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Evaluating the Impacts of Xenobiotic- induced Genotoxicity on Fish Health and Population Dynamics: A Review of Xenobiotic Stressors in Aquatic Ecosystems

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Abstract- Aquatic ecosystems are increasingly threatened by xenobiotic pollutants released through agricultural runoff, industrial discharge, domestic wastewater, pharmaceutical residues and other human-related activities. These contaminants may persist in water bodies, accumulate in fish tissues and interfere with normal biological processes, including the integrity of genetic material. Fish are important indicators of aquatic ecosystem health because they are continuously exposed to contaminants through the gills, skin, sediments and food chain, making them useful organisms for assessing environmental pollution. This review examines the impacts of xenobiotic-induced genotoxicity on fish health and population dynamics, with emphasis on DNA damage, biomarkers of exposure, reproductive impairment, growth reduction, survival decline and ecosystem consequences. The article is based on a secondary-sourced narrative review of relevant literature on aquatic pollution, genotoxic mechanisms, fish toxicology, biomarkers and environmental management. Findings from reviewed studies indicate that xenobiotics such as pesticides, heavy metals, hydrocarbons, pharmaceuticals and endocrine-disrupting chemicals can cause DNA strand breaks, micronuclei formation, chromosomal abnormalities, oxidative stress and impaired DNA repair in fish. These effects may weaken immunity, reduce growth, affect reproductive performance, alter sex ratios, lower recruitment and contribute to population decline. The review concludes that genotoxicity should be treated not only as a cellular problem but also as an ecological and public health concern. Regular biomarker-based monitoring, improved wastewater treatment, stricter pollution control and effective enforcement of environmental regulations are recommended to protect fish populations, aquatic ecosystems and human health.

Keywords: Genotoxicity, Fish, Xenobiotics, Population Dynamics, Aquatic Pollution, Biomarkers

I. INTRODUCTION

Aquatic pollution has become a persistent ecological concern because rivers, lakes, streams, estuaries and coastal waters increasingly receive complex mixtures of agricultural, industrial, domestic, pharmaceutical and petroleum-derived contaminants. Wilkinson et al. (2022) demonstrate the global scale of this concern by reporting active pharmaceutical ingredients across 258 rivers in 104 countries, while Ebele et al. (2017) argue that many emerging contaminants remain biologically active in freshwater systems even after conventional wastewater treatment. These pollutants are commonly described as xenobiotics because they are foreign chemical substances that are not naturally expected within an organism's normal biochemical system, and they include pesticides, herbicides, heavy metals, hydrocarbons, pharmaceuticals, personal-care residues and endocrine-disrupting compounds (Ubulom et al. 2023). Although chemical monitoring is useful for identifying contaminant concentrations, Kroon et al. (2017) caution that chemical data alone may not fully explain biological harm because aquatic organisms respond to mixtures through biochemical, cellular, physiological and reproductive pathways.

Fish are particularly important in this context because they occupy central positions in aquatic food webs as predators, prey and nutrient-transfer organisms, while also serving as food resources and practical bioindicators of environmental contamination. Dalzochio et al. (2016) reviewed biomarker studies in aquatic ecosystems and found that fish were the most frequently used bioindicator organisms, which supports their value for linking environmental pollution to biological effect. Their usefulness is strengthened by their continuous exposure to contaminated water, sediments and food, since xenobiotics can enter fish through the gills, skin, ingestion and contact with polluted sediments. However, fish exposure should not be interpreted as a simple accumulation process, because Celino-Brady et al. (2021) show that pollutants such as endocrine-disrupting chemicals may interfere with development, metabolism, growth, behaviour and reproductive function across different life stages.

The link between xenobiotic exposure and genotoxicity is especially significant because many aquatic contaminants can damage DNA, produce chromosomal abnormalities, induce oxidative stress and disrupt DNA repair processes. Hussain et al. (2018) argue that comet and micronucleus assays in fish erythrocytes provide useful evidence of pollution-induced DNA damage, while Drąg-Kozak et al. (2022) show that cadmium and zinc exposure increased micronuclei, nuclear abnormalities and DNA-integrity defects in Prussian carp. This evidence refutes the narrow view that genotoxicity is only a cellular endpoint, because DNA damage can weaken immunity, reduce growth, impair reproduction, increase mortality and lower the ability of fish populations to replace themselves. Aich et al. (2025) further support this broader interpretation by showing that long-term pharmaceutical exposure can affect fish body condition, sperm performance and behavioural traits that are relevant to survival and reproductive success.

