CHAPTER 10

Epigenetic And Transgenerational Toxicity In Freshwater Fishes Exposed To Emerging Pollutants And Its Effects On Aquatic Life

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Aquatic environments are drastically burdened with a wide range of pollutants and carcinogens originating from massive industrialization, agriculture, domestic waste, and pharmaceutical consumption, radioactive waste. These contaminants produce complex physiological and biochemical load in freshwater organisms bodies, particularly fishes, which serve as key ecological indicators of pollution and other sever changes of the ocean. Recent advances in molecular biology have revealed that pollutants can induce epigenetic alterations other genetic leathal mutations in the organisms—heritable changes in gene expression DNA sequence. Epigenetic toxicity is a growing research frontier because environmental chemicals can alter DNA methylation and perform the DNA Acylation on specific DNA sequences , histone structure, and regulatory RNA pathways, ultimately affecting reproduction, survival, and adaptation. Epigenetics is the investigation of meiotically and/or mitotically heritable fluctuations in gene function that cannot be clarified by modifications in the DNA sequence (Waddington, 1957). Epigenetic alterations act as molecular switches, influencing gene expression and facilitating

phenotypic plasticity in response to environmental variation (Makvandi-Nejad and Moghadam, 2023). More concerning is the evidence of transgenerational toxicity, where pollutant-induced epimutations are passed to offspring that were never directly exposed. Understanding these mechanisms is critical for predicting long-term ecological risks and developing sustainable conservation strategies.

2. EMERGING POLLUTANTS IN FRESHWATER ECOSYSTEMS

PFAS as an Emerging Pollutant in Freshwater Ecosystems (Rivers and Lakes)

Per- and polyfluoroalkyl substances (PFAS) are a large class of synthetic chemicals widely used for their oil- and water-repellent properties (e.g., in surface coatings, textiles, food-contact materials, and certain industrial processes). PFAS are increasingly treated as *emerging pollutants* in freshwater systems because many compounds are highly persistent, can be mobile in water, and some are bioaccumulative and toxic, creating long-term exposure risks for aquatic food webs and downstream human uses of freshwater. (Jennifer L. Guelfo2021)

1) Major sources and pathways into freshwater

Freshwater PFAS contamination typically arises from a combination of point sources and diffuse sources:

- **Wastewater treatment plant (WWTP) effluent**: PFAS enter WWTPs via municipal wastewater, industrial inputs, and landfill leachate; conventional treatment often does not reliably destroy PFAS, so effluent can remain a continuous loading source to rivers. (Donald M. Reeves2024)

- **Industrial releases and legacy sites:** Manufacturing and use sites can release PFAS directly to surface waters or indirectly through groundwater that later discharges to rivers/lakes.

- **Urban runoff and atmospheric deposition:** PFAS can be transported via stormwater and deposition, especially where PFAS-containing products are widely used. (Jennifer L. Guelfo2021)

2.1 Microplastics and Nanoplastics

Microplastics

Microplastics (MPs) and nanoplastics (NPs) are now widely treated as emerging pollutants because they are pervasive across environmental compartments and can interact with organisms through both physical and chemical pathways. MPs are commonly discussed as plastic particles ≤ 5 mm, originating either as primary particles (e.g., pellets, microbeads, industrial abrasives) or as secondary particles produced by fragmentation of larger plastic debris. Current syntheses emphasize that definitions are still being refined across disciplines, but the "≤5 mm" convention remains a dominant operational standard in research and monitoring.(Richard C. Thompson 2024). In freshwater ecosystems, MPs enter rivers, lakes, and reservoirs mainly via wastewater effluent, stormwater/urban runoff, mismanaged waste, agricultural inputs (including sludge), and atmospheric deposition. Reviews of freshwater systems report MP occurrence in water columns, sediments, and biota, with impacts ranging from reduced feeding efficiency and energetic stress to inflammation, oxidative stress, and altered behavior in aquatic organisms—often modulated by polymer type, particle size/shape, and co-contaminant loading.(K. Bexeitova 2024)

Nanoplastics (NPs)

Nanoplastics (typically discussed at sub-micrometre to nanoscale ranges) raise additional concern because they have higher surface area-to-volume ratios and can exhibit greater mobility and bioavailability, including potential for cellular uptake. A key environmental control on NP fate is aggregation/heteroaggregation with natural colloids and minerals, which can strongly influence whether NPs remain suspended, settle, or become incorporated into biofilms and sediments—meaning toxicity and exposure can be highly context-dependent.(Alice Pradel 2023)

3. Epigenetic Mechanisms in Fish

Epigenetics refers to heritable and/or reversible changes in gene regulation that occur without altering the DNA sequence. In fish, epigenetic regulation is central to embryonic development, phenotypic plasticity (responses to environment), stress physiology, and pollutant/toxicant responses. Core mechanisms include DNA methylation, histone modifications and chromatin remodeling, and non-coding RNAs.(Mackenzie Gavery 2017)

1) DNA methylation: writing and reading the "methylation code"

DNA methylation in vertebrates typically involves addition of a methyl group to cytosine (mostly at CpG sites), often linked to transcriptional repression when present at promoters, and broader roles in genome stability, transposon silencing, and developmental gene regulation.

