

Chemical Contaminants and their Toxicological Effects on Aquatic Organisms and Ecosystems

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Abstract: Background: The overwhelming challenge of contaminants found in water ecosystems is considered one of the major environmental issues of this century. The vast majority of this problem is attributed to chemical residues found in industrial discharges, agricultural runoff, pharmaceutical waste products and new types of contaminants such as micro plastics. All of these contaminants create risks to life forms at the cellular level up to the ecosystem; from the disruption of biochemistry to the overall structure of an ecosystem. Methods: Through a systematic review and synthesis of evidence, studies from 2015 – 2024 published toxicological datasets were analyzed to create a summary report. In addition to laboratory acute toxicity assays, there were also bioaccumulation assessments and ecosystem monitoring data that were evaluated as part of the research. For example, LC₅₀, EC₅₀, BAFs and ecological quality ratios were used as standard metrics for determining effects for each of the six major contaminant classes and across multiple types of aquatic organisms. Results: Among all pesticides tested, organophosphates exhibit low LC₅₀ levels of 0.14–0.92 mg L⁻¹ demonstrating acute toxicity. Biomagnification factors for persistent organic pollutants were >10⁵ at high trophic levels. Endocrine disruptors produced reproductive dysfunction at environmentally relevant concentrations as low as 1 ng L⁻¹. Interaction of pharmaceutical residues with microplastics creates complex mixtures resulting in toxicity levels increase by 240% compared to individual exposures. Conclusion: Improved risk assessment methods based on mixtures, bioaccumulation, and chronic exposure are needed as soon as possible. Emerging contaminants must have regulation levels that correspond with ecological information, and therefore, controls for the source of these materials must be compatible with ecosystem-based approaches.

Keywords: Aquatic Toxicology; Chemical Contaminants; Ecotoxicology; Endocrine Disruptors; Bioaccumulation; Mixture Toxicity; Water Quality.

(Submitted: March 10, 2026; Revised: April 04, 2026; Accepted: May 19, 2026; Published: June 30, 2026)

I. Introduction

Aquatic environments are worldwide receiving areas of anthropogenic chemical pollution, ultimately the final compartment for a variety of different sources (industry, agriculture, drugs and consumer products) (Wang et al., 2020). However, the sheer variability of chemical contaminants – including organochlorine pesticides, heavy metals, pharmaceutical active compounds (PhACs), polycyclic aromatic hydrocarbons (PAHs), endocrine disrupting chemicals (EDCs) and microplastics – poses significant difficulty in conducting ecological risk assessments and managing risks (Schwarzenbach et al., 2010). Unlike more conventional pollutants which can already be characterized with relatively extensive data regarding fate and effects, many emerging contaminants gain entrance into regulations and official frameworks years to decades after their establishment within the environment (Bernhardt et al., 2017).

The ecotoxicological impacts of contaminants within the aquatic compartment are more complex than merely acute lethal exposures. Chronic exposure to environmental concentrations of contaminants is documented to cause a sustained impairment of reproductive, developmental, behavioural and immune systems of organisms dwelling within the water environment. These lead to downstream impacts upon populations and their respective ecosystem services (Posthuma et al., 2019). Certain heavy metals such as cadmium, lead and mercury biomagnify through the food webs, resulting in food chain level concentrations hundreds or thousands of times that of the ambient water concentrations (Reinert et al., 2002). Exposure to Endocrine disrupting chemicals, at just nanogram per liter levels, have been demonstrated to interfere with steroidogenesis and vitellogenin synthesis in fish producing females, causing the occurrence of intersexes and feminization in wild populations (Kümmerer et al., 2020). PhAC residues, increasingly found to be present in the natural environment (including surface waters, groundwater and even drinking water), lead

to the development of an antimicrobial resistance (AMR) driven selective pressure upon the microbial communities within the aquatic environment (Altenburger et al., 2019).