Despite this evidence, a clear gap remains in how genotoxicity is often discussed. Many studies focus strongly on cellular markers such as micronuclei, comet-tail DNA, nuclear abnormalities and chromosomal damage, but fewer studies connect these markers critically to fish health, reproductive performance, recruitment failure, population dynamics, ecosystem stability and human health risk. This gap matters because the ecological consequence of genotoxicity is not limited to damaged cells; it becomes more serious when impaired fish growth, fertility and survival alter food-web structure, fisheries productivity and contaminant transfer to humans through fish consumption. Chronic genotoxic stress, including persistent mutations or DNA damage, can eventually lead to cancer in humans the process called Carcinogenesis, and also alter genetic code, often as a result of DNA damage through the process called mutagenesis (Aich et al. 2025)

Therefore, this review examines the impacts of xenobiotic-induced genotoxicity on fish health and population dynamics, with emphasis on mechanisms of DNA damage, biomarkers of exposure, reproductive consequences, ecosystem implications and pollution-control strategies.

The conceptual pathway linking xenobiotic pollution to fish exposure, DNA damage, health decline, reproductive impairment, population decline and wider ecosystem and human health risks is presented in Figure 1.

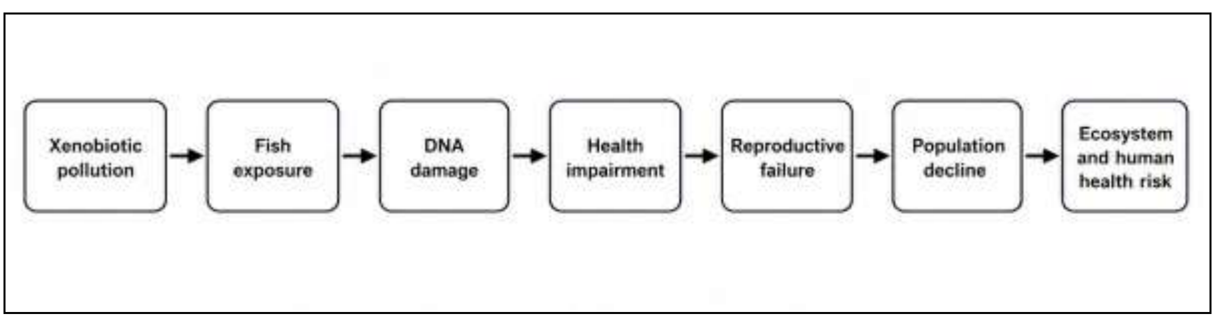

Fig.1: Diagram showing Xenobiotic pollution pathways (Drąg-Kozak et al. 2022)

II. MATERIALS AND METHODS

This article adopted a secondary-source narrative review design because it sought to synthesise existing evidence on xenobiotic-induced genotoxicity in fish rather than generate new laboratory or field data. As Ferrari (2015) notes, a narrative review is appropriate where dispersed literature must be interpreted around a defined scientific issue, while Snyder (2019) argues that review studies require transparent search, selection and synthesis procedures to improve scholarly reliability. Relevant literature was identified from peer-reviewed journal articles, toxicology books, environmental toxicology studies and selected regulatory documents. However, because this article is primarily a scientific review, peer-reviewed DOI-indexed sources published between 2015 and 2025 were prioritized for evidence on biological effects, while institutional documents were used only for contextual understanding of pollution control.

The literature search focused on xenobiotics in aquatic environments, genotoxicity in fish, DNA damage and repair mechanisms, biomarkers such as the micronucleus assay and comet assay, fish growth, survival, reproduction and population dynamics, bioaccumulation, biomagnification, human health risks, and environmental regulation. Search terms included “fish genotoxicity,” “xenobiotics in aquatic environments,” “micronucleus assay in fish,” “comet assay and fish DNA damage,” “heavy metals and fish genotoxicity,” “pharmaceutical pollution in fish,” “bioaccumulation in fish,” and “population effects of aquatic pollutants.”

