***Review Article***

**Impact of Aquatic Pollution on Embryonic and Larval Development in Fish: A Comprehensive Review**

**ABSTRACT**

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| For environmental degradation fish is considered as an early warning system, they are also the biomarkers to measure the specific existence of toxic and carcinogenic components in the environment. Aquatic pollution stemming from industrial, agricultural, and urban activities exerts a profound impact on ecosystems, notably affecting fish reproduction, development, and overall well-being, leading to significant ecological and economic repercussions. The contamination of toxic metals such as copper, zinc, and cadmium severely impact fish embryos, causing delayed hatching, disruptions in development, and heightened mortality rates. These metals disrupt physiological processes, jeopardizing embryonic survival and resulting in organogenesis defects, metabolic imbalances, and altered enzyme activity. Similarly, pesticides, particularly herbicides like Thiobencarb, persist in the environment, perturbing hormonal functions and inducing developmental, behavioural and reproductive alterations in fish, with younger stages being more susceptible than older ones. Microplastics have arisen as a persistent environmental menace, disturbing aquatic ecosystems by affecting fish larvae and embryos. Ingestion of microplastics leads to growth retardation, diminished survival rates, and developmental abnormalities primarily due to metabolic disruptions, oxidative stress, and alterations in gut microbiomes. These consequences underscore plastic pollution's significant ecological and health hazards, disrupting nutrient absorption and impairing long-term survival. Oil spills, which expose organisms to polycyclic aromatic hydrocarbons (PAHs), trigger developmental anomalies, particularly in species like Atlantic cod, resulting in malformations, anaemia, and organ edema. These pollutants notably impact fish embryonic development, causing deformities, delays in development, and increased mortality rates, consequently exacerbating ecosystem well-being. Furthermore, exposure to toxicants disrupts fish behaviour, affecting feeding habits, visual reflexes, and predator avoidance mechanisms through neurotoxic effects and sensory system disturbances. **Conclusion:** Aquatic pollution poses significant risks to fish health, reproduction, and survival, with widespread implications for aquatic ecosystems. |

*Keywords: Aquatic pollution, Embryonic development, Larval Development, Toxic metal*

1. **INTRODUCTION**

Aquatic environments are significant recipients of contaminants that might lead to detrimental effects on aquatic organisms over time. Aquatic pollution contaminates water bodies, including rivers, lakes, oceans, and groundwater, by deleterious compounds resulting from human activity. This pollution can adversely impact aquatic ecosystems, marine life, and human well-being. These implications become apparent only after changes occur in the community, ecology, or population; at this point, it may be impossible to reverse the change. Over the past few years, this has become a significant worry because various contaminants poison water bodies. The primary causes of aquatic pollution are rapid industrialization, toxic metal discharge from commercial and domestic wastes, farming operations, chemical and physical weathering of rocks, soil erosion, waste disposal, and atmospheric deposition, which heavily contaminate natural aquatic systems. The increase in river pollution and contamination of other water bodies has emerged as a significant concern in recent years, attributed to the direct discharge of industrial effluents and urban sewage with minimal or no treatment. Despite being non-target creatures, fish have fallen prey to water pollution through the accumulation of poisons and toxic metals. The accumulation of toxicants negatively impacts the human population through fish eating (Ansari *et al*., 2004). For environmental degradation fish is considered as an early warning system, they are also the biomarkers to measure the specific existence of toxic and carcinogenic components in the environment. Aquatic pollution has the potential to biologically influence biota by affecting their metabolic, respiratory, and immunological capabilities, development and structural anomalies as well as causing changes in population structure. The measures of the ecological consequences are the pollutant buildup in biota, the considerable rise in pollution-related reproduction, developmental anomalies, and other essential modifications in biochemical or physiological processes in fish and invertebrates (Arukwe, 2001).

To identify early indicators of these effects, it is essential to pick sensitive measures to detect tiny environmental disruptions. Embryos and larvae of fish have been employed for decades in toxicity testing because these phases of development are well-documented and very susceptible to toxicants (Rosenthal and Alderdice, 1976; Westernhagen, 1988). This has been applied to monitoring the sublethal biological impacts of pollution on the marine environment throughout the last decade. The incidence of morphological deformities and chromosomal aberrations in developing fish embryos has been utilized successfully to detect pollution effects in vast sections of the North Atlantic (Longwell and Hughes, 1980; Chang and Longwell, 1984; Westernhagen *et al*., 1988). Furthermore, there are indications of strong negative relationships between levels of chlorinated hydrocarbons in tissues of wild marine fish and the quality and survivability of their eggs (Westernhagen *et al*., 1981, 1989; Hansen *et al*., 1985; Spies and Rice, 1988). Additionally, water pollutants are believed to directly impact spawned eggs, particularly in the concentration zone at the sea-surface microlayer (Westernhagen *et al*., 1987; Cameron *et al*., 1992). So, Aquatic pollution negatively impacts the embryonic and larval development of fish by causing abnormalities, reduced hatching success, and increased mortality due to exposure to toxic metals, pesticides, and other contaminants. This review examines the physiological, biochemical, and genetic effects of pollutants on early fish development, highlighting the need for conservation and pollution control measures.