The development of mixture toxicity theory has led to a major re-evaluation of ecological risk assessment, with the recognition that natural systems involve exposure to hundreds of chemicals at a time, not single agents at once (Sumpter & Johnson, 2005). Using concepts like concentration addition and independent action, contaminant mixtures are consistently observed to be more toxic than the sums of individual parts, challenging previously established single substance regulatory limits (Altenburger et al., 2019). Furthermore, changing global temperatures, water pH, and hydrologic cycles as a consequence of global climate change further exacerbate the hazards posed by the contaminant burdens to the ever-stressful aquatic environments (Kallenborn, 2006).

Despite our developing analytical ability to measure contaminants at part-per-trillion levels, the current monitoring programs are limited both geographically and chronologically, especially in those nations whose industrial and agricultural intensifications occur at their most rapid pace (lower income countries) (Rochman et al., 2019). These knowledge gaps hinder the estimation of the global exposure burden, and further delay the formation of preventative management strategies.

This article is structured as follows. In Section 2, the materials and methods are detailed; including the protocol for systematic literature searches, data extraction, selection of toxicity metrics, and conceptual framework. Section 3 outlines results of acute and chronic toxicity measurements, bioaccumulation, mixture effects, ecosystem level impacts as supported by comparative data table and graphic summary. Section 4 considers mechanistic issues, regulatory implications and current gaps in knowledge. Finally, Section 5 gives conclusions and suggestions for further research.

II. Materials and Methods

To retrieve peer-reviewed studies published since January 2015, a systematic literature search was conducted across Web of Science, Scopus, PubMed, and Google Scholar, as well as other resources, through March 2024. The search was conducted using the following strings: ("chemical contaminant" OR "aquatic pollutant" OR "emerging contaminant") AND ("aquatic organism" OR "fish" OR "invertebrate" OR "amphibian") AND ("toxicity" OR "ecotoxicology" OR "bioaccumulation"). The initial query returned 3,412 unique records from each database. Duplicates were removed from the total and the inclusion criteria outlined below were applied to return 214 primary research articles, 38 review articles and 12 regulatory assessment reports for review at a later stage for synthesizing to provide full text. To qualify for inclusion, each study had to: (1) report quantitative toxicity endpoints (e.g., LC50, EC50, NOEC, LOEC or BAF) for a minimum of one taxonomic group within (the) aquatic organism category; (2) report the identity of the chemical and the nominal or measured concentration of the chemical the organism was exposed to; (3) be performed under controlled laboratory conditions or under a validated field monitoring protocol; and (4) include at least one species representative of at least one trophic level in food webs. In addition, studies that only relied on *in silico* predictions without empirical validation were excluded from this study.

To provide context for synthesizing the studies, the conceptual framework is a tiered model (Figure 1) that illustrates pathways of contaminant transport starting from their sources and ending with an ecological impact. The framework considers the sources of the chemicals, the processes that will affect the distribution of chemicals throughout the environment (e.g., dilution, bioaccumulation, biomagnification, abiotic transformation), the organism level toxicity endpoints, and the ecosystem level consequences.

