIT
Shrimps (marine)
• No significant impacts on surv growth or development
• Number of microvilli ↓
• Mitochondria in epithelial cells ↑
Sulfamethoxazole, Erythromycin
50 µg L⁻¹ 0.05, 0.005
Measured
7 days
Experimental
DNA damage, ROS, CAT
Gut, gills, muscles
Shrimps (freshwater)
• Exhibited cell memb Hyperpolarization
• Loss of integrity due to accumulation of ROS
• Hormesis effect on growth
• Growth rate ↓
• Oxidative stress ↑
Erythromycin
5 µg L⁻¹
Alteration of cytoplasmic membrane integrity
Muscles
Shrimps
• Hormesis effect on growth
• Oxidative stress ↑
[269]
Polystyrene NPs and MPs with benzo-pyrene
0.1 mg L⁻¹ to10
CAT
Nominal
120 hours
Experimental
CAT
Embryo
Shrimps (marine)
• No significant impact
• Sensitivity ↑ throughout developmental stages
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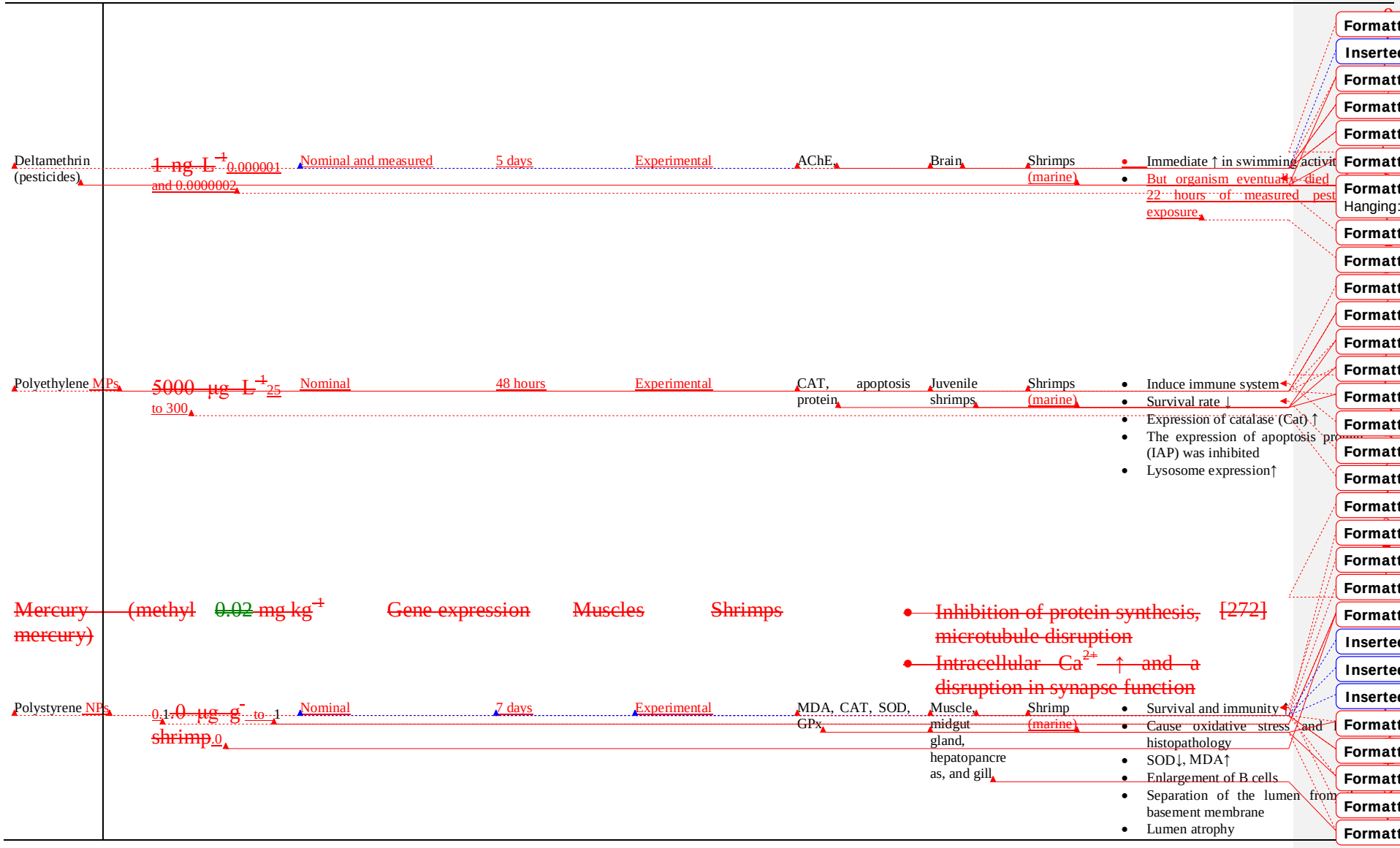
Deltamethrin (pesticides)
1 ng L-1 0.000001 and 0.0000002
Nominal and measured
5 days
Experimental
AChE
Brain
Shrimps (marine)
Immediate ↑ in swimming activit
But organism eventually died 22 hours of measured pest exposure
Polyethylene MPs
5000 μg L-1 25 to 300
Nominal
48 hours
Experimental
CAT, apoptosis protein
Juvenile shrimps
Shrimps (marine)
Induce immune system
Survival rate ↓
Expression of catalase (Cat) ↑
The expression of apoptosis pro (IAP) was inhibited
Lysosome expression↑
Mercury (methyl mercury)
0.02 mg kg-1
Gene expression
Muscles
Shrimps
Inhibition of protein synthesis, microtubule disruption
Intracellular Ca2+ ↑ and a disruption in synapse function
[272]
Polystyrene NPs
0.1, 0 μg g- to 1 shrimp.0
Nominal
7 days
Experimental
MDA, CAT, SOD, GPx
Muscle, midgut gland, hepatopancreas, and gill
Shrimp (marine)
Survival and immunity
Cause oxidative stress and
histopathology
SOD↓, MDA↑
Enlargement of B cells
Separation of the lumen from basement membrane
Lumen atrophy
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Arrow ~~(↑)~~ (↑) - Signifies increase; Arrow ~~(↓)~~ (↓) - Signifies decrease/reduction. SOD - Superoxide dismutase; CAT - Catalase; MDA - Malonidiadehyde; GST - Glutathione S-transferase; CYP450 - Cytochrome P450; GSH - Glutathione; G6PDH - Glucose-6-phosphate dehydrogenase; GPx - Glutathione peroxidase; VTG- Vitellogenin; TH - Thyroid hormone; HA - Hemagglitinin; GHR - Growth hormone receptor; GIT - Gastro intestinal tract; ROS - Reactive oxygen species; GABA - Gamma-aminobutyric acid; GR - Growth hormone; T3 - Triiodothyronine; T4 - Thyroxine; MT - Metallothionein; ChE - Cholinesterase; TNF - Tumor necrosis factor; MMP2 - Matrix metalloproteinase; BDE - Brominated diphenyl ethers; 4-MBC - 4-methylbenzylidene; BPF - Bisphenol F; DEHP - Di-2-ethylhexyl phthalate; DBP - Dibutyl phthalate; PFOA - Perfluorooctanoic acid; CPX - Ciprofloxacin; H_2O_2 - Hydrogen peroxide; NPs- Nanoparticles/plastics; MPs- Microplastics