- **Enzymes ("writers")**: Fish possess conserved DNA methyltransferases (DNMTs). DNMT1 primarily maintains methylation after replication, while DNMT3-family enzymes perform de novo methylation during early development and differentiation. Zebrafish studies show dynamic DNMT expression patterns across developmental stages, consistent with methylation reprogramming windows.(Mary G Goll 2011 & Pawat Seritrakul 2013)

- **Functional significance**: During fish embryogenesis, methylation patterns help coordinate cell fate decisions and tissue-specific transcriptional programs. This system is broadly conserved with other vertebrates, making zebrafish a widely used model for mechanistic work.(Mary G Goll 2011)

- **Environmental sensitivity**: Early-life stages can be particularly sensitive because methylation patterns are being established. For example, combined stressors (e.g., temperature and copper) can alter DNMT expression in zebrafish embryos, suggesting disruption of methylation programming during critical windows.(Jennifer Dorts2016)

2) Histone modifications and chromatin remodeling: controlling DNA accessibility

Fish chromatin regulation relies on post-translational modifications of histone tails (e.g., acetylation, methylation, phosphorylation), which influence how tightly DNA is packaged and therefore gene accessibility.

- Histone acetylation (often associated with active transcription) is regulated by HATs/HDACs; histone methylation can activate or repress transcription depending on the residue and degree of methylation (e.g., H3K4me3 vs H3K27me3 in many vertebrates).
- Teleost epigenetic toolkits for histone modification are evolutionarily conserved, and these pathways interact with DNA methylation to fine-tune transcription in development and physiology.(Carol Best 2018)

3) Non-coding RNAs: post-transcriptional regulation and epigenetic crosstalk

Fish express diverse non-coding RNAs, especially microRNAs (miRNAs), that regulate gene expression post-transcriptionally by binding target mRNAs and affecting translation or stability.

- miRNAs are strongly implicated in stress responses (thermal stress, hypoxia, pollutants), immunity, and development in teleosts. Reviews summarizing teleost miRNA profiling show that miRNAs can act as molecular switches linking environmental stimuli to coordinated gene-expression programs
- ncRNA pathways also interact with chromatin modifiers and DNA methylation machinery, creating multi-layer regulatory networks rather than isolated mechanisms (Guoqing Lu 2025)

4) Epigenetic reprogramming and (trans)generational inheritance in fish

A major topic in fish epigenetics is when and how epigenetic marks are reset during development, and the implications for inheritance.

- Recent synthesis on teleost DNA methylation reprogramming highlights how early developmental stages involve extensive remodeling of methylation landscapes, but patterns and timing can differ across vertebrates and fish lineages.
- There is growing evidence that environmentally induced epigenetic marks can be stable across generations in some fish contexts. For example, work in sticklebacks showed stable inheritance of DNA methylation differences after removal of an environmental stressor (supporting germline transmission of methylation states)
- Pollutant and stress exposure studies increasingly integrate methylation, gene expression, and phenotype across generations to assess whether epigenetic variation contributes to rapid adaptation or persistent health effects.(Fabien Pierron 2022)

4. Epigenetic Toxicity Induced by Emerging Pollutants

Epigenetic toxicity (epitoxicity/epigenotoxicity) refers to adverse biological effects driven by pollutant-induced changes in epigenetic regulation—primarily DNA methylation, histone modifications, chromatin organization, and non-coding RNAs (ncRNAs, including miRNAs and lncRNAs)—that alter gene expression without changing the DNA sequence. These

epigenetic perturbations can be persistent, may occur at low or developmentally relevant doses, and can contribute to disease susceptibility and, in some cases, transgenerational effects. Reviews covering multiple pollutant classes highlight that epigenetic pathways often intersect with oxidative stress, endocrine disruption, inflammation, mitochondrial dysfunction, and impaired DNA repair, creating a mechanistic bridge from exposure to phenotype (Sandeep R Reddy 2025)

4.1 Disruption of DNA Methylation

DNA methylation (5-mC) and hydroxymethylation (5-hmC)