1. **CONTAMINANTS IN AN AQUATIC ENVIRONMENT AND THEIR EFFECT**
	1. **Toxic metal**

Toxic metals contamination is a raising concern in aquatic ecosystems even in minute concentrations (Aldavood et al. 2020). Increasing use of feed, pesticides, chemicals, fertilizers and discharge effluents carrying inorganic components which could build up in the sediment (Cd and Cu) can pose issues for culture systems (Pinto et al. 2021). Toxic metals can diffuse in to the tissues through skin and gills or through diet and then interfering with kidney and liver the primary targets and with other unconventional ones like muscles, lateral line and retina (Motta et al. 2021). Some non-essential trace metals long term exposure (Cd) affects central and peripheral nervous system, urinary system, liver, cardiovascular and reproductive system causing serious cancers (Puangprasert and Prueksasit 2019). For human consumption Cd is declared as carcinogenic metal by International Agency for Research on Cancer (IARC) and some studies stated that Cd can reach 0.1–0.3 μg L-1 in water and can reach up to 30 μg L-1 in wastewater discharge (Ahmed and Mokhtar 2020). Fish embryonic morphological, physiological, and behavioural endpoints of numerous various species are used to measure chemical's toxicity (Liu et al. 2021). These metals get accumulated in the tissues and other organs. Franco-Fuentes et al. (2021) noticed wide range of concentrations for different metallic compounds. In *S. violacea* and *P. clemensi*, they noticed concentrations of Zn were higher in liver in comparison to gonads (31.1 and 17.1 mg/kg wt. and 51.6 and 27.0 respectively) and for *C. princeps, C. affinis* and *P. albomaculatus* higher levels of Cd were recorded in the liver (23.1, 10.6 and 18.5 mg/kg wt. respectively). Fish sensitivity to various metallic compounds will vary based on food availability and probably other environmental stressors (Ye et al., 2021)

Toxic metal pollution of water impacts fish development and reproduction, among other physiological functions. According to Jezierska and Witeska (2001), the effects of waterborne metals on fish are linked to their absorption and storage by the organism, which causes metal-induced disruptions in the structure and function of different tissues and organs. Fish embryos are especially vulnerable to poisoning during their early development, including when the eggshell shields them.

Metals in water may have an impact on spawners, which could lead to disruptions in embryonic development. Fish exposed to metals frequently exhibit higher gonad metal levels. Szarek-Gwiazda (1999) found that the ovaries of *Noemacheilus barbatulus* had elevated levels of zinc, lead, and cadmium. Inhibition of embryonic development may occur at several developmental stages. According to Perry *et al*. (1988), methylmercury prolonged the early stages of *Fundulus heteroclitus* embryonic development by delaying it. On the head, the embryos grow what are known as hatching glands before hatching. Chorionase, required for the dissolution of the eggshell during hatching, is produced by the glands. Waterborne metals may alter the growth and functioning of these glands. Metal-induced disruptions of transcription and translation resulting in lower synthesis of proteins, including chorionase, were found by Kapur and Yadav (1982). Norrgren and Degerman (1993) discovered that the chorion of non- or incompletely formed *Salmo salar* eggs developed at low pH and in an Al-rich environment maintained intact inner surfaces.

In contrast, regularly hatched eggs had a considerably looser structure. This possibly developed from decreased activity of chorionase. The hatching glands of Cyprinus carpio embryos treated with zinc or copper exhibited intracellular granules and smaller surfaces than the controls (Mis and Bigaj, 1997). These alterations resulted in reduced chorionase production.

The provided findings demonstrate that the commencement of hatching is often delayed, which is probably connected to the functioning of hatching glands. However, the entire process may be slowed down or expedited. Delayed hatching of *Salvelinus fontinalis* treated to aluminum at low pH was found by Cleveland *et al*. (1986). Dave and Xiu (1991) observed delayed hatching in metal-treated *Brachydanio rerio*. Their studies reveal that copper and nickel are the biggest hatching inhibitors. Even at low doses that do not result in larval death, copper impacts hatching.

Woodworth and Pascoe (1982) reported that *Oncorhynchus mykiss* embryos exposed to 0.1 mg dm-3 of Cd hatched 50–60 hours earlier than the control. Similar results were achieved by Somasundaram *et al*. (1984). Brungs (1969) found premature hatching of *Pimephales promelas* subjected to zinc; however, the hatching process was protracted. Klein-MacPhee *et al*. (1984) found that *Pseudopleuronectes americanus* treated with silver exhibited a similar response.