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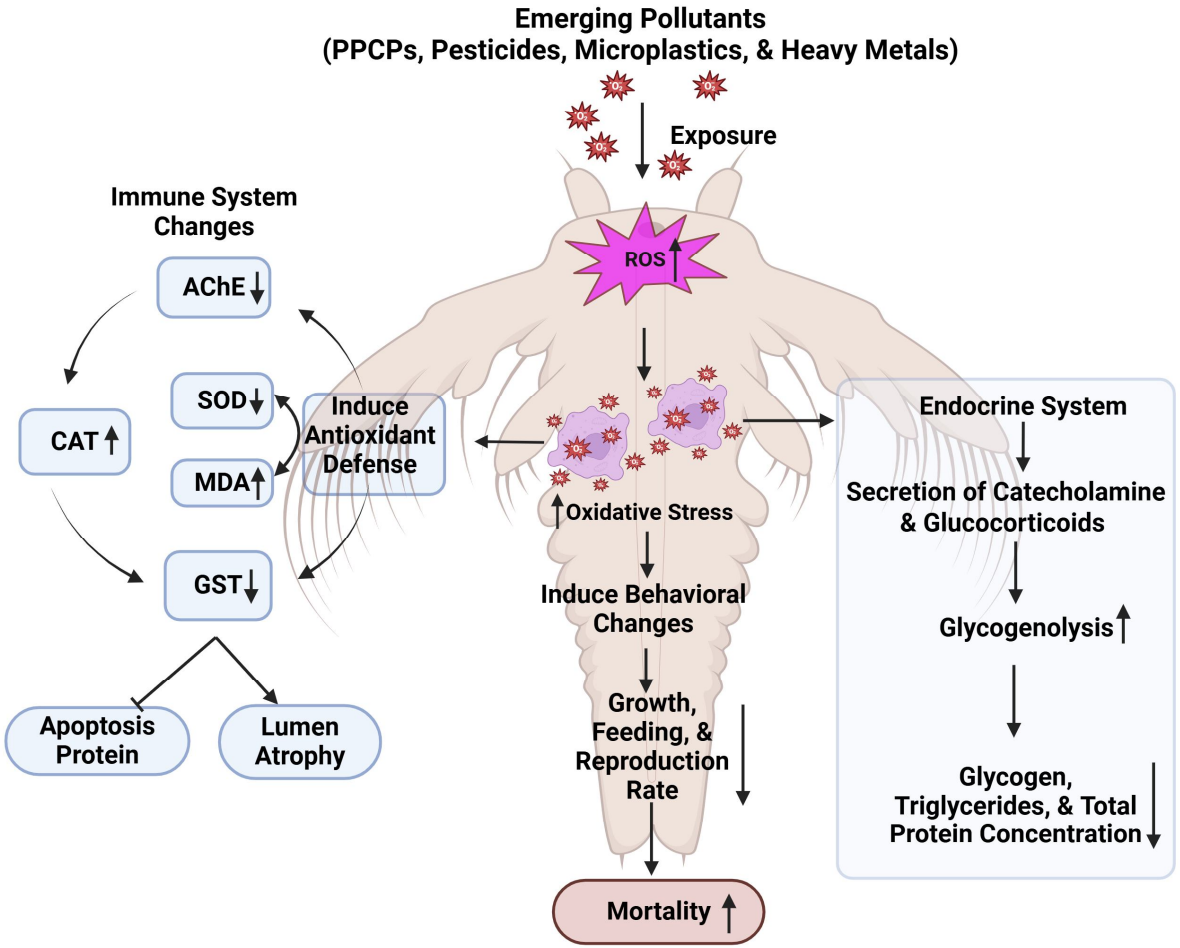

Fig. 4 ~~Toxicological~~ Schematic representation~~Effects~~ of ~~Emerging Pollutants on Crustaceans. The figure illustrates~~ the ~~mechanism by which exposure to~~mechanistic pathways underlying the toxicological effects of emerging pollutants ~~affects~~ (PPCPs, pesticides,

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microplastics, and heavy metals) on crustaceans. ~~The~~Pollutant exposure ~~increases~~ induces oxidative stress and reactive oxygen species (ROS~~), causing oxidative stress and disrupting the~~) generation, triggering immune system ~~by altering antioxidant defenses, such as decreasing~~ alterations, for instance, changes in acetylcholinesterase (AChE~~) and~~), superoxide dismutase (SOD~~) levels,~~), catalase (CAT), glutathione S-transferase (GST), and ~~increasing~~ malondialdehyde (MDA~~).~~), endocrine disruption, and oxidative imbalance. These ~~changes~~cascades lead to ~~immune impairments~~altered antioxidant defence, apoptosis, ~~and lumen atrophy. In the endocrine system, exposure to pollutants stimulate catecholamine and glucocorticoid secretion, which increases glycogenolysis but reduces glycogen, triglycerides, and total protein levels. Altogether, these effects impair~~metabolic impairment, behavioural changes, and reduced growth, feeding, and reproduction~~, induce behavioural changes, and raise~~ rates, ultimately increasing mortality ~~risk in crustaceans~~.

