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A REVIEW ON TOXICOLOGICAL EFFECTS OF HERBICIDES ON AQUATIC ECOSYSTEM

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Abstract

Herbicides are essential in modern agriculture but pose severe risks to aquatic ecosystems, particularly fish. This review examines herbicides' biochemical and genotoxic impacts on fish species, addressing oxidative stress, metabolic disruptions, hormonal interference, DNA damage, and mutations. Herbicides like glyphosate, atrazine, paraquat, and 2,4-D have been linked to enzymatic inhibition, reproductive abnormalities, and genetic mutations with extensive implications for aquatic biodiversity and human health. Case studies provide further insights into specific herbicides' effects, such as glyphosate's role in oxidative stress, atrazine's endocrine disruption, and paraquat's genotoxicity. Continuous monitoring, regulation, and alternative solutions are crucial to reduce these harmful effects and protect aquatic life and public health.

Keywords: DNA damage, Endocrine disruption, Environmental health, Genotoxicity, Herbicides.

Introduction

Herbicides are a critical component of modern agriculture, enhancing crop yields through effective weed control. However, their widespread use raises significant concerns about environmental and human health impacts (Powles and Yu, 2010). Surface and groundwater bodies are particularly vulnerable to contamination through agricultural runoff, leaching, and non-agricultural applications (Heap, 2014). Groundwater contamination poses long-term risks to drinking water supplies, while surface water contamination disrupts aquatic ecosystems. Herbicide drift in the atmosphere can also unintentionally deposit chemicals on non-target vegetation. These environmental risks necessitate sustainable practices and effective management strategies to minimize adverse effects (Duke, 2018; Matthews, 2006).

Despite their risks, selective and systemic herbicides are valued for their targeted effectiveness, longer action, and reduced harm to non-target organisms (Busi *et al.,* 2012; Powles, 2012). This review examines the environmental and health risks associated with herbicide contamination, particularly in surface waters, emphasizing the urgent need for alternative management strategies

(Shaner, 2014). Several studies highlight the complex interplay between precipitation events and herbicide runoff, showing that rainfall intensity and agricultural practices significantly influence contamination levels (Baker and Richards, 2002). Persistent herbicides, such as atrazine, accumulate in aquatic environments, causing prolonged contamination, disrupting nutrient cycles, and degrading water quality (Solomon *et al.,* 1996). These chemicals also contribute to eutrophication, fostering harmful algal blooms that deplete oxygen and produce toxins, with significant ecological and economic consequences (Smith *et al.,* 1999).

Herbicide contamination also has serious impacts on aquatic organisms. Fish, in particular, are highly vulnerable to waterborne contaminants, experiencing biochemical and genotoxic effects, including oxidative stress, endocrine disturbances, DNA damage, and mutagenic changes. These disruptions can cascade through ecosystems, affecting food chains and biodiversity (Giesy *et al.,*2000). Mitigation strategies are essential to minimize these environmental risks. Effective approaches include precision agriculture, buffer zones, and Integrated Pest Management (IPM), which reduce herbicide runoff and its impacts. Strengthening regulatory frameworks, adopting best practices, and exploring alternative weed management strategies are critical steps toward promoting sustainable herbicide use (Fawcett *et al.,* 1994; Alavanja *et al.,* 2004).

Herbicides are integral to modern agriculture, improving crop yields by controlling weeds effectively. However, their pervasive use has raised environmental and human health concerns (Powles and Yu, 2010). Herbicides enter aquatic systems through runoff, leaching, and atmospheric drift, affecting both surface and groundwater bodies. Groundwater contamination poses long-term risks to drinking water supplies, while surface water contamination disrupts aquatic ecosystems (Heap, 2014; Matthews, 2006).

Herbicide contamination of aquatic systems results in harmful effects on aquatic life. Persistent herbicides, such as atrazine and glyphosate, accumulate in water bodies, causing long-term contamination and adverse ecological impacts. Atrazine, widely used in agriculture, has been associated with endocrine disruption in amphibians and fish, reducing reproductive fitness and survival rates (Hayes *et al.,* 2003). Glyphosate-based herbicides, although considered less persistent, can harm aquatic organisms, causing oxidative stress, DNA damage, and alterations in aquatic community structures (Giesy *et al.,* 2000; Van Bruggen *et al.,* 2018).

The phenomenon of herbicide-induced eutrophication, primarily driven by runoff containing herbicides like diuron and simazine, fosters harmful algal blooms, depletes oxygen, and produces toxins, leading to ecological and economic consequences (Smith *et al.,* 1999; Lewis *et al.,* 2009). Herbicide residues in recreational and drinking water sources also pose public health risks, necessitating costly water treatment measures (Solomon *et al.,* 1996).