During embryonic development, toxic metal intoxication decreases embryo survival and, consequently, hatching success. Most malformed embryos and some usually developed ones perish during embryonic development. The highest mortality of embryos occurred within the first 24 hours following fertilization, and roughly 20% of embryos die even under control conditions (Słomin'ska, 1998). During the blastula development stage, the highest embryonic mortality rate (>15%) was recorded (Ługowska, 2005). Metal exposure during this stage strongly influences the survival of the embryos. According to Słomin'ska (1998), at concentrations more than 1 mg dm-3, lead significantly increased mortality compared to the controls, copper at concentrations greater than 0.3 mg dm-3. According to Jezierska and Słomin´ska's (1997) findings, 100% mortality occurred at 0.3 mg dm-3, and the survival of Cu-exposed embryos (0.1 mg dm-3) 24 hours after fertilization was significantly lower than that of controls. According to Ługowska (2005), embryos primarily died during the blastula (>25%) and body segmentation (>15%) stages following exposure to highly toxic copper.

* 1. **Pesticide**

A class of chemical substances known as pesticides is designed to eradicate pests, such as bacteria, fungi, and weeds. Based on their target organism, Pesticides are divided into herbicides, insecticides, nematicides, molluscicides, piscicides, avicides, rodenticides, bactericides, and fungicides. According to Jayaraj *et al*. (2016), these compounds are increasingly utilized in domestic animals, livestock, agriculture, and home goods. The persistence of certain pesticides in the environment is one of their characteristics. They have lengthy half-lives, are highly lipophilic, bioaccumulate in the trophic chain, and are non-biodegradable. Their effects can be detected even several years after their application (Jayaraj *et al*., 2016; Mhadhbi and Beiras, 2012).

Worldwide, the overuse and abuse of pesticides are accountable for harmful effects on the ecosystem's health, impacting numerous terrestrial and aquatic species. However, the negative impacts on microbes, invertebrates, plants, fish, and amphibians can be severe in aquatic ecosystems (Jayaraj *et al*., 2016; Corcellas *et al*., 2017). Various pesticides and related chemical substances can alter hormone control, chemical messengers, and metabolic pathways. In the early stages of embryo development, hormones play a vital role in cell and tissue differentiation. Hence, exposure to these endocrine-disrupting drugs can lead to a change in the expected growth of the embryo. Endocrine-disrupting drugs can influence both the embryo and the adult. It may take until later in the embryos' development to notice the impacts of pesticide exposure, which include behavioral, reproductive, and disease-susceptibility changes (Ahmad *et al*., 2010; McAloose and Newton, 2009).

Since fishes exist solely in aquatic environments, they are outstanding bioindicators of environmental pollution. The contact with excessive quantities of pesticides and related compounds is generally lethal. However, prolonged exposure to low levels significantly impacts individuals over the medium and long term. They can produce minor changes in behavior, physiology, development, longevity, and reproduction due to metabolic abnormalities and enzyme inhibition. Zebrafish (*Danio rerio*) embryos are one of the most prevalent species in toxicology essays. However, the effects of pesticides have been explored in other fish species (Khan and Law, 2005). Chhaba et al., (2024) founded out that the LC50 (lethal concentration 50) of chloropyrifos (CPF) for *Pangasianodon hypophthalmus* is 0.106 mg L-1. Upon exposure to CPF significant changes in enzymes like acid phosphatase (ACP), alkaline phosphatase (ALP), aspartate aminotransferase (AST), serum alanine aminotransferase (ALT) and acetylcholine of blood serum were observed in comparison to control. They also observed that erythrocyte count, haematocrit level and haemoglobin level significantly decreased in fish exposed to CPF. Karmakar et al., (2021) revealed that up on exposure of *Labeo rohita* to nonylphenol (NP), the acute toxicity of NP exhibited many behavioural alterations. Chemical treated fish, showed decreased total erythrocyte count and haemoglobin. Decreasing AChE activity levels supported the neurological disorder occurred in fish.

* 1. **Herbicide**

Herbicides represent the most significant share of pesticides used in agriculture. In 2007, Herbicides accounted for 40% (950 million kg) of the projected 2.4 billion kilograms of pesticides globally (USEPA, 2012a). Although agricultural applications dominate the market, herbicides are also used in forestry and other applications, such as controlling vegetation on rights-of-way, industrial, and urban sites.