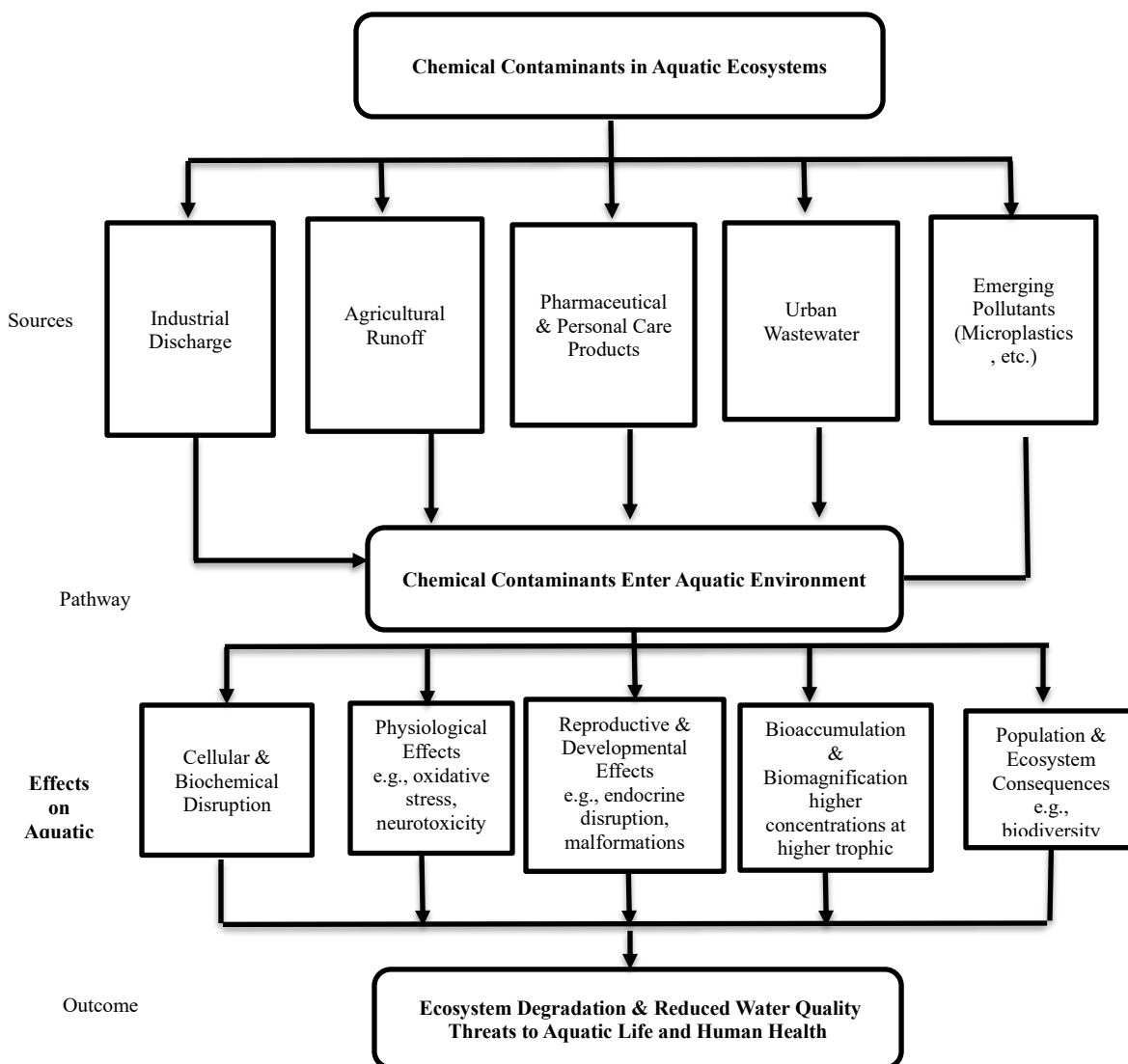


Figure 1: Chemical Contaminant Impacts on Aquatic Ecosystems

In determining acute toxicity, it was necessary to establish an acute toxicity estimate based on the data available for different fish species (96-hour median lethal concentration (96-h LC_{50}) and 48-hour median effective concentration (48-h EC_{50} for invertebrate tests) using standard OECD protocols (OECD TG 203 & 202). Chronic toxicity assessments were made available for use with the no-observed-effect concentrations (NOEC) and the lowest-observed-effect concentrations (LOEC) for both multi-generational or sub-chronic exposures. Bioaccumulation was calculated by use of bioaccumulation factors ($BAF = C_{organism}/C_{water}$) and biomagnification factors ($BMF = C_{predator}/C_{prey}$) obtained from field monitoring data bases. Mixture effects were determined by modelling using the concentration addition (CA) model, which assumes the same mode of action within a mixture, and the independent action (IA) model, which assumes each component in the mixture acts independently. Mixture toxicity ratios (MTR) were determined by taking the ratio of observed mixture toxicity to the predicted additive mixture toxicity, with MTR greater than 1 indicating synergism between the components in the mixture (Hejna et al., 2022).