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4.5 Impacts on aquatic mammals

Emerging contaminants, including pesticides, heavy metals, pharmaceuticals, personal care products (PPCP), and other pollutants, present significant threats to top predators and aquatic mammals within aquatic ecosystems [274]. These pollutants can induce various alterations in behaviour, physiology, the immune response, endocrine function, and reproductive health, contributing to increased mortality and decreased population dynamics [275] (Table 5). Ingestion of microplastics has been shown to reduce feeding efficiency, reproductive success [276], energy reserves, and gastrointestinal health. Microplastics can also alter gastrointestinal physiology, induce cytotoxicity, disrupt gene expression, and cause histomorphological changes in organs such as the kidneys while inducing inflammation and increasing oxidative stress [277] (Fig. 5). Exposure to such pollutants can also result in microbial imbalance, damage to the gut epithelium, reduced mucosal protection [171], and immune cell toxicity. Microplastic accumulation in tissues such as the skin, lungs, liver, kidneys, muscles, blubber, bones, and reproductive organs has extensive physiological effects [278]. However, these contaminants are readily detectable in developing animals, including calves, through amniotic fluid, placental tissue, maternal urine, and breast milk, contributing to various developmental impacts [279]. Marine mammals, such as bottlenose dolphins, killer whales, pilot whales, fin whales, and sperm whales, can serve as indicators of pollution due to their high trophic positions and long lifespans [278]. However, when these marine organisms are exposed to pollutants such as phthalates (PAEs), dolphins mainly present changes in biomarkers related to stress and oxidative damage, including markers such as malondialdehyde and lipid peroxidation, reflecting reactive oxygen species (ROS) production and steroid hormone alterations [280]. Such alterations in hormonal activity can further lead to increased blood volume, blood pressure, osmoregulatory issues, and cardiovascular effects [281].

~~Endocrine disrupting compounds (EDCs), such as pharmaceuticals, pesticides, metals, and PPCPs, are lipophilic in nature and tend to accumulate mostly in adipose tissues, resisting metabolic breakdown, slowing excretion, and binding to endocrine receptors, where they alter hormone synthesis, signalling, and endocrine functions [282]. EDCs can also affect aquatic mammals' growth, reproduction, metabolism, thyroid function, and immune responses, leading to developmental issues, cancer, miscarriages, and population decline [283]. The EDC ethinyl estradiol, for example, can increase apoptosis and reduce cell viability during DNA synthesis. At the same time, carbamazepine can disrupt lymphocyte transformation and the completion of mitosis [284]. Additionally, exposure to pesticides such as DDT and its metabolites can impact steroid signalling by interfering with gonadal, adrenal, and placental functions, influencing stress responses, metabolism, and reproductive health [285]. A decrease in superoxide dismutase (SOD), which is essential for diving stress protection, was also observed, along with increased ROS production, oxidative stress, and suppressed immune functions [286]. However, pollutants bioaccumulate through dietary intake, dermal absorption, and respiratory pathways in water or air, which serve as models for studying contaminant effects on such organisms via bioaccumulation, bioconcentration, and biomagnification factors [287]. Bioaccumulated pollutants can impact behaviour, physiology, reproduction, and overall population health at the tissue, molecular, and ecosystem levels [1]. These contaminants can be transferred through trophic levels, impacting population health and contributing to species decline due to increased toxicity [56].~~

EPs, including pesticides, heavy metals, pharmaceuticals, personal care products (PPCP), and other pollutants, present significant threats to top predators and aquatic mammals within aquatic ecosystems [281]. These pollutants can induce various alterations in behaviour, physiology, the immune response, endocrine function, and reproductive health, contributing to increased mortality and decreased population dynamics [282] (Table 5).

Ingestion of microplastics has been shown to reduce feeding efficiency, reproductive success [283], energy reserves, and gastrointestinal health. Microplastics can also alter gastrointestinal physiology, induce cytotoxicity, disrupt gene expression, and cause histomorphological changes in organs such as the kidneys, while inducing inflammation and increasing oxidative stress [284]. Exposure to such pollutants can also result in microbial imbalance, damage to the gut epithelium, reduced mucosal protection [178], and immune cell toxicity. Microplastic accumulation in tissues such as the skin, lungs, liver, kidneys, muscles, blubber, bones, and reproductive organs has extensive physiological effects [285]. However, these contaminants are readily detectable in developing animals, including calves, through amniotic fluid, placental tissue, maternal urine, and breast milk, contributing to various developmental impacts [286]. Marine mammals, such as bottlenose dolphins, killer whales, pilot whales, fin whales, and sperm whales, can serve as indicators of pollution due to their high trophic positions and long lifespans [285]. However, when these marine organisms are exposed to pollutants such as phthalates (PAEs), dolphins mainly present changes in biomarkers related to stress and oxidative damage, including markers such as malondialdehyde and lipid peroxidation, reflecting reactive oxygen species (ROS) production and steroid hormone alterations [287]. Such alterations in hormonal activity can further lead to increased blood volume, blood pressure, osmoregulatory issues, and cardiovascular effects [288].

Endocrine-disrupting compounds (EDCs), such as pharmaceuticals, pesticides, metals, and PPCPs, are lipophilic in nature and tend to accumulate mostly in adipose tissues, resisting metabolic breakdown, slowing excretion, and binding to endocrine receptors, where they alter hormone synthesis, signalling, and endocrine functions [289]. EDCs can also affect aquatic mammals' growth, reproduction, metabolism, thyroid function, and immune responses, leading to developmental issues, cancer, miscarriages, and population decline

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Toxicological evidence indicates that endocrine-disrupting pollutants (EPs) interfere with mammalian physiology primarily through endocrine modulation and oxidative stress–mediated mechanisms. Mechanistically, EPs can bind to or antagonize hormone receptors (e.g., estrogenic, androgenic, and thyroid receptors), and altering gene transcription and disrupting hormonal homeostasis. Concurrently, these compounds induce reactive oxygen species (ROS) generation, leading to oxidative damage of lipids, proteins, and nucleic acids, impairing cellular signaling and metabolic pathways (Fig. 5). The bioaccumulation of these pollutants within tissues results in their biomagnification across trophic levels, thereby amplifying physiological and reproductive impairments at higher levels of the food web [1]. Studies showed that these contaminants can be transferred through trophic levels, impacting population health and contributing to species decline due to increased toxicity [66].