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| **Figure 1**. Graphical presentation of the fate of herbicides in surface waters |

In aquatic environments, herbicides can be administered directly to surface water to suppress aquatic weeds or carried to surface water by various paths (Figure 1). Direct overspray deposition or spray drift from adjacent areas is one possible exposure pathway. Fish exposure is more likely when weeds are purposefully added to water to eliminate aquatic nuisances or weeds in rice paddies.Many herbicides can negatively impact the physiological process, body functions causing long-term harm to health and ecosystems. These chemicals can interfere with fish metabolism, reproductive systems, and overall survival, leading to a decline in fish populations and disturbing the balance of aquatic ecosystems. For instance, glyphosate-based herbicides action is based on shikimate pathway which inhibits the enzyme 5-enolpiruvilshikimate-3 phosphate synthetase (EPSPS) activity and this leads to the protein shortage and ultimately death (Boocock and Coggins, 1983). Fish in their earlier phases are often considered more sensitive to toxins than those in their older stages. A few studies have examined fish's sensitivity to herbicides at different developmental stages. Similar findings were found in a study on the toxicity of the herbicide thiobencarb in Atlantic silverside (*Menida menida*), Tidewater silverside (*Menida peninsulae*), and California grunion (*Leuresthes tenuis*) (Borthwick *et al*., 1985). The sensitivity of younger fish (0–7 days post-hatch) was higher than that of older fish (28 days post-hatch), and the species' sensitivity differences varied from two to three times. According to a study on developmental end-points in medaka (*Oryzias latipes*) using the same herbicide, thiobencarb was more hazardous to stage 10 blastulas (EC50 of 3600 mg/L) than to stage 23 fish (EC50 of 4100 mg/L), which were the beating heart (Villalobos *et al*., 2000).

* 1. **Plastic and microplastic**

Plastic pollution, particularly in the form of microplastics (1 to 5,000 μm particles), has become a pervasive environmental issue, infiltrating ecosystems worldwide (Hale et al., 2020). The contamination of aquatic environments by plastic waste, ranging from large debris to microscopic particles, poses significant threats to water quality, biodiversity, and the overall health of ecosystems. Microplastics are especially concerning due to their durability, low production cost, and strong resistance to degradation, allowing them to persist and spread globally. These particles are found in oceans, rivers, soil, and even groundwater systems, making them a ubiquitous pollutant in the biosphere.

Over 8 million tons of microplastics are estimated to enter the oceans annually from land-based sources, while the total amount of microplastic waste in land soils and surface waters remains uncertain (Jambeck *et al*., 2015; Zhang *et al*., 2020). The widespread distribution of microplastics is driven by their lightweight nature, ease of transport, and ability to be fixed in various environments. Some microplastics remain in the soil, while others are washed into water bodies through runoff (Bergmann *et al*., 2019; Raven Hurt *et al*., 2020). Additionally, microplastics are increasingly recognized as a pollutant in freshwater ecosystems and even the atmosphere (Wong *et al*., 2020; Wang *et al*., 2020). Microplastics are pervasive across all environmental compartments. They have been detected in surface waters, midwater columns, deep-sea sediments, and even remote regions like polar ice (Obbard *et al*., 2014). Environmental factors such as wind, water currents, and temperature influence their distribution. For example, denser polymers like polyvinyl chloride tend to sink to the ocean floor, while less dense materials like polyethylene and polypropylene float, often concentrating in gyres and coastal zones (Eriksen *et al*., 2014). Plastic and microplastic contamination of aquatic ecosystems disrupts various ecological processes and poses significant risks to aquatic organisms. These contaminants affect ecosystems in multifaceted ways, ranging from physical blockages and ingestion to chemical toxicity and habitat disruption. Microplastics in aquatic environments have been shown to disrupt the expected growth of fish larvae and embryos. Microplastics can physically obstruct feeding, block the digestive tract, and alter the energy allocation in developing organisms. In fish larvae, the ingestion of microplastics can lead to reduced food intake and digestive efficiency, diverting energy away from growth and development to deal with the physical stress caused by the presence of foreign particles in the gastrointestinal tract. For instance, fish larvae exposed to MPs have exhibited reduced growth rates and developmental delays (Limonta *et al*., 2019). In a study on zebrafish (*Danio rerio*), exposure to polystyrene microplastics resulted in delayed growth due to the accumulation of nanoparticles in vital organs like the liver and brain, which induced cellular stress responses (Lu et al., 2016).