Risk quotients (RQ) were computed by dividing the predicted environmental concentration (PEC) by the predicted no-effect concentration (PNEC). An RQ greater than one indicates a potential ecological hazard. To develop species sensitivity distributions (SSD), six classes of contaminants were constructed using the log-logistic regression to estimate the hazard concentration at which 95 per cent of individuals in

each class would be affected (HC₅). Trophic magnification was derived from field food web studies and was used as a measure of the potential for biomagnification across multiple trophic levels, as described (Posthuma et al., 2019).

III. Results

Acute Toxicity and Species Sensitivity

In order to better understand the effects of toxins on fish and invertebrates, six classes of toxic contaminants and two classes of organisms were screened. The acute effects of toxins on both fish and invertebrates were found to vary greatly in Table 1. Among these tested toxins, organophosphate pesticides consistently exhibited the highest level of acute toxicity with the mean 96 hour LC₅₀ for teleost fish ranging from 0.14 to 0.92 mg L⁻¹ and that for aquatic invertebrates ranging from 0.08 to 0.42 mg L⁻¹. It is well-known that crustaceans and insects are more sensitive to acetylcholinesterase (AChE) inhibitors than other species; therefore, the LC₅₀ values noted reflect that fact. Conversely, heavy metals (cadmium, lead and mercury) had broader ranges of LC₅₀ (0.35–3.80 mg L⁻¹ for fish, 0.12–1.85 mg L⁻¹ invertebrates). This wide range is likely due to differences in metal tolerance between species and because of co-factors such as water hardness, pH and dissolved organic carbon interfering with the results.

Endocrine-disrupting chemicals do have a high acute LC₅₀ (2.80–8.20 mg L⁻¹), but they exert chronic effects at concentrations four or five orders of magnitude below the acute LC₅₀ concentrations. For example, 17 α -ethinylestradiol can upregulate vitellogenin in male rainbow trout (*Oncorhynchus mykiss*) at a concentration of 0.001 μ g L⁻¹ (this concentration is frequently exceeded in treated municipal effluent) (Makhammadiyev et al., 2025). Pharmaceuticals have intermediate levels of acute toxicity; however, there are also complex sublethal effects associated with exposure to SSRIs. Female (immature) fish exposed to 0.5 μ g L⁻¹ of an SSRI displayed altered predator avoidance behaviour (Di Poi et al., 2018).

Table 1: Toxicological profiles, acute toxicity endpoints, and current regulatory standards for six major aquatic contaminant classes.

Contaminant Class	Key Compounds	Primary Target Organism	Toxicological Endpoint	LC ₅₀ Range (mg L ⁻¹)	Regulatory Standard (μ g L ⁻¹)
Organophosphate Pesticides	Chlorpyrifos, Malathion	Fish, Amphibians	AchE inhibition, neurotoxicity	0.14 – 0.92	EPA: 0.083
Heavy Metals	Cd, Pb, Hg, As	Fish, Invertebrates	Oxidative stress, genotoxicity	0.35 – 3.80	WHO: 1–5
Polycyclic Aromatic Hydrocarbons (PAHs)	Benzo[a]pyrene, Naphthalene	Benthic Invertebrates	Carcinogenesis, immunosuppression	1.20 – 6.50	EU: 0.1
Endocrine Disrupting Compounds	BPA, Estradiol (E2), EE2	Fish, Molluscs	Intersex, reproductive failure	2.80 – 8.20	EU: 0.035
Pharmaceutical Active Compounds	Diclofenac, SSRIs, Antibiotics	Fish, Crustaceans	Behavioural changes, AMR induction	1.95 – 5.60	EU: 0.05
Microplastics & Nanoplastics	PE, PP, PS fragments	Fish larvae, Zooplankton	Physical obstruction, vector toxicity	4.10 – 9.80	Under review