Table 5 Overview of the toxicological impacts of different emerging pollutants on mammals and reptile species

Compound	Size/ Concentration ~~(mg·kg^{-1})~~	Nominal vs Measured	Exposure duration	Exposure type (Experimental/ field)	Biomarker	Tissue	Species name	~~Effects~~ Effect	References
Atrazine	~~2 µg/L~~ 0.002	Nominal	24 hours	Experimental	GnRH2, estradiol, mRNA	Hypothalamus	Turtle- *Chelydra serpentine*	• Steroidogenesis and hypothalamus-pituitary-gonad axis genes ↓ • Hypothalamic gene expression altered	~~[288]~~ [295]
Hg	0.168 ~~µg/g dw~~	Measured	-	Field	Corticosterone	Blood	Crocodiles	• Osmoregulation (sodium levels) and hepatic function (alkaline phosphatase levels) ↓ • Endocrine processes corticosterone levels ↓	~~[289]~~ [296]
Selenium	0.76 ~~µg/g dw~~	Measured	-	Field	Corticosterone	Blood	Crocodiles	• Na+ excess Se positively influences corticosterone levels against Hg	~~[289]~~ [296]
Lead	0.04 ~~µg/g dw~~	Measured	-	Field	Corticosterone, ALT	Blood	Crocodiles	• Organism ↑ the production of ALT (alanine aminotransferase concentration)	~~[289]~~ [296]
Megestrol acetate	0 to 60 mg	Nominal	1 year	Field	Cortisol, T3, T4	Blood, serum	*Tursiops truncatus*	• Appetite ↑ and suppress reproductive behaviour • Suppression of the HPA-axis results in changes to adrenal morphology	~~[290]~~ [297]
DEHP	0.01 and 5 mM	Nominal	49 hours	Experimental	DNA stability	GIT	*T. truncatus*	• Cytotoxic ↓ and genotoxic effect ↑ • Cell viability ↑ no cell death • Inhibition of cell proliferation ↑	~~[291]~~ [298]
DEHP	~~8.13 ng/ml~~ 0.00813	Measured	-	Field	Thyroxine	Serum and urine	*Tursiops truncatus*	• Impacting thyroid hormone homeostasis	~~[292]~~ [299]

Arrow ~~(↑)~~ (↑) - Signifies increase; Arrow ~~(↓)~~ (↓) - Signifies decrease/reduction. DEHP - Di-2-ethylhexyl phthalate; GIT - Gastrointestinal tract; Hg - Mercury; ALT - Alanine aminotransferase

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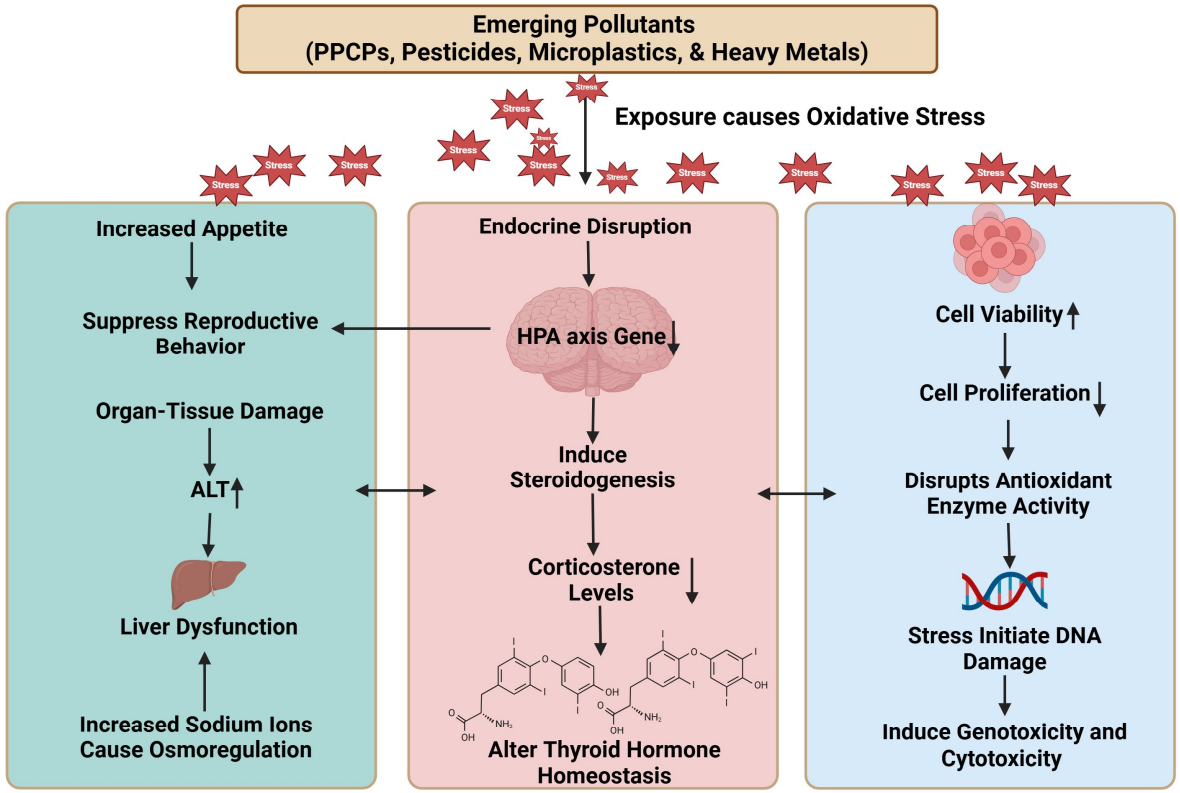

Fig. 5 ~~Toxicological~~ Mechanistic pathways~~Effects~~ of ~~Emerging Pollutants~~toxicological effects of emerging pollutants (EPs) on ~~Mammals~~mammals and ~~Reptiles. The~~reptiles. This figure represents the mechanistic pathways by which exposure to emerging pollutants affects aquatic mammals

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and reptiles. It highlights endocrine disruption leading to altered hormone levels such as decreased thyroid hormones, corticosterone levels, steroidogenesis, and impaired reproductive functions, alongside oxidative stress that ~~triggers~~induce DNA damage and cellular dysfunction. Increased alanine aminotransferase (ALT) levels lead to liver dysfunction and hypothalamic disruptionlike reduced hypothalamus-pituitary-gonad axis (HPA), resulting in behavioral changes and physiological impairments. Ultimately, these effects contribute to genotoxicity and cytotoxicity in aquatic species, inhibiting growth, and survival rates.