Studies were included if they addressed fish, aquatic pollution, xenobiotics, DNA damage, biomarkers, reproductive effects, growth, survival, bioaccumulation or population-level consequences. Studies were excluded if they focused only on non-aquatic organisms, lacked relevance to fish genotoxicity or fish health, provided incomplete bibliographic information, or could not be verified through credible academic sources. Evidence was extracted narratively by identifying pollutant type, exposure pathway, fish species or biological model, biomarker used, reported effect and relevance to fish health or population dynamics. Although PRISMA provides useful guidance for systematic reviews, this study did not claim to be a systematic review because its purpose was interpretive synthesis rather than exhaustive meta-analysis (Page et al., 2021).

III. RESULTS

3.1 Sources and Types of Genotoxic Xenobiotics in Aquatic Environments

The reviewed literature shows that fish are exposed to multiple xenobiotic classes rather than single pollutants, which makes aquatic genotoxicity a mixture-based problem. Pharmaceuticals and personal care products enter freshwater systems mainly through human excretion, hospital waste, domestic wastewater and incomplete removal during wastewater treatment, and Ebele et al. (2017) argue that their persistence is concerning because many remain biologically active at low environmental concentrations. Similarly, Pironti et al. (2021) identify endocrine-disrupting compounds, industrial chemicals, persistent organic pollutants, pesticides, artificial sweeteners and flame retardants as contaminants of emerging concern in aquatic systems. However, the problem is not limited to emerging contaminants, because Ghafarifarsani et al. (2024) show that pesticides and heavy metals remain among the most potent aquatic toxicants due to their effects on fish growth, physiology, immunity, reproduction and organ structure. The evidence therefore indicates that genotoxic xenobiotics in aquatic environments include pesticides, herbicides, heavy metals, pharmaceuticals, hydrocarbons, industrial chemicals, persistent organic pollutants and endocrine-disrupting compounds (Okwet et al. 2025). These substances enter water bodies through agricultural runoff, industrial effluents, municipal wastewater, pharmaceutical waste, petroleum contamination and contaminated sediments (Ubulom et al. 2023). The major xenobiotic categories, their common sources and potential genotoxic effects in fish are summarized by Ghafarifarsani et al. (2024) as shown in Table 1.

Table 1: Common Xenobiotics, Sources and Potential Genotoxic Effects in Fish (Ghafarifarsani et al. 2024)

Xenobiotic category	Common sources	Examples	Potential genotoxic/biological effects in fish
Heavy metals	Industrial effluent, mining, urban runoff, contaminated sediments	Mercury, cadmium, lead, arsenic	DNA strand breaks, micronuclei formation, oxidative stress, impaired growth, organ damage
Pesticides and herbicides	Agricultural runoff, drainage from farmlands, improper chemical disposal	Chlorpyrifos, endosulfan, atrazine	Chromosomal abnormalities, oxidative stress, reproductive dysfunction, developmental defects
Pharmaceuticals	Domestic wastewater, hospital waste, pharmaceutical residues	Antibiotics, antidepressants, synthetic hormones	Endocrine disruption, altered behaviour, impaired reproduction, DNA damage
Petroleum hydrocarbons	Oil spills, refinery discharge, shipping activities, urban runoff	PAHs, crude oil fractions	DNA adduct formation, oxidative stress, reduced survival, carcinogenic effects
Persistent organic pollutants	Industrial processes, waste burning, contaminated sediments	PCBs, dioxins	Bioaccumulation, endocrine disruption, immune suppression, reproductive impairment
Industrial chemicals	Manufacturing discharge, plastic production, chemical industries	Solvents, BPA, flame retardants	DNA damage, hormonal disruption, impaired development and population-level risks