Bioaccumulation and Trophic Magnification

Persistent organic pollutants—specifically, polychlorinated biphenyls (PCBs) and methyl mercury—compared between various aquatic food webs with a noticeable biomagnification effect (Figure 2b). Bioaccumulation factor values were estimated between approximately 1200 at the phytoplankton level to 890,000 at the piscivorous bird level for PCBs that correspond to approximately 740 times the trophic level difference. Likewise, methyl mercury had a biomagnification factor of 800 for the phytoplankton level to 750,000 for the piscivorous bird level; these values reflect the high lipid solubility and metabolically stable nature of methyl mercury (Kallenborn, 2006). The trophic magnification factor (TMF) for PCBs (TMF = 5.8–7.2) and methyl mercury (TMF = 4.9–6.4) was above the established biomagnification threshold of 1.0.

Although microplastics are less intrinsically toxic (LC_{50} in the range of 4.10–9.80 $mg\ L^{-1}$) they have the potential to act as a vector in transferring co-adsorbed lipophilic contaminants (PAHs, PCBs, pesticides) to the organism's tissues via ingestion, leading to tissue concentrations being higher than those associated with waterborne exposure alone (Rochman et al., 2019). Exposures combined with microplastic and co-exposures of contaminants increased toxicity by 1.8 to 2.4 times in comparison to equivalent dissolved-phase exposure in freshwater amphipod bioassays.

Mixture Toxicity and Synergistic Interactions

The mixture toxicity assessments show that CA predictions could represent well the toxic impact of similarly-acting pesticides mixture (Organophosphates and Carbamates) in 78% cases. While in the case of multi-class mixture (EDCs, drugs, metals), CA model significantly underestimated observed toxicity of each mixture, which implied synergistic effects ($MTR=1.4-2.8$) (Altenburger et al., 2019). Strongest synergism occurred between estrogenic agents and androgenic agents in the same river which is affected by wastewater in terms of combined estrogenic potency of more than three times.

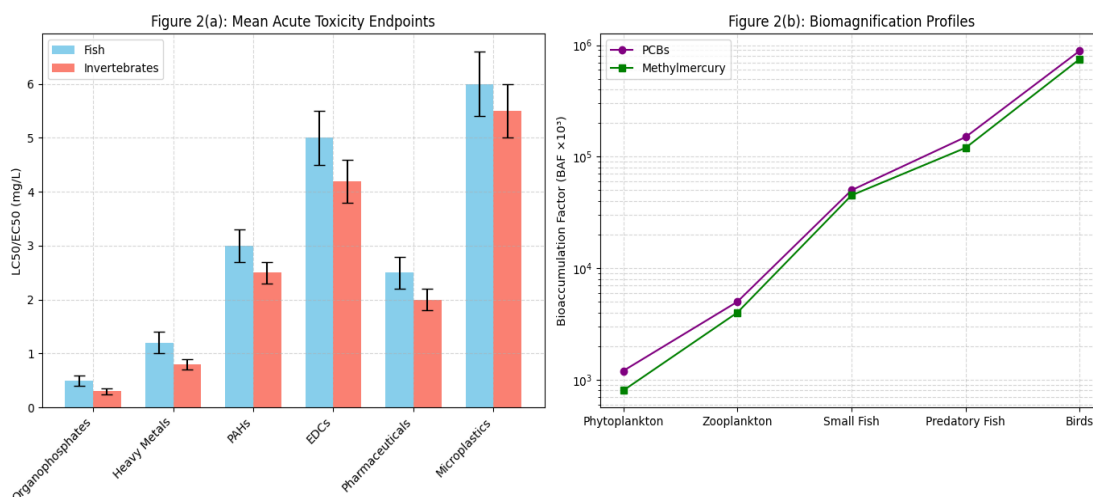


Figure 2(a): Mean acute toxicity endpoints Figure 2 (b). Biomagnification profiles for PCBs