4.6 Impacts on reptiles

As apex predators with extensive geographic ranges, long life spans, and physiological adaptations, American alligators can accumulate pollutants such as heavy metals from both their prey and the environment in which they live ~~[293]. These toxic metals, including iron, cadmium, and lead, can often lead to increased metallothionein (MT) binding rates, aiding in detoxification processes, as these hepatic proteins circulate heavy metals through the circulatory system [222]. Crocodiles are also bioaccumulators that can experience negative physiological impacts from metals such as mercury, lead, and selenium (Table 5), affecting osmoregulation, hepatic and renal function, immune responses, and hormonal balance [294]. For example, lead exposure can affect alanine aminotransferase and glucose levels in crocodiles, disrupting hepatic function [295]. Similarly, when exposed to lead (Pb), marine turtles exhibit a reduced lymphocyte response and glucose levels, with increased globulin and total protein levels, which impact their inflammatory response [296]. Endocrine-disrupting compounds (EDCs), such as estrogen, can alter American alligators' reproductive structures, sex determination, immune competence, and proteins through dietary or embryonic transfer [297]. In reptiles, EDCs can cause thyroid, reproductive, and metabolic changes (Fig. 5), impacting gene expression and enzyme activity [298]. Phthalates, another EDC group, adversely affect loggerhead turtles (*Caretta caretta*) through ingestion, leading to bioaccumulation in blood and muscle tissues and impacting diving ability and metabolic activity [299]. Additionally, micro- and nanoplastics ingested by reptiles can cause respiratory issues, including breathing difficulties, physiological deterioration, and reduced feeding behaviour, often leading to death. Over time, these particles can induce physiological, biochemical, and genetic harm [300]. Direct and indirect exposure to certain pesticides can also harm reptiles. For example, atrazine exposure affects the expression of sex hormones and the proliferation of lymphocytes, altering sex determination. Maternal pesticide exposure~~

~~in *Caiman latirostris* resulted in decreases in eggshell porosity and lowered hatching and survival rates of the studied organism [255]~~[300]. These toxic metals, including iron, cadmium, and lead, can often lead to increased metallothionein (MT) binding rates, aiding in detoxification processes, as these hepatic proteins circulate heavy metals through the circulatory system [233]. Crocodiles are also bioaccumulators that can experience negative physiological impacts from metals such as mercury, lead, and selenium (Table 5), affecting osmoregulation, hepatic and renal function, immune responses, and hormonal balance [301]. For example, lead exposure can affect alanine aminotransferase and glucose levels in crocodiles, disrupting hepatic function [302]. Similarly, when exposed to lead (Pb), marine turtles exhibit a reduced lymphocyte response and glucose levels, with increased globulin and total protein levels, which impact their inflammatory response [303]. Endocrine-disrupting compounds (EDCs), such as estrogen, can alter American alligators' reproductive structures, sex determination, immune competence, and proteins through dietary or embryonic transfer [304]. Phthalates, another EDC group, adversely affect loggerhead turtles (*Caretta caretta*) through ingestion, leading to bioaccumulation in blood and muscle tissues and impacting diving ability and metabolic activity [305]. Additionally, micro- and nanoplastics ingested by reptiles can cause respiratory issues, including breathing difficulties, physiological deterioration, and reduced feeding behaviour, often leading to death. Over time, these particles can induce physiological, biochemical, and genetic harm [306]. Direct and indirect exposure to certain pesticides can also harm reptiles. For example, atrazine exposure affects the expression of sex hormones and the proliferation of lymphocytes, altering sex determination. Maternal pesticide exposure in *Caiman latirostris* resulted in decreases in eggshell porosity and lowered hatching and survival rates of the studied organism [263].

~~**4.7 Impacts on plankton and small invertebrates**~~

Plankton, including zooplankton, phytoplankton, and bacterioplankton, as well as small invertebrates such as insect larvae, form the baselines of aquatic food chains by typically moving near the water surface [301]. These primary consumers serve as biological indicators, offering insights into water quality and the impacts of pollutants, such as pesticides, pharmaceuticals, metals, and flame retardants (Table 6), which can disrupt aquatic ecosystems [302]. These organisms are widely distributed across freshwater and marine systems and play crucial roles in nutrient recycling, atmospheric carbon dioxide regulation, and fish biomass, all of which are essential for maintaining the equilibrium of the ecosystem [303]. As other aquatic organisms feed on such tiny organisms for their food, this organism's intake can indirectly and directly affect growth and development because harmful chemicals affect population dynamics. Pollutant accumulation in the bodies of such organisms can cause stress among primary consumers such as zooplankton and impact nutrient cycling, carbon sequestration, and food availability for relatively high trophic levels, ultimately threatening fish populations [304]. For example, when exposed to polystyrene microplastics, *Daphnia pulex* experiences reduced survival, reproduction, and growth, along with changes in oxidative stress markers such as SOD, GST, and HSP90 [305] (Fig. 6). Similarly, *D. galeata* exhibited delays in hatching and reduced lipid storage under similar conditions, causing metabolic disruptions and hindering embryonic development [306]. As more trophic organisms consume these zooplankton, microplastics bioaccumulate and biomagnify, leading to organ damage, malformations, and increased mortality in subsequent populations [307].

Additionally, endocrine disrupting compounds (EDCs), such as phthalates, PCBs, PAHs, PFCs, and flame retardants, severely impair plankton and small invertebrates by reducing growth and inducing reproductive abnormalities, thus increasing mortality rates [308]. Zooplankton, when exposed to PFOS, for example, presented elevated heart rates, reduced growth, and fewer offspring, with genetic impacts leading to changes in locomotion,

~~reduced swimming distance, and altered reproductive quality [309]. Mixtures of PFOA and PFOS further resulted in induced oxidative stress, disrupted enzymatic functions such as SOD and GST, and decreased reproduction rates [310]. Pesticides exacerbate these issues by targeting motor functions, potentially causing paralysis, hyperactivity, and spasms while disrupting mitochondrial oxidative phosphorylation and altering genes associated with growth and reproduction [311]. Xenobiotic exposure, as in *Mytilus galloprovincialis*, resulted in reduced detoxification enzymes, harming the gills, digestive systems, antioxidant defenses, and hemolymphatic parameters essential for cell volume regulation. This exposure also increased mortality, caused physiological malfunctions (Fig. 6), and led to many histopathological changes in critical organs, such as the gills and digestive glands [108].~~

In reptiles, exposure to Eps has been shown to generate reactive oxygen species (ROS), leading to oxidative stress that disrupts cellular homeostasis and metabolic processes. The resulting oxidative imbalance modulates the activity of key antioxidant and detoxification enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione S-transferase (GST). These biochemical alterations contribute to endocrine disruption, genotoxicity, and cytotoxicity, ultimately affecting thyroid, reproductive, and metabolic functions [307]. The mechanistic pathways underlying these effects encompassing oxidative stress induction, enzymatic modulation, and molecular interference are integrated in Fig. 5, with specific toxicological endpoints summarized in Table 5.