Similarly, MPs have been linked to slower development and reduced survival rates in species like bighead carp (*Aristichthys nobilis*) (Zhang et al., 2024) and hybrid snakehead (*Channa maculata × Channa argus*) (Wang et al., 2022). The sensitivity to microplastic contamination varies across species. For instance, bighead carp, more sensitive to water quality deterioration, showed significant developmental delays and reduced survival rates when exposed to MPs (Zhang *et al*., 2021.). In hybrid snakehead, a carnivorous species, MPs did not accumulate as heavily in the digestive tract as in filter-feeding fish. However, chronic exposure still led to oxidative stress and mild metabolic disturbances. The reduced growth observed in these species can be attributed to the diversion of metabolic resources from developmental processes to detoxification and stress responses, thus impairing proper organ and tissue formation during early life stages. This disruption can lead to physical deformities, increased susceptibility to disease, and reduced overall fitness, all indicative of impaired growth and development. Cattaneo et al. (2023) studied the effect of MPs of various sizes (size range 1–5 µm and 40–47 µm) and concentrations (50 and 500 mg/kg) on *Danio rerio* fish developmental stage (larval vs. juvenile) through diet. They observed that smaller MPs and longer dietary exposure translocated MPs from gut to various organs and tissues. In juveniles, elevated levels of oxidative stress markers is observed which is the result of hepatic accumulation of MPs.

* 1. **Oils and hydrocarbon**

In instances of oil spills in the marine environment, oil endures several weathering procedures altering the physical and chemical nature of the oil. One of the most essential processes in weathering is the creation of oil-in-water dispersions, as this enhances the surface-to-volume ratio of the oil. Formations of oil droplets will naturally arise when oil is liberated from the seabed during blowouts and when surfaced oil is spread into the water column by wave energy during heavy weather. Even though oil is denser than water, micro-sized droplets may stay in the water column due to relatively low surface velocity. Each droplet's bulk oil matrix and the surrounding water will equilibrate oil components like polycyclic aromatic hydrocarbons (PAHs) in proportions dictated mainly by the droplet surface-to-volume ratio and their water solubility. Physical factors, including temperature, pressure, light, currents, the type of oil, time, location, and depth of the leak, may further affect this (NRC, 2005). PAHs are group of organic compounds two or more fused aromatic rings. Other sources of PAHs are anthropogenic processes, particularly from incomplete combustion of organic fuels, volcanic eruptions and forest fires, also contribute to an ambient existence of PAHs.

On the other hand, oil droplets contain most of the oil components found in an oil dispersion, with only a tiny fraction dispersed. It is well established that there is a high association between aberrant fish embryonic development and pollution load in the natural environment (Westernhagen *et al*., 1988). There have been reports of developmental abnormalities in fish embryos exposed to PAHs concerning surface oil spills at sea (Incardona *et al*., 2012) and unintentional oil spills after train derailments (Debruyn *et al*., 2007). According to laboratory studies, fish embryos and larvae exposed to PAHs and crude oil have developmental effects such as failed swim bladder inflation, anemia, pericardial and yolk sac edema (Incardona *et al*., 2004), dorsal curvature (Li *et al*., 2011), and malformations of the craniofacial skeleton (de Soysa *et al*., 2012; Shi *et al*., 2012). The Atlantic cod (*Gadus morhua L*.) embryo is one the most susceptible species to exposure to hydrocarbons, and the first phases of fish embryo formation are generally more vulnerable than the late embryonic stage (Kjørsvik, 1986). The embryo stage of *G. morhua* spans roughly 18–20 days at 5–6 °C (Geffen *et al*., 2006; Hall *et al*., 2004; Kjørsvik *et al*., 1984).

1. **EFFECT OF POLLUTANTS ON HATCHING, EMBRYONIC DEVELOPMENT AND SURVIVAL OF FISH**

Various developmental processes of the early stages of fish are hindered or affected by the pollutants, as the early stages are sensitive to water pollution. The accumulative ability of pollutants adversely affects gamete production and may exhibit direct toxic effects upon early developing embryos, reducing the offspring's quality and quantity. Accumulation of metals like Zn, Pb, and Cd has been found in the gonads of *Noemacheilus barbatulus* (Szarek-Gwiazda, 1999). *Oreochromis aureus*, upon exposure to Cd and Pb, Allen (1995) observed metal accumulation in testes and ovaries, mainly of Cd. It resulted in the contamination of sperm and eggs, adversely affecting fish fertility and embryonic development. Some documents show that metal pollutants may affect the spermatozoa motility time, an essential parameter for successful fertilization. Jezierska *et al*. (1995) observed that Cu, Pb, and Cd reduced the mobility of spermatozoa in *Cyprinus carpio*. The eggshell is ineffective in protecting the embryo from penetrating pollutants/metals, mainly during the swelling phase, leading to the accumulation of contaminants in the egg. These entered metal ions change the chorion structure and permeability. Reports stated that Pb binds mucopolysaccharides to the membrane, altering the permeability, which results in ion-exchange disturbance between the perivitelline fluid and the external environment (Stouthart *et al*., 1994). The level of swelling alters the entire embryonic development process. The embryo changes its position in usually swollen eggs or shows movement every five to ten seconds.