3.2 Pathways of Exposure, Persistence and Bioaccumulation

The findings further show that fish exposure occurs through direct and indirect pathways, making them suitable indicators of both water-column and food-chain contamination. Ray and Shaju (2023) explain that pesticides are readily absorbed into fish bodies and may enter the human food chain when contaminated fish are consumed. This observation is important because fish do not only contact pollutants externally; they absorb xenobiotics through the gills, skin, ingestion of contaminated prey and sediment contact (Ubulom et al. 2022). Dey et al. (2023) add that fish embryos and developing stages are especially vulnerable because they develop in water and are exposed to metals, pesticides, synthetic hormones and other genotoxic agents during sensitive phases of cell division. Persistence strengthens this risk because non-biodegradable or slowly degradable contaminants may remain in water and sediments long after discharge. Consequently, bioaccumulation occurs

when xenobiotics build up in fish tissues such as liver, kidney, gills, muscles and fat, while biomagnification occurs when contaminant concentrations increase across trophic levels. The result is that low-dose environmental contamination may become biologically significant over time, especially for predatory fish, fish consumers and ecosystems dependent on stable food-web relationships.

3.3 Biomarkers Used in Detecting Genotoxicity in Fish

The reviewed studies identify the micronucleus assay, comet assay, erythrocyte nuclear abnormalities and chromosomal aberration tests as major biomarkers for detecting genotoxicity in fish. Kroon et al. (2017) argue that biomarkers are valuable because they connect external contaminant exposure with internal tissue contamination and early biological effects, thereby making them more informative than chemical measurements alone. The micronucleus assay detects chromosomal fragments or whole chromosomes that fail to integrate into daughter nuclei during cell division, and it is therefore useful for identifying clastogenic and aneugenic damage. In contrast, the comet assay detects DNA strand breaks at the level of individual cells, which makes it sensitive for identifying early DNA damage before it becomes visible as chromosomal abnormality. Hussain et al. (2018) demonstrated the practical value of these assays in Labeo rohita, reporting significant DNA fragmentation in fish from polluted sites, including comet-tail DNA values of 42.21 ± 2.06%, 31.26 ± 2.41% and 21.84 ± 2.21% across three polluted locations. Drąg-Kozak et al. (2022) also found that Prussian carp exposed to 4.0 mg/L cadmium, 4.0 mg/L zinc and their mixture for 14, 21 and 28 days showed significantly higher micronuclei, nuclear abnormalities and DNA-integrity defects than controls. These findings indicate that biomarker-based monitoring is useful because it detects early genetic stress before wider ecological consequences become irreversible. The major biomarkers used for detecting genotoxicity in fish and their environmental relevance are presented in Table 2.

Table 2: Biomarkers Used in Assessing Genotoxicity in Fish (Hussain et al. 2018)

Biomarker/test	What it detects	Common sample/cell type	Environmental relevance
Micronucleus assay	Chromosomal fragments or whole chromosomes not included in daughter nuclei	Fish erythrocytes, gill or liver cells	Indicates chromosomal damage caused by clastogenic or aneugenic pollutants
Comet assay	DNA strand breaks at single-cell level	Blood, gill, liver or kidney cells	Detects early DNA damage before severe cellular abnormalities develop
Erythrocyte nuclear abnormalities	Nuclear deformities linked to genetic or cytotoxic stress	Peripheral blood erythrocytes	Useful for detecting pollution-induced cellular stress in fish
Chromosomal aberration test	Structural or numerical chromosome damage	Dividing cells from blood or tissues	Indicates mutagenic effects and genome instability
Oxidative stress biomarkers	Imbalance between reactive oxygen species and antioxidant defence	Liver, gill, blood or muscle tissues	Supports interpretation of DNA damage caused by pollutant-induced oxidative stress

3.4 Reported Effects on Fish Health and Biological Function

The major biological effects reported in the literature include DNA strand breaks, micronuclei formation, chromosomal abnormalities, oxidative stress, impaired growth, weakened immunity, reproductive dysfunction, developmental abnormalities and increased mortality. Li et al. (2022) found significantly increased DNA damage in several tissues of insecticide-exposed fish, which supports the argument that genotoxicity is not confined to blood cells but can affect organs involved in detoxification, respiration and metabolism. Aich et al. (2025) further show that long-term pharmaceutical exposure can affect body condition, behavioural traits and sperm performance in fish, suggesting that pollution can influence traits directly linked to survival and reproduction. This is important because genotoxicity may begin as molecular injury but becomes ecologically significant when it reduces fish fitness. Grădinariu et al. (2025) also show that pharmaceutical pollutants can induce oxidative stress in fish, with biomarkers such as catalase, superoxide dismutase, glutathione peroxidase, glutathione-S-transferase and glutathione reductase serving as early-warning indicators of physiological disturbance. Therefore, the reviewed evidence suggests that xenobiotic-induced genotoxicity affects fish at several biological levels: DNA damage disrupts cellular stability, oxidative stress weakens physiological resilience, organ damage reduces biological performance, and reproductive impairment threatens population renewal.