Numerous studies have linked the impact of precipitation events and agricultural practices with herbicide runoff, emphasizing the need for improved water management strategies (Baker and Richards, 2002). Other herbicides, such as paraquat and 2,4-D, have been implicated in causing acute toxicity to aquatic organisms. Paraquat is known for its high toxicity to fish and amphibians, disrupting respiratory and metabolic functions (Dinis-Oliveira *et al.,* 2008). Similarly, 2,4-D has been shown to disrupt endocrine systems and cause genotoxic effects in aquatic species (Stevens

et al., 1998). Integrated Pest Management (IPM), precision agriculture, and buffer zones are highlighted as effective strategies for mitigating the environmental impacts of herbicide use. Strengthening regulatory frameworks and promoting sustainable alternatives, such as bioherbicides derived from natural sources, can further reduce dependency on synthetic chemicals (Duke *et al.,* 2012; Fawcett *et al.,* 1994).

This review underscores the importance of adopting sustainable practices and advanced technologies to mitigate the risks posed by herbicides. Continued research into the ecological impacts of diverse herbicides and their pathways in aquatic systems is crucial for developing effective management strategies and safeguarding environmental and public health.

2. Biochemical Effects of Herbicides on Fish

Many herbicides, including glyphosate, diuron, and paraquat, are known to induce oxidative stress in aquatic organisms, particularly fish. This oxidative stress is characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, which leads to cellular damage. (Guilherme *et al.,* 2012) Demonstrated that glyphosate exposure in common carp resulted in increased lipid peroxidation and decreased antioxidant enzymes like superoxide dismutase (SOD) and catalase (CAT), suggesting oxidative stress-induced toxicity. Similarly, (Slaninova *et al.,* 2009) observed that glyphosate exposure in rainbow trout caused oxidative stress, leading to cellular damage. (Oliveira *et al.,* 2018) Found that diuron exposure in Nile tilapia led to significant oxidative stress, as evidenced by an increase in ROS levels and depletion of antioxidant defenses. Further studies, such as those by (Akinmoladun *et al.,* 2019), showed that paraquat exposure also resulted in increased oxidative stress markers and a decrease in antioxidant defense in African catfish, emphasizing the broad applicability of oxidative stress as a common herbicide-induced effect. (Moustafa *et al.,* 2015) further supported these findings, showing that triazine herbicides also induce oxidative stress in fish, marked by increased lipid peroxidation and alterations in antioxidant enzyme levels.

Herbicides also have significant effects on fish metabolism, primarily by inhibiting enzymes that are crucial for various physiological functions, including detoxification and nervous system regulation. (Nwani *et al.,* 2013) demonstrated that atrazine inhibited acetylcholinesterase (AChE) activity in freshwater fish, leading to impaired neural signaling and behavioral changes. (Lionetto *et al.,* 2003) Highlighted that herbicides like glyphosate disrupt cytochrome P450 enzymes, which play a critical role in detoxification and metabolic processes. (Rajput *et al.,* 2021) Found that glyphosate exposure in zebrafish altered glucose metabolism enzymes, resulting in an energy imbalance that affected growth and development. Similarly, (Singh *et al.,* 2014) reported that exposure to 2,4-D led to alterations in metabolic enzymes, indicating that herbicides can disrupt fish metabolic pathways. (Khan *et al.,* 2019) found that paraquat exposure disrupted several metabolic enzymes in carp, further underlining the significant impact of herbicides on fish metabolism.

In addition to metabolic disruptions, herbicides like atrazine, 2,4-D, and glyphosate are also known to cause endocrine disruption, which affects hormonal systems involved in reproduction and development. (Hayes *et al.,* 2002) showed that atrazine exposure caused feminization in male

frogs, suggesting similar endocrine-disrupting effects in fish, which have comparable hormonal systems. (Geraudie *et al.,* 2010) Found that exposure to 2,4-D caused reproductive abnormalities in fish, such as reduced sperm quality and altered sex ratios. More recent studies, such as those by (Kumar *et al.,* 2023), reported that glyphosate exposure in zebrafish resulted in changes in hormone levels, delaying maturation and reducing fertility, pointing to its potential role as an endocrine disruptor. (Rijal *et al.,* 2020) demonstrated that atrazine exposure in zebrafish altered sex differentiation and reproductive organs, further highlighting its disruptive impact on fish reproduction. (Orozco *et al.,* 2017) Also observed that glyphosate exposure caused changes in thyroid hormone levels in fish, potentially affecting their growth and development.