In contrast, eggs that do not swell adequately enough are too small, providing less space than required and may result in abnormal larvae hatching (Korwin-Kossakowski, 1996). Stouthart *et al*. (1996) observed that Cu may be disturbed by selective membrane permeability, resulting in a disturbance in the cation exchange between perivitelline fluid and water. Along with affecting the various developmental processes, these pollutants also affect the early embryo's development rate, and the results depend on the concentration of these pollutants.

Post fertilization, the early stages are susceptible to pollutants, mainly metal intoxication, where most deformities and heavy mortalities occur. These waterborne metal pollutions promote developmental abnormalities, mainly body malformations during organogenesis. Many developmental processes are hindered by these metal pollutants, such as the hatching process, deformities, premature hatching, and even mortality of newly hatched larvae. Ługowska (2005) observed that when embryos of *Cyprinus carpio* were exposed to Cu and Pb (0.2 and 2 mg dm-3), respectively exhibited first developmental retardation at the eye pigmentation stage. As stated earlier, most of the metals also affect the organogenesis stage. Some common malformations observed are shortening of vertebrae, craniofacial anomalies, curvature, yolk sac, and cardiac malformations. Damage to the blood vessels and hemorrhages were also observed by many authors by metals (Jezierska and Słominska, 1997; Jezierska and Gorzynska, 1998; Słominska, 1998) and mutagenic and teratogenic effects on embryos were also observed. Speranza *et al*. (1977) observed abnormal protoplasmic protrusions in *Brachydanio rerio* eggs on exposure to Zn, which showed several cases of a crenated disc on top of the yolk. Mercury-exposed fish (*Fundulus heteroclitus*) embryos exhibited three main malformations: i) Cardiovascular disturbances (underdeveloped heart, poor heartbeat, abnormal heart division, and sometimes no cardiac muscle was observed), ii) Skeletal malformations, and iii) Cranial malformations (eye fusion, partial/underdeveloped skull, and brain (acephalia) (Weis *et al*. 1982).

The organisms' uptake and accumulation of metals results in disturbances in the function and structure of various organs and tissues (Jezierska and Witeska, 2001). Most of the metal toxicity paths of fish described for larvae and adults also apply to the embryos as they share similar metabolic processes of juvenile and adult fish. Similarly, Cd affects sodium and Chloride concentrations and kinetics by altering Na+/K+-ATPase activity, resulting in osmoregulatory failure (Grosell *et al*., 2004). Cadmium ions also bind to sulfur groups containing proteins like cysteine and glutathione, inhibiting the function of these molecules. Behra (1993) noted that cadmium and lead bind to a free calcium sensor protein called calmodulin, affecting various cellular functions. Pb ions also may occupy calcium-binding sites of many calcium-dependent proteins. Cd has reduced the activity of multiple enzymes like citrate synthase, an enzyme of oxidative metabolism, and other related enzymes succinate dehydrogenase (SDH), glucose-6-phosphate dehydrogenase (G6PDH) (Gargiulo *et al*., 1996), lactate dehydrogenase (LDH) (Hilmy *et al*., 1985). The metal Pb also decreased the activity of G6PDH, LDH, and pyruvate kinase (PK) (Osman *et al*.*,* 2007). Caldwell and Phillips (1998) observed that hemoglobin synthesis had been disturbed by inhibiting two essential enzymes, ferrochelatase and gamma levulinic acid dehydrogenase (ALA-D), respectively. Toxic metals have also documented endocrine disruptions. For instance, thyroid hormone levels were reduced by Cd (Hontela *et al*., 1996). Similarly, Cd inhibits estrogen receptors (Le Guevel *et al*., 2000) and disrupts growth hormone expression (Jones *et al*., 2005).

Many authors stated that the increase in developmental time and developmental retardation occurred by the activity of the metals (Cleveland *et al*., 1986; Perry *et al*., 1988; Yulin *et al*., 1990; Ellenberger *et al*., 1994). In *Fundulus heteroclitus*, embryonic development was retarded by methylmercury, extending the early stages, and the authors stated that this may be due to the effect of metal on the mitotic divisions (Perry *et al*., 1988). The embryos that are ready to hatch develop hatching glands over the head. These glands secrete an enzyme called chorionase, which helps disintegrate the shell during hatching. The gland's development and function may be affected by these waterborne metals. Kapur and Yadav (1982) noticed that transcription and translation are disturbed by metals, resulting in reduced synthesis of many proteins, including chorionase. Additionally, detoxification is an energy-consuming process, which is activated by heavy metals, resulting in less growth.