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Emerging pollutants (EPs) induces oxidative stress, disrupts antioxidant and endocrine regulation, and alters behavioral and metabolic responses. The underlying mechanisms involve oxidative imbalance, immune suppression, and modulation of key enzymatic activities (GPx, SOD, CAT, GST, MDA), leading to membrane destabilization and subsequent reductions in survival, growth, and reproductive success. Furthermore, studies

have highlighted the involvement of pro-inflammatory cytokines, such as TNF and IL-6, in promoting pathways associated with potential carcinogenic outcomes upon EPs exposure. These mechanistic interactions are summarized in Fig. 6.

1287 **Table 6** Overview of the toxicological impacts of different emerging pollutants on plankton and small invertebrate species

Compound	Size/ Concentration (mg kg^{-1})	Nominal vs Measured	Exposure duration	Exposure type (Experimental / field)	Biomarker	Tissue	Species name	~~Effect~~Effects	References
Sulfamethoxazole	1.2 ~~mg/L~~	Nominal	56 days	Experimental	TNF, Growth factor, DNA stability	Muscles, gut, intestine	Sea cucumber	• Thickness of muscle and mucosal layers ↓ • Inhibit growth and development	[312][319]
Dibutyl phthalate	4.31 ~~mg/L~~	Nominal	24 and 48 hours	Experimental	CAT, GST, MDA, GSH, GPx	Newly hatched neonates, adults	*Daphnia magna*	• Acute toxic effects • Oxidative damage ↑ • Antioxidant capacity ↓ • Inhibit cytochrome c • MDA levels ↑ in neonates only	[313][320]
Estradiol	~~10 ng/L~~ 0.00001	Nominal	3, 6 and 12 hours	Experimental	Sex steroid hormones (E2 and T, H102 and H090), interleukin-6 (H007), and dopamine (H170), E2 and testosterone	Gonads	*Daphnia magna*	• Transcriptional changes in the immune system, disintoxication, disease prevention, and the protein degradation pathway, intestinal immune networks for IgA production • Inflammatory bowel disease • Cytokine–cytokine receptor interaction was initially active, which promoted inflammation-associated cancer development; this intoxication-related gene followed a reverse "U"-shaped response (STEM6) with increasing E2	[314][321]
Azithromycin	0.005 ~~m~~	Nominal	7 days	Experimental	The activity of amylase (AMS) and Trypsin	Neonates	*Daphnia magna*	• Feeding behaviour inhibited • Accumulation of polysaccharides and total protein ↓ • AMS and Trypsin activity ↓ • It affects digestive enzyme activities and the oxidative stress-antioxidative system	[315][322]

		g / L								
Polyethylene	10-45 µm	Nominal	45 days after hatching (dph)	Experimental	CYP450, IL-1β	GIT	Sea bass larvae	• Mortality ↑ • Induce immunological effects and inflammatory response	[316][119]	
PROS	10 µM/L	Nominal	7 days	Experimental	Estradiol, VTG2, SOD, CAT	Gonads	*Daphnia magna*	• Alterations in developmental and reproductive genes • Fecundity ↓ that regulates moulting and reproduction	[317][323]	
Polychlorinated biphenyls	6.87 mg/L				CAT, GSH	Embryo	*Daphnia magna*	• Mortality ↑ • Alteration of the swimming behaviour resulted in the hyperactivity of embryos • Malformation ↑	[270]	
PFOA	1 µM/L	Nominal	7 days	Experimental	Estradiol, VTG2, SOD, CAT, GPx,	Gonads, liver	*Daphnia magna*	• Growth, reproduction, lipid metabolism, survival rate ↓ • Down regulation of genes involved in the development and reproductive process, including vtg2, vasa, EcRA, EcRB, usp, the HR3, ftz-F1, E74 and E75	[317][323]	
Methylene blue	4.7 µg/L 0.0047	Nominal	24 hours	Experimental	GST, SOD, MDA	Thoracic limb, intestine, tissues	*Daphnia magna*	• Growth and reproduction ↓ • Heart rate ↓ • Induce antioxidant stress • Thoracic limb beat frequency ↓	[318][324]	

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Estradiol		0-2 ~~mg/L~~	Nominal	24 hours	Experimental	MDA, CAT, H_2O_2 levels	Whole body	*Thalassiosira weissflogii*	• Growth and metabolism ↓ • High negative correlation with the cell count, growth rate, and biomass productivity • MDA levels dramatically ↑	[319] ~~[318]~~[325]

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Arrow (↑)- Signifies increase; Arrow (↓)- Signifies decrease/reduction. CYP-1a - Cytochrome P1a; CAT - Catalochrome; GST - Gastrointestinal tract; MDA - Malinodialdehyde; GSH - Glutathione; GPx - Gluthione peroxidase; PVC - Polyvinyl chloride; H_2O_2 - Hydrogen per oxidase; LDH - Lactate dehydrogenase; ChE - Cholinestrase; IL-1β - Interleukin-1β; VTG2 - Vitelliogenin; SOD - Supraoxide dimutase; PFOS- Perfluorooctane sulfonate; PFOA- Perfluorooctanoic acid.