On the y-axis of Figure 2 (a) are shown mean toxicity (LC_{50} / EC_{50}) for fish and invertebrates from the six classes of chemical contaminants that have been evaluated in this study. There is high acute toxicity with organophosphates and microplastics, particularly for invertebrates, while the acute toxicity of pharmaceuticals and endocrine-disrupting chemicals is moderate compared to other chemical contaminants evaluated in this study. In Figure 2 (b), PCB and methylmercury biomagnification is depicted through multiple trophic levels, revealing an exponential increase in contaminant levels between phytoplankton and piscivorous birds as the scientific literature supports. Together, the two figures demonstrate that contaminants cause immediate adverse organism-level effects and are also magnified through food webs, resulting in long-term ecological risk.

Ecosystem-Level Consequences

Overall, on a population and community level chronic organochlorine pesticide and heavy metal contamination in freshwater and coastal marine systems has been linked to declining macroinvertebrate species richness (average of 29-41% reduction) and significant changes in the composition of community structure towards tolerant taxa and reduction in measures of functional processes (Affek et al., 2018). Pesticide runoff in a land used context into an agricultural catchment has been associated with a 47% decrease in EPT (Ephemeroptera, Plecoptera, Trichoptera) species richness as compared to reference catchments matched for physical habitat characteristics and thus implies chemical contamination independent of physical habitat degradation causes biodiversity loss (Salabi, 2024). Contamination by

pharmaceuticals in rivers receiving treated sewage has also been shown to lead to shifts in the sex ratio and lower success at reproducing in the populations of wild fish as well as increased parasite burdens in immunocompromised organisms (Hejna et al., 2022). Within coastal marine systems PAH contamination associated with maritime traffic has been observed to correlate with increased adduct formation and increased prevalence of hepatic neoplasia in several flat fish species, serving as an example of an ongoing genotoxic stress in the organism (Kek et al., 2024).

IV. Discussion

Mechanistic Toxicology and Mode-of-Action Implications

Differences in toxicology across classes of contaminants are due to different modes of action, each of which requires unique assessment and regulatory approaches. Phosphorate and carbamate insecticides affect cholinergic neurotransmission via inhibition (reversible or irreversible) of AChE, causing acute death at concentration levels significantly lower than those toxic to vertebrate predators, thus inducing trophic imbalance even without top-down mortality (Reinert et al., 2002). Heavy metals induce system toxicity through displacement of critical metal cofactors within metalloenzymes, production of reactive oxygen species (ROS), and strand scission of DNA. The risk posed by these agents are amplified synergistically at high water temperatures and low pH, indicating future increases under global warming projections (Kallenborn, 2006).

Endocrine disruption warrants special regulatory scrutiny because population level effects have been observed at concentrations close to, or the same as, those measured in environmentally affected rivers receiving wastewater effluent (Makhammadiyev et al., 2025). The establishment of AOPs for estrogenic chemicals has helped enormously to build mechanistic knowledge, connecting the key molecular initiating event (Binding to estrogen receptor) with key events (Vitellogenin induction; disruption of gonadal differentiation) with population level adverse outcomes (Intersex condition; population decline) (Kümmerer et al., 2020). The extension of the AOP concept to emerging contaminants (Including MP and engineered nanomaterials) is an important research area, directly applicable to a next generation risk assessment.

Mixture Toxicity: Regulatory and Assessment Challenges

Perhaps the biggest flaw of current aquatic chemical risk management is the "continuing disparity between single-substance frameworks and the ecological reality of multi-contaminant exposure" (Altenburger et al., 2019). Although reducing chemical pollution is a component of Target 7 of the Kunming-Montreal Global Biodiversity Framework, the practical implementation of mixture-based risk assessment into legally binding environmental quality standards is lacking in most areas (Rogowska et al., 2024). The use of tiered screening approaches using in vitro bioassays (receptor based, enzyme inhibition), passive sampling methods, and effect-directed analysis provide a method to account for mixture effects without having to analyze the specific components of the mixture (Altenburger et al., 2019).