- Pollutants may cause global hypomethylation (genomic instability) and/or locus-specific hyper/hypomethylation at promoters/enhancers of genes involved in development, metabolism, immunity, reproduction, and neurobiology. PFAS literature increasingly discusses both methylation and hydroxymethylation as exposure-responsive marks. (Rebekah L. Petroff 2023)

4.2 Histone Modification Changes

Changes in acetylation/methylation states (e.g., altered HDAC/HAT balance) can shift chromatin accessibility, amplifying or silencing transcriptional programs. Reviews on endocrine disruptors such as BPA summarize consistent evidence for histone and DNA methylation perturbation. (Federica Cariati2023)

6. Biological and Ecological Consequences

A. Reproductive impairment and developmental failure (core population-level driver)

Many emerging pollutants act as endocrine disruptors and/or developmental toxicants, and epigenetic reprogramming is a plausible mechanism for **delayed** reproductive phenotypes that appear in descendants.

- **Reduced fertility and embryo viability across generations:** Early developmental exposure to bisphenol A (BPA) or 17α-ethinylestradiol (EE2) in medaka produced no obvious abnormalities in directly exposed generations but later caused reduced fertilization (F2) and reduced embryo survival (F3)—a hallmark of transgenerational outcomes with major demographic implications.

- Mechanistically, these outcomes are consistent with epigenetic disruption of the hypothalamic–pituitary–gonadal axis, gonadal differentiation pathways, and gametogenesis programs. (Medaka work and related endocrine-disruptor literature emphasize these pathways.)

Biological significance: Even modest reductions in fertilization rate or early survival can sharply reduce recruitment, especially in short-lived or seasonally breeding freshwater species.

B. Metabolic dysregulation, growth effects, and reduced stress tolerance

Epigenetic marks frequently sit upstream of endocrine and metabolic regulators; therefore, pollutant-induced epigenetic drift can alter growth, energy allocation, and resilience.

- In fish and other models, environmentally induced methylation changes often map to genes involved in metabolism and detoxification, shaping physiological performance under stress. A field-to-lab study in *Poecilia mexicana* showed >80% overlap of differentially methylated regions across generations even after the stressor was removed, illustrating how stable methylation states can become biologically entrenched.

- For PFAS, mechanistic syntheses report disruption of DNMT/TET balance, chromatin remodeling, oxidative stress, and persistent gene-regulatory changes that plausibly affect growth and metabolic homeostasis (including in offspring).

Biological significance: Shifts in energy budgeting can reduce growth rate, delay maturation, and lower competitive ability—indirectly depressing fitness even when mortality is not immediately elevated.

C. Neurobehavioral and sensory impacts that alter survival and reproduction

A frequent "silent" consequence of epigenetic toxicity is altered neurodevelopment and behavior (e.g., anxiety-like behavior, locomotion, feeding, predator avoidance), which can be carried into subsequent generations if germline programming is affected.

- PFAS reviews describe convergent evidence for epigenetic and transcriptomic disruption involving neurodevelopmental signaling, inflammation, and oxidative stress pathways, and discuss multigenerational persistence in some model systems.

- Micro- and nanoplastics literature increasingly points to pollutant-associated gene expression and epigenetic modulation in exposed organisms, with concern that molecular reprogramming could translate into persistent organism-level effects, including behavior and immune function.

Biological significance: Behavioral traits are tightly linked to ecological performance—small changes can increase predation, reduce foraging efficiency, and impair mate finding/spawning success.

D. Immunotoxicity and disease susceptibility

Epigenetic regulation is central to immune cell differentiation and inflammatory signaling. Emerging pollutants that induce oxidative stress and disrupt chromatin states can therefore increase infection risk and inflammatory pathology.

- Reviews on plastic particles and PFAS emphasize oxidative stress, inflammatory signaling changes, and downstream molecular dysregulation that aligns with altered immune competence.

Biological significance: Increased disease susceptibility can create density-dependent feedbacks, amplifying population declines under environmental stress (temperature, hypoxia, pathogens).

3) Ecological consequences: from individuals to populations and communities

A. Population viability and recruitment bottlenecks

The clearest ecological pathway is: epigenetic disruption → impaired reproduction/early survival → reduced recruitment → population decline. The medaka BPA/EE2 transgenerational reductions in fertilization and embryo survival provide a direct example of how early-life exposure can manifest as delayed population-level harm.

What this looks like in the wild:

- declining year-class strength,
- skewed sex ratios in endocrine-disruption contexts,
- increased frequency of recruitment failure during drought/heat waves (because stressed populations have less buffering capacity).