IV. DISCUSSION

4.1 From DNA Damage to Fish Health Decline

The reviewed evidence suggests that xenobiotic-induced genotoxicity should not be interpreted as a narrow cellular event, because DNA damage can initiate wider physiological decline in fish. According to Drąg-Kozak et al. (2022), exposure of Prussian carp to cadmium, zinc and their mixture for 14, 21 and 28 days produced significantly higher micronuclei, nuclear abnormalities and DNA-integrity defects than control groups. This is important because micronuclei and DNA strand breaks indicate that pollutants are not only present in the aquatic environment but are biologically active within fish cells. However, the significance of these findings goes beyond laboratory diagnosis. As Dey et al. (2023) explain, DNA repair genes play essential roles during fish embryo development, organ formation and responses to genotoxic stress, which means that disruption of DNA integrity can interfere with growth and developmental stability.

This point is strengthened by Grădinariu et al. (2025), who argue that oxidative damage to proteins and DNA can impair metabolic activity, suppress immunity and reduce growth and reproductive performance in fish exposed to pharmaceutical pollutants. Therefore, DNA damage becomes ecologically meaningful when it weakens the systems that allow fish to survive, feed, develop and reproduce. Although some fish may activate antioxidant and DNA-repair responses after toxicant exposure, this compensatory ability is limited when exposure is chronic, mixed or persistent. Consequently, genotoxicity should be treated as an early-warning signal of declining fish health rather than as an isolated molecular endpoint.

4.2 Reproductive Effects and Population Dynamics

The strongest ecological implication of genotoxicity is its effect on reproduction and population renewal. Fish populations depend on successful gamete production, fertilization, embryo development, larval survival and recruitment into adult age classes; therefore, any pollutant that disrupts these processes can shift genotoxicity from an individual-health issue to a population-dynamics problem. Aich et al. (2025) demonstrate this connection by showing that long-term exposure to fluoxetine affected body condition, behavioural traits and sperm performance in guppies, indicating that pharmaceutical pollution can influence traits directly linked to reproductive success. This evidence is important because sperm velocity, mating behaviour and body condition are not secondary effects; they determine whether individuals can contribute effectively to the next generation.

Endocrine-disrupting chemicals create an additional reproductive pathway through which aquatic pollutants may affect population structure. Celino-Brady et al. (2021) explain that endocrine-disrupting chemicals can affect growth, development, metabolism and reproduction in fish across different life stages. Similarly, Dasmahapatra et al. (2023), in a systematic review of endocrine-disrupting chemical evaluation in Japanese medaka, identify the species as a useful model for assessing reproductive and developmental disruption. The relevance of these studies is that endocrine disruption can alter gonadal development, sex differentiation, vitellogenin expression, sperm quality, egg viability and larval outcomes.

However, it would be too simplistic to claim that every genotoxic exposure automatically leads to population collapse. Matthiessen (2018) cautions that evidence for endocrine-disrupting effects at wildlife-population level varies by chemical, exposure intensity, species sensitivity and ecological context. Nevertheless, this caution does not weaken the argument; rather, it refines it. The key issue is that when DNA damage and endocrine disruption are persistent enough to reduce fertility, skew sex ratios, impair larval survival or lower recruitment, the consequence becomes demographic. Population decline therefore occurs not merely because individual fish are damaged, but because fewer viable offspring survive to maintain the population.

4.3 Ecosystem and Food-Chain Implications

The population-level effects of genotoxicity have wider ecosystem implications because fish connect several trophic levels within aquatic food webs. When fish populations decline or become reproductively unstable, predator-prey relationships, nutrient cycling, fisheries productivity and biodiversity may also be affected (Aich et al.2025). This is particularly important in freshwater and coastal systems where fish serve as both consumers of smaller organisms and prey for birds, reptiles, mammals and larger fish. As Ray and Shaju (2023) explain, pesticides absorbed by fish can move through the food chain and create toxicological risks beyond the exposed organism. This means that fish are not only victims of aquatic contamination; they can also become carriers of contaminants to higher trophic levels (Okwet et al.2025).