1. **EFFECTS OF POLLUTANTS ON BEHAVIOURAL CHANGES IN LARVAE**

In contrast to conventional physiological and morphological biomarkers, behavioural responses can be sensitive indicators of toxicant exposure (Beitinger, 1990). Disruption of spontaneous activity in response to a toxicant indicates the ability to avoid predators and catch food. Still, these complex behaviours are not limited to these behaviours. In early life stages, toxic substances can disrupt visual function and inherent visual reflexes, as observed through monitoring the optomotor response (OMR). *F. heteroclitus* was deprived of *Artemia* sp. in clear water after embryonic exposure to MeHg (Zhou *et al*.*,* 1996). Dietary contaminants can be introduced to early life through parental care strategies. The larvae of cichlids Symphysodon spp. can be exposed to nutritional contaminants when they feed on parental mucus at first feeding, as demonstrated by Maunder *et al*. (2011). Besides exposure to waterborne and dietary contaminants, maternal contaminants can also be transmitted to offspring through egg-protected eggs (Latif *et al*., 2001; Peake *et al*., 2004). The expected behaviour of fish is interrupted by the high quantity of water pollutants, either directly or indirectly. Fish behaviour changes brought on by pollution may raise exposure levels even further and create positive feedback loops that suggest pollution has a detrimental effect on fish health.

Neurotoxic chemicals can cause behavioural changes and alterations of specific behaviours, affecting an organism's ability to deal with the environment. The expected behaviour is critical for survival as larvae exhibit significant mortality due to predation and starvation. Behaviour changes in larvae can be induced even by a lower level of chemicals required to cause anatomical abnormalities. Fish larvae's behaviour patterns evolve in tandem with their sensory and locomotor systems. The authors' explanations for the MeHg (methyl mercury) transient effects were either due to a delay in neurological development or depression in neurochemical processes (Weis and Weis, 1995b; Zhou *et al*., 1996). In *D. rerio*, MeHg has been found to have comparable effects on feeding behaviour (Samson *et al.*, 2001). According to Miller *et al*. (1993), the behaviour and histological measures of visual ability were studied, which revealed that larvae's reaction to prey was slower than suggested by anatomical measures. This indicates that behavioural limitations are more likely to be behavioral rather than anatomical. Schooling behavior in fish usually develops at the larval stage, which is considered a defence mechanism against predators (Neill and Cullen 1974) and a hydrodynamic aid (Weihs 1975). Larvae of *F. heteroclitus*, upon exposure to MeHg as embryos, exhibited a higher frequency of collisions with each other than control larvae (Ososkov and Weis, 1996).

Egan *et al*. (2009) subjected *D. rerio* to a stressor (anxiolytic drugs) in a pretreatment container before being moved to a unique trapezoidal container for behavioral assessments. The author observed that it increased anxiety manifested as staying in the lower section of the tank. The behavioral parameters evaluated encompassed the time taken to reach the upper section, the duration spent in the upper region, the frequency of transitions to the upper part, occurrences of erratic movements, instances of freezing, and the duration of freezing bouts. Various research studies examining the impact of pollutants on the sense of smell have concentrated on the juvenile and adult phases. Disruption of fish olfaction can impede predator evasion (Scott *et al*., 2003), social interactions (Sloman *et al*., 2003), and reproductive activities (Moore and Waring, 2001). Exposure during development to chemical signals influences gene regulation in the olfactory system (Harden *et al*., 2006), facilitating olfactory imprinting crucial for future successful migrations and adaptive behaviours. Exposure to toxic substances can potentially change gene expression within the olfactory region of fish brains, which could result in behavioural alterations. Exposure to cadmium has been associated with inducing cell death in the olfactory epithelium and affecting the ciliated sensory cells present in the olfactory pits, a phenomenon that Matz and Krone (2007) correlated with changes in aversion behaviours exhibited by *D. rerio* larvae. Upon exposure to marine gas oil and North Sea crude oil, zebra larvae showed no behavioural response to light and dark phases and low baseline swimming activity in comparison to untreated larvae (Johann et al., 2020). In contrast, higher swimming activity was observed in European sea bass, *Dicentrarchus labrax L* juveniles under predation pressure when exposed to petroleum hydrocarbons. The exposure also negatively affected individual fitness through impaired ability to respond to predation (Aimon et al., 2022).

1. **PHYSIOLOGICAL IMPACTS ON GROWTH AND METABOLISM**

Aquatic contaminants can affect the normal physiological processes of fish eggs and larvae. There may be alterations in respiratory, metabolic, and enzyme activity that impact growing fish's general well-being and development. Delays in development and stunted growth can affect fish populations' size and age distribution, affecting their reproduction ability. Fish embryos and larvae exhibit high metabolic rates during their development, driven by rapid cell division, organ formation, and the establishment of various physiological systems. The energy required for these processes is primarily sourced from the yolk during early stages and from exogenous feeding as the larvae mature. Microplastics, however, can disrupt the efficient utilization of this energy in several ways.