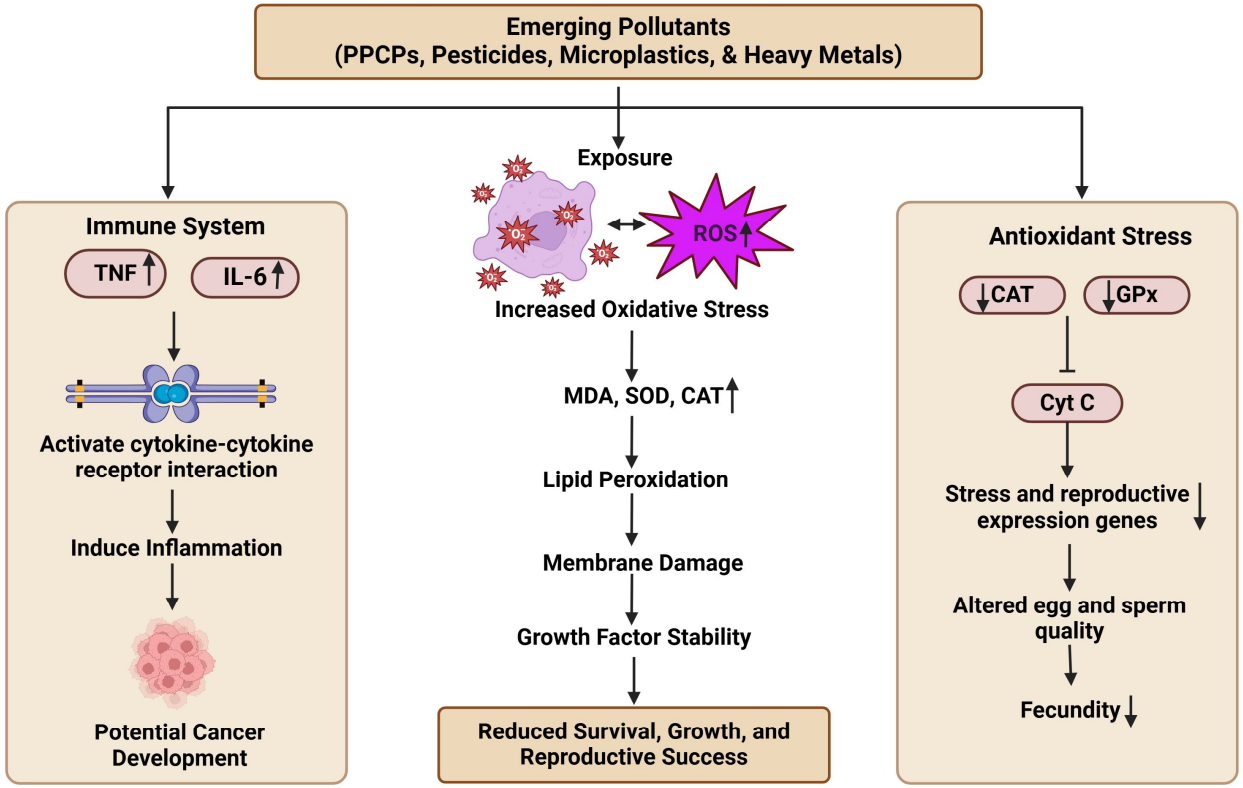

Fig. 6 ~~Toxicological Effects~~Mechanistic representation of ~~Emerging Pollutants on Planktons and Small Invertebrates. The figure illustrates~~ the ~~mechanism by~~toxicological pathways through which ~~exposure to~~ emerging pollutants ~~affects~~affect plankton and small invertebrates. This

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exposure triggers primary effects on cellular and molecular pathways, including oxidative stress, inflammation, and disruption of enzyme and gene activity. Key consequences involve increased oxidative markers such as increased Malondialdehyde (MDA, superoxide dismutase (SOD) and catalase (CAT) levels, leading to lipid peroxidation, increased inflammatory responses *via* cytokines, and decreased activity of detoxifying enzymes Cytochrome C (Cyt C). Organ-specific toxicity ~~manifests in the~~in aquatic animals disrupts digestive ~~system through membrane disruption, as well as reproductive~~membranes and ~~developmental issues marked by reduced~~ impairs reproduction and development by reducing hormone expression and ~~altered~~ gamete quality~~. These effects altogether contribute to~~, leading to growth inhibition, behavioral changes, and increased mortality~~ rates, ultimately lead to reduced survival, growth, and reproductive success in aquatic animals.~~

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5 Ecological impact of ~~ERs~~EPs and climate change

~~Emerging pollutants, such as pharmaceuticals, pesticides, personal care products, microplastics, and industrial chemicals, are abundantly found in ecosystems, especially aquatic environments, where they significantly impact biodiversity, water quality, and ecosystem stability as well as functionality [320]. A central problematic issue associated with these pollutants is their bioaccumulation and biomagnification, in which pollutants accumulate within organisms and become more concentrated at higher trophic levels, affecting apex predators such as birds, fish, and marine mammals [321]. This bioaccumulation in various tissues, mainly the liver, kidneys and blood, can cause long term toxicity and severe health problems within aquatic species [322]. Additionally, many pollutants, especially endocrine disrupting chemicals (EDCs), interfere with the functions of the endocrine system, leading to reproductive and developmental issues in aquatic species such as fish and amphibians [285]. These pollutants can also alter animal behaviour directly or indirectly, influencing critical activities such as feeding, mating, and migration [323]. Changes in behaviour can often reduce survival rates by impairing foraging efficiency and increasing vulnerability to predators [324]. For example, some pharmaceuticals have been shown to suppress the foraging behaviour of zebrafish, thus increasing their risk of predation [325], and the accumulation of pollutants in sediments and water further results in habitat degradation by reducing water quality and altering their physical and chemical properties [5]. Pollutants such as heavy metals and pesticides can make environments inhospitable, as they affect dissolved oxygen levels, pH balance, and water turbidity, which indirectly disrupt aquatic animal life by affecting their growth, health, survival, and development [326]. Nutrient rich pollutants from agricultural runoff can lead to eutrophication, which causes excessive harmful algal growth, oxygen depletion, and hypoxic conditions intricately linked to the loss of aquatic biodiversity [327]. Emerging pollutants also induce changes in species~~

composition, abundance, and ecosystem functions [328]. Certain pollutants selectively harm specific species by causing competition among the more resistant species and mortality of the most sensitive ones, often resulting in population declines or local extinctions [329]. More tolerant species may dominate, altering ecosystem dynamics and reducing biodiversity, ultimately affecting ecosystem stability and services. Furthermore, the release of pharmaceuticals, particularly antibiotics, can promote the spread of antibiotic resistance genes in bacteria, posing health risks to aquatic ecosystems and humans alike, as resistant bacteria can alter human food chains by consuming aquatic animals with resistance genes, which can further complicate disease management [330]. Prolonged pollutant exposure can also affect genetic mutations and cause physiological stress and shifts in gene expression, thus reducing the genetic diversity and fitness of the population [323].

In addition, emerging pollutants also indirectly contribute to climate change, amplifying its profound impacts on aquatic ecosystems and animals in complex but significant ways [60]. Climate change and pollution act as synergistic stressors; for example, increasing temperatures can increase the toxicity of certain toxic pollutants, leading to more severe effects on aquatic organisms, resulting in reduced animal resilience and impacting their growth, reproduction, and survival rates [331]. Warmer waters increase the metabolic rates of aquatic animals, which consequently increases the food consumption rate, intensifies pollutant uptake [332], accelerates toxic effects and negatively influences the survival rates of sensitive species, population size and overall ecosystem functionality [333]. Climate change can also lead to hypoxia or low oxygen levels due to increased temperatures, which deplete oxygen in water bodies, affecting aerobic metabolism and reducing the body size of aquatic species [334]. Many aquatic species are also forced to migrate to calmer waters or adapt to altered habitats, encountering pollutants that may not have previously been present in these areas, disrupting food web structure, reducing populations due to reproductive failure or

increasing predation/competition [335]. This can also influence temperature-dependent behavioural changes, exacerbated by the changing climate, and alter predator-prey interactions, leading to reduced reproductive rates and altered sex ratios. Population declines in sensitive species. Climate change impacts, such as ocean acidification and changes in salinity, reduce ecosystem resilience and can even alter the chemical forms and toxicity of pollutants, increasing harm to already vulnerable species such as shellfish and corals [332]. Additionally, extreme weather events driven by climate change, such as heavy rainfall and flooding, facilitate the movement of high amounts of pollutant runoff into aquatic ecosystems, overwhelming natural filtration processes and affecting water quality [336]. As a result, this can increase aquatic animals' exposure to pollutants, further reducing biodiversity and habitat availability, thus compounding the challenges faced by aquatic animals.