B. "Hidden carryover" effects and ecological time lags

Epigenetic inheritance creates time lags: ecosystems may show continued impairment even after pollutant inputs are reduced, because descendants carry altered regulatory states. Stable cross-generation methylation signals observed in fish populations after stressor removal support the plausibility of such lags.

Ecological significance: Standard monitoring that focuses only on immediate toxicity can underestimate risk.

C. Trophic transfer, food-web structure, and biodiversity impacts

When fish exhibit reduced growth, altered behavior, or lower fecundity, knock-on effects propagate:

- **Predator–prey dynamics:** altered prey capture and predator avoidance can restructure size classes and species interactions.
- **Food-web efficiency:** reduced fish recruitment can shift energy flow toward invertebrates or tolerant species, decreasing functional diversity.
- **Cross-taxa implications:** emerging pollutant classes (PFAS, plastics) affect multiple taxa; epigenetic mechanisms are broadly conserved, so similar regulatory disruption is plausible in aquatic invertebrates and amphibians, potentially compounding community-level instability.

D. Adaptation versus maladaptation: an important ecological nuance

Not all inherited epigenetic change is necessarily harmful; in some contexts it may facilitate rapid phenotypic adjustment to stress. The *Poecilia mexicana* work shows that stable inherited methylation differences can be associated with persistence in extreme environments. However, pollutant-driven epigenetic changes are more likely to be non-

adaptive (or maladaptive) because they can disrupt conserved endocrine and developmental programs. The ecological outcome depends on whether the inherited change improves performance under the prevailing environment or imposes fitness costs.

Consequences for broader aquatic life (beyond fish)

Freshwater fish are often sentinels; epigenetic/transgenerational toxicity can affect aquatic life more broadly through:

1. **Shared exposure pathways:** dissolved contaminants, sediments, diet-associated plastics, and maternal transfer.
2. **Shared molecular machinery:** methylation systems, histone modifiers, and small RNAs occur across aquatic taxa; thus, endocrine disruptors and persistent pollutants can plausibly alter development, immunity, and reproduction in invertebrates and amphibians as well.
3. **Ecosystem service impacts:** biodiversity loss, reduced fisheries productivity, and impaired nutrient cycling if community composition shifts toward tolerant but less functionally diverse assemblages.

Practical implications: biomarkers and ecological risk assessment

Because epigenetic changes can precede overt pathology, they can be used as early warning indicators in environmental monitoring:

- DNA methylation signatures (global and locus-specific),
- histone mark profiles,
- miRNA panels linked to endocrine, immune, and neurodevelopmental pathways,
- paired with life-history endpoints (fertility, hatch success, growth, behavior).

Impaired Reproduction

- Emerging pollutants such as endocrine-disrupting chemicals (EDCs), pharmaceuticals, pesticides, and microplastics can alter epigenetic regulation of genes involved in gametogenesis and hormone signaling in freshwater fishes. DNA

methylation changes in reproductive genes (e.g., *cyp19a*, *vtg*, *ar*) lead to reduced fecundity, abnormal sex ratios, delayed maturation, and decreased spawning success. These effects may persist across generations through epigenetic inheritance, even after pollutant removal.

Developmental Defects

- Exposure to emerging pollutants during embryonic and larval stages induces epigenetic modifications affecting genes regulating cell differentiation, organogenesis, and growth. Histone modifications and altered microRNA expression can result in skeletal deformities, cardiac abnormalities, impaired yolk utilization, and reduced survival rates. Transgenerational exposure amplifies these defects, compromising early life-stage fitness.

Behavioral Impairments

- Neuroactive pollutants such as antidepressants, pesticides, and heavy metals cause epigenetic disruption of neural development and neurotransmitter pathways. Altered DNA methylation of neurobehavioral genes leads to abnormal swimming, reduced predator avoidance, impaired feeding, and altered social behavior. Such behavioral changes decrease survival and reproductive efficiency in natural populations.

Immune Dysfunction

- Emerging contaminants can epigenetically suppress immune-related genes, including cytokines and antioxidant defense enzymes. Hypermethylation or histone alterations reduce immune responsiveness, making fishes more susceptible to pathogens and diseases. Transgenerational immune suppression can increase disease prevalence and mortality in freshwater fish populations.

Population-Level Decline

- Cumulative epigenetic effects on reproduction, development, behavior, and immunity translate into reduced population recruitment and survival. Transgenerational toxicity magnifies vulnerability across successive generations, leading to population

instability, loss of genetic resilience, and eventual population decline in contaminated freshwater ecosystems.