One of the most significant metabolic disruptions caused by MPs is oxidative stress. The ingestion of microplastics often produces reactive oxygen species (ROS), which can damage cellular structures, including lipids, proteins, and DNA. Exposure to MPs combined with pesticides like imidacloprid in zebrafish increased lipid peroxidation and altered glycolipid metabolism, leading to further metabolic disturbances and inflammation. These disruptions not only affect the immediate health of the larvae but also have long-term consequences for their growth and survival. Furthermore, microplastics are known to alter the gut microbiome in fish, a key regulator of metabolic functions. The gut microbiome plays an essential role in nutrient absorption and digestion, and any alteration in its composition can lead to reduced metabolic efficiency. Wan *et al*. (2019) found that MPs in the gut lumen of fish altered microbial composition, leading to decreased nutrient absorption and reduced metabolic efficiency. This effect was particularly noticeable in omnivorous species, where MPs persisted longer in the gastrointestinal tract compared to carnivorous or filter-feeding species (Zhang *et al*., 2019; Wang *et al*., 2020).

Various metals also exhibited their negative effects on growth and metabolism of fish. Additional energy cost (swimming, respiratory and more energy is required to main homeostasis) and metabolic load are one of the most important consequences of metal toxicity which can lead to disruption of oxidative metabolism and enhanced anaerobiosis. Metals like Cd, Pb, Zn, Hg may also interact competitively with elements that are important for normal functioning of body, hamper the structure of biomolecules and biological membranes, interfere with the ATP synthesis process etc (Gashkina, 2024). Toxic substances mimic the environmental stressors effects (hypoxia, starvation) which may decrease rates of feeding and ventilation (Tuffnail et al., 2009). Studies have shown that toxic metals like Cu can disturb the normal process of lipid metabolism, which effects the TCA cycle, lipid content, lipogenic and isocitrate dehydrogenase enzymes activity in the liver of yellow catfish juveniles (Chen et al. 2013).

1. **CONCLUSION**

In conclusion, the effect of aquatic pollution on the embryonic and larval development of fish is a complex and multifaceted issue with far-reaching ecological consequences. The presence of pollutants directly or indirectly in aquatic systems generates measurable environmental and financial repercussions. The ecological effects include pollutant buildup in biota, a significant rise in pollution-related reproduction, developmental defects, and other crucial alterations in biochemical or physiological processes in fish and invertebrates. Heavy metal intoxication lowers embryo survival and hatching success, with most deformed embryos and some typically developed ones perishing during embryonic development. Fish in their initial stages are frequently more vulnerable to pollutants than mature ones. Microplastics can impair fish larvae and embryos' normal growth, resulting in lower food intake, digestive efficiency, and changed energy allocation. Ingestion of microplastics can contribute to lower growth rates and developmental delays in fish larvae. Moreover, microplastics can change the gut microbiome in fish, a crucial regulator of metabolic activities. This can contribute to reduced metabolic efficiency and decreased food absorption, particularly in omnivorous species where MPs stay longer in the gastrointestinal tract than carnivorous or filter-feeding species. Fish embryonic development is closely related to pollution load in the natural environment, with developmental defects in fish embryos exposed to polycyclic aromatic hydrocarbons (PAHs) and crude oil. These pollutants can induce physical abnormalities, greater disease susceptibility, and diminished general fitness, all indicating impaired growth and development. Pollutants in fish can dramatically impair hatching, embryonic growth, and survival. The early stages of fish are sensitive to water pollution, which can lead to the accumulation of metals like Zn, Pb, and Cd, which can severely influence gamete formation and embryonic development. These contaminants can also impact the spermatozoa motility time, which is crucial for successful fertilization. The harmful impact of metal pollution is mainly connected to osmotic disturbances and changes in the production and activity of enzymes. Methylmercury can slow embryonic development, lengthening the early stages and suppressing embryogenesis at the gastrulation stage or complete organogenesis. Overall, pollution has a deleterious influence on fish health and overall well-being. Top of Form

Considering all these negative impacts of toxic metals or pollutants, preventive measure should be taken to control the entry of these contaminants into the aquatic ecosystem. Aquaculture enterprises also need to know these effects and should develop methods of avoiding such negative impacts. Domestic and industrial effluents are also the source of contaminants, so these should be treated before releasing into natural water bodies. Further studies are needed to know and understand the different mechanisms underlying such alterations at each stage and at different concentrations for various toxic contaminants. How these contaminants compete with each other or there is any selection of these metals by the species. Further studies need to be done to understand this hypothesis. There is also a need for the development of standard protocols to treat waste water from industries, domestic and aquacultural waste to maintain these contaminants under desired levels. A set of prerequisite government policies which governs these policies would be helpful to monitor the sources of pollution.

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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