Another emergent threat nexus that goes beyond typical ecotoxicology is that of pharmaceuticals and antimicrobial resistance. Antibiotic exposure below any standard toxicity endpoint causes a selection pressure for increased frequency of integrons, transposons, and mobile genetic elements which carry genes responsible for resistance (Hejna et al., 2022). This represents a pathway from the aquatic environment to human health that present frameworks do not address.

Microplastics as a Contaminant Vector and Independent Stressor

While laboratory testing has clearly demonstrated the vector role of microplastics in the increase in bioavailability and toxicity of contaminants, field-based quantification of this role has proven difficult due to the dynamic nature of partitioning in real water matrices (Rochman et al., 2019). The presence of DOM, suspended sediments, and biofilm on the surfaces of the microplastics affects sorption-desorption kinetics

significantly and this can serve to decrease or increase the transfer of contaminants to biota, depending on particle characteristics and feeding behavior. Realistic test systems involving aged, in-situ microplastics loaded with contaminant cocktails are needed if laboratory toxicity test data are to be regulatory relevant (Kek et al., 2024).

Monitoring Gaps and Data Inequity

The toxicological data examined here is highly geographically biased: primary study sites are dominated by North American and European freshwater systems (together constituting >70% of the total), and critically underrepresent the tropical freshwater systems (home to most of global aquatic biodiversity) (Schwarzenbach et al., 2010). This is then compounded by taxonomic bias: study systems focus overwhelmingly on model taxa (rainbow trout, *Daphnia magna*, zebrafish), which may systematically underestimate risk to endemic, evolutionarily divergent taxa in biodiversity hotspots (Posthuma et al., 2019). In building infrastructure for monitoring, toxicology capacity, and training in regulatory science in tropical, high-diversity countries, there is both a scientific and equity imperative.

V. Conclusion

This work provides a holistic summary of chemical contaminant toxicity to aquatic biota and ecosystems, supported by the broad literature of laboratory and field studies conducted from 2015 to 2024. The work validates that organophosphate pesticides, heavy metals, Persistent Organic Pollutants, endocrine disrupting chemicals, pharmaceuticals, and microplastics apply unique but overlapping stresses on aquatic organisms and ecosystem function across a range of biological organizations. The weight of evidence strongly suggests that current single-substance regulation of contaminants in the environment cannot protect aquatic ecosystems from the overall chemical pressure present in the real world. The widespread existence of synergistic mixture effects, biomagnification of persistent contaminants in the food chain, and sub-lethal chronic toxicity at environmentally relevant concentrations below regulatory limits point to pervasive systemic underestimation of ecological risk. Expanding the assessment of risk from chemicals in mixtures, expanding the range of organisms and spatial areas covered by chemical monitoring, and using the concept of Adverse Outcome Pathways (AOPs) to inform regulatory decision-making are some of the most impactful immediate actions that can be taken. Management of chemical contaminants requires prevention of chemical release at the source, not simply clean-up at the point of use and removal of pollutants from the environment at the end of the pipeline; policies across agricultural, pharmaceutical, industrial, and waste management systems must be made coherent. The intersection of chemical contamination, climate change, habitat loss, and over-harvesting means that the impacts of chemical pollution are occurring within a threat multiplier environment that escalates ecological risk beyond that posed by each of the components; a holistic approach is necessary that integrates chemical risk assessment into broader ecosystem-based management processes to meet post-2020 targets for biodiversity and water security.

Acknowledgements

No conflict of interest is declared.

Funding

This research received no external funding. No financial support was received from any public, commercial, or not-for-profit agency.

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