6 Future perspectives and research directions

Advancements in detection and monitoring technologies are crucial for understanding the presence and impact of emerging pollutants in aquatic environments, as current techniques may not effectively address all emerging pollutants. Techniques such as high-resolution mass spectrometry and biosensors hold promise for detecting even low concentrations of pollutants, including microplastics and nanoparticles, which could increase real-time monitoring and assessment efforts [337]. Moreover, there is an increasing need to develop biodegradable alternatives to commonly used pollutants, such as certain plastics, pesticides, and pharmaceuticals. Biodegradable materials reduce pollutant accumulation in aquatic ecosystems, thereby minimizing their environmental footprint and providing a sustainable solution to pollution challenges [338]. Future studies should focus on enhancing risk assessment models to address pollution impacts effectively. The development of models that reflect the ecological relevance of bioaccumulation and biomagnification in aquatic food webs would aid in predicting long-term effects on various species and informing regulatory

~~frameworks [339]. Studying the molecular mechanisms of pollutant toxicity will deepen our understanding of how different pollutants interfere with physiological pathways in aquatic organisms [340].~~

~~Additionally, ecological impact studies across diverse aquatic habitats are essential for understanding the cumulative effects of pollutants on biodiversity and ecosystem dynamics [341]. As climate change progresses, future research should also explore how factors such as rising temperatures and extreme weather events influence pollutant transport, transformation, and toxicity [336]. Additionally, strengthening the collaboration between researchers, policymakers, and industries can lead to the sharing of knowledge, resources and strategies to mitigate the spread of emerging pollutants into the aquatic ecosystem, which could further lead to stricter regulations and encourage green manufacturing practices, making pollution control technologies more accessible across the industrial and agricultural sectors.~~

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Effective mitigation of emerging pollutants requires both technological and policy interventions. Advanced wastewater treatments such as activated carbon adsorption, membrane bioreactors, and advanced oxidation processes and regulatory measures promoting

green chemistry, biodegradable alternatives, and extended producer responsibility can collectively reduce pollutant discharge and safeguard aquatic and human health.

7 Conclusion

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~~Emerging pollutants (EPs) in aquatic ecosystems have become an alarming environmental concern, with far-reaching consequences for biodiversity, ecological balance, and potentially aquatic health. This review highlights the sources and pathways by which EPs, such as pharmaceuticals, pesticides, personal care products, and microplastics, enter and persist in aquatic environments. Given that wastewater treatment plants are ineffective at removing these pollutants, their bioaccumulation and biomagnification through the food web in fish, amphibians, molluscs, crustaceans, and other aquatic organisms induce a wide range of toxic effects. Key findings reveal that EP exposure results in physiological stress, endocrine disruption, oxidative damage, immune suppression, reproductive failure, and behavioural changes across species. These effects are particularly concerning given that many of these organisms serve as food sources for higher trophic levels, including top predators and humans. The impacts of EP extend beyond individual species, influencing population dynamics, reducing biodiversity, and compromising ecosystem functionality. The growth, reproduction, and survival rate changes among aquatic species indicate potential long-term disruptions to food webs and nutrient cycles. These cumulative effects underscore the need for stringent regulatory measures and advanced water treatment technologies to effectively manage and mitigate EP contamination. Furthermore, it is imperative to pursue ongoing research to understand the long-term effects of these pollutants on aquatic organisms and develop targeted mitigation strategies. This approach will support efforts to restore and protect aquatic ecosystems and ensure the sustainability of these vital habitats and the safety of aquatic species, including the humans that rely on them.~~

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~~Bardhan A, Abraham TJ, Das R, Patil PK (2022) Biological Responses of Nile tilapia Oreochromis~~ Emerging pollutants (EPs) represent a critical and escalating threat to aquatic ecosystems, with profound implications for biodiversity, food web dynamics, and ecological stability. This review highlights that pharmaceuticals, pesticides, personal care products,

microplastics, nanoparticles, and heavy metals enter aquatic environments through wastewater discharge, agricultural runoff, and industrial effluents, where they persist and bioaccumulate. Key findings indicated that EPs exert their toxicological impacts primarily through oxidative stress, endocrine disruption, immunotoxicity, and genotoxicity, leading to physiological and reproductive impairments across diverse taxa. In fishes, EPs alter antioxidant enzyme systems, disrupt thyroid and reproductive hormones, and induce behavioural and developmental abnormalities. Amphibians show delayed metamorphosis, thyroid dysfunction, and neurotoxicity, while molluscs experience reduced filtration efficiency, DNA damage, and gamete deformities. These mechanistic disruptions collectively translate into population declines, altered trophic interactions, and diminished ecosystem functionality. The synthesis of current evidence reveals major research gaps in understanding the combined and chronic effects of low-level pollutant mixtures, emphasizing the need for long-term, ecologically relevant studies. Strengthening regulatory policies, upgrading wastewater treatment technologies, and developing pollutant-specific mitigation frameworks are essential for minimizing environmental and health risks. Interdisciplinary research integrating molecular, physiological, and ecological endpoints will be crucial for predicting ecosystem resilience under continued contaminant exposure. Addressing these challenges is vital to sustain aquatic biodiversity, ensure food web stability, and protect the health of both aquatic organisms and human populations dependent on these ecosystems.

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~~**Statements & Declarations**~~

Author(s) contributions

All authors contributed significantly to this work and met authorship criteria based on the ICMJE guidelines. S.S.: Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation. R.R.: Visualization, Software, Methodology, Investigation, Formal analysis, Data curation, Writing – review & editing. D.D.: Visualization, Validation, Writing – review & editing. A.G.: Writing – review & editing, Visualization, Validation, Supervision, Software, Project administration, Methodology, Investigation, Conceptualization. No writing assistance was utilized in the production of this manuscript. All the authors have read and approved the final version of the manuscript.

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