Effects on Marine Life and Connected Ecosystems

- Freshwater systems are ecologically linked to marine environments through rivers and estuaries. Epigenetically compromised freshwater fishes can affect migratory species and nutrient cycling, indirectly influencing marine biodiversity. Pollutant-induced epigenetic alterations may propagate through connected aquatic ecosystems, impacting marine fish health and productivity.

Transport of Pollutants to Marine Environments

- Emerging pollutants from freshwater sources are transported to coastal and marine systems via river discharge, runoff, and sediment flow. Persistent pollutants such as microplastics, PFAS, and pharmaceuticals accumulate in estuaries and oceans, extending epigenetic toxicity beyond freshwater habitats and exposing marine organisms to similar molecular disruptions.

Epigenetic Changes in Marine Fishes

- Marine fishes exposed to freshwater-derived pollutants exhibit epigenetic alterations in stress response, metabolism, and reproductive genes. Changes in DNA methylation and non-coding RNA expression affect growth, fertility, and adaptive capacity, potentially leading to transgenerational impacts similar to those observed in freshwater species.

Impact on Marine Food Webs

- Epigenetically altered fishes may exhibit reduced growth, altered behavior, and decreased survival, disrupting trophic interactions. Bioaccumulation of pollutants and inherited epigenetic effects can impair predator–prey dynamics, reduce fisheries

productivity, and destabilize marine food webs, ultimately affecting ecosystem services and human food security.

9. Mitigation and Future Directions

Mitigating epigenetic and transgenerational toxicity in freshwater fishes exposed to emerging pollutants requires an integrated approach combining pollution control, ecological management, and advanced research strategies. At the mitigation level, strengthening wastewater treatment technologies is critical to reduce the discharge of pharmaceuticals, personal care products, microplastics, PFAS, and endocrine-disrupting chemicals into freshwater ecosystems. Implementation of advanced treatment methods such as membrane filtration, activated carbon adsorption, and advanced oxidation processes can significantly lower pollutant loads. In parallel, stricter regulatory frameworks and environmental quality standards should incorporate epigenetic endpoints, not just acute toxicity measures, to better assess long-term ecological risks.

Habitat restoration and conservation strategies also play a vital role in mitigation. Protecting spawning grounds, maintaining environmental flows, and reducing agricultural runoff can help minimize chronic exposure of fish populations to contaminants. Bioremediation approaches using microbes and aquatic plants capable of degrading or sequestering pollutants offer eco-friendly solutions to reduce contaminant bioavailability. Additionally, promoting sustainable agricultural and industrial practices can limit the introduction of novel pollutants into freshwater systems

9.1 Pollution Reduction Strategies

Effective reduction of emerging pollutants is essential to prevent epigenetic and transgenerational toxicity in freshwater fishes. At the source level, improving industrial effluent control through strict enforcement of discharge limits and mandatory pre-treatment of wastewater can significantly reduce the release of toxic chemicals. Adoption of green chemistry principles in pharmaceutical and chemical industries can minimize the production of persistent and bioactive compounds that induce epigenetic alterations. Upgrading municipal wastewater treatment plants with advanced technologies such as activated carbon filtration, ozonation, ultraviolet oxidation, and membrane bioreactors is critical for removing

pharmaceuticals, endocrine-disrupting chemicals, microplastics, and PFAS. Establishing drug take-back programs and proper disposal guidelines can further reduce pharmaceutical contamination of aquatic systems.

9.2 Bioremediation

Bioremediation is an eco-friendly and sustainable pollution mitigation strategy that uses living organisms to detoxify, degrade, or remove emerging pollutants from freshwater ecosystems. In the context of epigenetic and transgenerational toxicity in freshwater fishes, bioremediation plays a crucial role in reducing chronic exposure to contaminants that induce heritable molecular changes.

Microbial bioremediation involves bacteria and fungi capable of degrading pharmaceuticals, pesticides, hydrocarbons, and endocrine-disrupting chemicals into less toxic forms. Species such as *Pseudomonas*, *Bacillus*, and *Phanerochaete* possess enzymatic systems that break down complex organic pollutants, thereby reducing their epigenetic impact on aquatic organisms.

Phytoremediation, using aquatic plants like *Eichhornia crassipes*, *Lemna minor*, and *Typha* species, helps remove heavy metals, nutrients, and organic pollutants through uptake, accumulation, and transformation. By lowering contaminant bioavailability, phytoremediation indirectly prevents epigenetic alterations in fishes associated with oxidative stress and endocrine disruption.

Algal bioremediation is particularly effective in nutrient-rich waters, where microalgae can absorb pharmaceuticals, metals, and microplastics while improving water quality. Biofilm-based remediation systems further enhance pollutant removal efficiency.

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