Nevertheless, the concept of biomagnification requires careful interpretation. Miller et al. (2020), after reviewing 116 publications on microplastics in marine organisms, found evidence that microplastics bioaccumulate within trophic levels but concluded that field evidence did not clearly support biomagnification across a general marine food web. This finding is useful because it prevents overgeneralization. Though Ubulom et al. (2023) didn't support this finding. Some contaminants, such as mercury and persistent organic pollutants, are well known for trophic transfer, whereas evidence for microplastics and associated additives remains more complex. Therefore, the ecological implication is not that all xenobiotics behave identically, but that persistent and bioaccumulative pollutants can alter food-web safety, species interactions and ecosystem resilience when exposure becomes chronic (Kroon et al. 2017)

4.4 Human Health and Policy Implications

The human-health implication of fish genotoxicity is significant because fish are an important source of protein, fatty acids and micronutrients, especially in communities that depend heavily on freshwater, coastal or aquaculture products (Miller et al.2020). However, the nutritional value of fish can become compromised when edible tissues accumulate heavy metals, pesticides, persistent organic pollutants or pharmaceutical residues. Noman et al. (2022), in a study of commercially important fishes and shellfishes from Hangzhou Bay, reported the metal concentration hierarchy as Zn > Cu > As > Cr > Cd > Pb > Hg and found that children were more susceptible than adults to non-carcinogenic risk, while arsenic and cadmium contributed to potential carcinogenic risk through seafood consumption. This evidence shows that aquatic pollution is not only an environmental issue but also a food-safety and public-health concern Policy responses should therefore move beyond occasional chemical testing toward integrated monitoring that combines water-quality analysis, sediment assessment, fish-tissue screening and biomarker-based surveillance. Kroon et al. (2017) argue that biomarkers are useful because they link contaminant exposure to biological effect, which makes them valuable for early detection of ecological risk. Yet monitoring alone is insufficient if pollution sources remain uncontrolled. Effective management requires improved wastewater treatment, stricter industrial and agricultural discharge control, enforcement of environmental regulations, bioremediation of polluted sites and public awareness on safe chemical disposal (Yawo et al. 2023). In this sense, biomarker evidence should not remain in academic studies alone; it should inform regulatory thresholds, environmental impact assessment, aquaculture safety and public-health advisories.

V. CONCLUSION

Xenobiotic-induced genotoxicity represents a serious threat to fish health and population sustainability because it links environmental contamination with biological damage at cellular, organismal and ecological levels. The evidence reviewed shows that pollutants such as heavy metals, pesticides, petroleum hydrocarbons, pharmaceuticals, persistent organic pollutants and endocrine-disrupting compounds can enter fish through the gills, skin, contaminated sediments and food-chain pathways. Once absorbed, these xenobiotics may damage DNA, produce micronuclei, induce chromosomal abnormalities, increase oxidative stress and weaken DNA repair responses. Although such effects are first detected at the cellular level, their significance becomes clearer when they are connected to impaired growth, weakened immunity, organ dysfunction, reproductive failure, reduced survival and poor recruitment. This means that genotoxicity is not only a laboratory indicator of pollution, but a warning sign of declining fish fitness and possible population instability.

The wider implication is that damaged fish populations can affect aquatic ecosystems, food chains, fisheries, aquaculture productivity and human health. Where reproduction is compromised through poor sperm quality, reduced egg viability, abnormal gonadal development, endocrine disruption or larval mortality, fish populations may lose their ability to replace themselves over time. Bioaccumulation and biomagnification also increase the risk that contaminants will move from smaller aquatic organisms to larger fish and eventually to human consumers. Therefore, protecting fish from genotoxic xenobiotics requires regular biomarker-based monitoring, improved wastewater treatment, stricter pollution control, stronger enforcement of environmental regulations and continuous research into emerging contaminants and remediation strategies.

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