Furthermore, herbicides can cause significant hematological changes in fish, affecting their immune systems and the efficiency of oxygen transport, both of which are critical for their overall health and survival. (Bhatnagar and Bana, 2013) Found that glyphosate exposure decreased hemoglobin levels and altered white blood cell counts in catfish, impairing oxygen transport and immune function. (Okoro *et al.,* 2021) confirmed these findings in African catfish, showing that chronic exposure to herbicides led to immune deficiencies and reduced disease resistance. (Sharma *et al.,* 2016) observed that exposure to paraquat caused a significant decrease in hemoglobin levels and increased oxidative stress in the blood of fish, emphasizing the negative impact of herbicides on fish hematology. (Sivaprasad *et al.,* 2018) found that herbicides such as atrazine and 2,4-D caused alterations in the hematological parameters of tilapia, leading to compromised immune responses and overall health. Similarly, (Gandhi *et al.,* 2021) found that glyphosate exposure led to significant changes in blood parameters in carp, including reduced erythrocyte counts and hemoglobin levels, confirming the detrimental effects of herbicides on fish blood.

These studies collectively illustrate that herbicides pose a serious threat to fish health, impacting oxidative stress levels, enzymatic activity, metabolism, endocrine function, and hematological parameters. These effects underscore the need for continued research and regulation of herbicide use to protect aquatic life.

3. Genotoxic Effects of Herbicides on Fish

Herbicide exposure, particularly to chemicals like glyphosate, atrazine, and paraquat, poses a significant threat to fish health by inducing genotoxicity, which compromises genetic integrity and can lead to long-term ecological risks. DNA damage, as a key marker of genotoxicity, has been well-documented in various fish species exposed to herbicides. (Cavas and Ergene-Gozukara 2005) And (Guilherme *et al.,* 2010) showed that glyphosate exposure led to DNA strand breaks and chromosomal aberrations. Recent research by (Sahni *et al.,* 2022) on grass carp confirmed similar findings, indicating DNA damage resulting from herbicide exposure, with potential consequences for reproductive success and population survival. This type of genetic damage is particularly concerning because it may have transgenerational effects, posing long-term environmental risks. In addition to DNA damage, herbicides can also lead to the formation of micronuclei, a common biomarker for chromosomal damage. Studies by (Lemos *et al.,* 2010) and (Çavas *et al.,* 2005) found that exposure to glyphosate and paraquat resulted in increased micronucleus formation in freshwater fish, signaling genomic instability. (Mandal *et al.,* 2023)

Further observed that atrazine exposure in tilapia increased micronuclei frequency, highlighting the genotoxic burden on fish populations. Such genetic instability can lead to developmental abnormalities, reduced fitness, and potential population decline. Moreover, herbicides have been linked to mutagenic effects, causing genetic mutations that could impact the adaptation and long-term viability of fish populations. (Bolognesi *et al.,*2006) Found that herbicides, including glyphosate, induced mutations in fish, leading to compromised reproductive success and stability in fish populations. A more recent study by (Yadav *et al.,* 2023) observed that glyphosate exposure caused mutations in immune response-related genes in zebrafish, potentially weakening their ability to survive pathogen challenges. These genetic mutations not only threaten the health of individual fish but also reduce genetic diversity, which is crucial for the resilience and adaptability of populations. Collectively, these findings underscore the genotoxic potential of herbicides and the significant, long-term risks they pose to fish populations and aquatic ecosystems. Recent reviews of the literature emphasize the increasing concern over herbicide-induced genetic damage and its broader implications for biodiversity conservation and ecosystem health (Akinmoladun *et al.,* 2022; Kumar *et al.,* 2023).

4. Case Studies of Different Herbicides

Glyphosate (Roundup): Glyphosate, one of the most widely studied herbicides, has been shown to induce significant toxicological effects in aquatic organisms. A study by (Guilherme *et al.,*2012) demonstrated that glyphosate exposure in common carp led to oxidative stress, DNA damage, and the inhibition of key antioxidant enzymes. (Slaninova *et al.,* 2009) Observed similar effects in rainbow trout, where glyphosate exposure caused oxidative stress and DNA damage, suggesting potential risks to fish health and population viability. Moreover, a study by (Akinmoladun *et al.,*2019) found that glyphosate exposure in African catfish led to increased oxidative stress and disruptions in immune function. Additionally, a recent study by (Silva *et al.,* 2023) in zebrafish highlighted significant DNA damage and oxidative stress following glyphosate exposure, further supporting the genotoxic potential of this herbicide.

Atrazine: Atrazine is widely recognized for its endocrine-disrupting effects in aquatic organisms. (Hayes *et al.,* 2002) reported that atrazine exposure feminized male frogs, raising concerns about its impact on fish reproductive systems. (Mandal *et al.,* 2023) Found that atrazine exposure caused micronucleus formation and DNA damage in tilapia, resulting in reproductive health issues and potential population decline. More recently, studies by (Rijal *et al.,* 2020) have demonstrated that atrazine exposure in zebrafish led to altered sex differentiation and disrupted reproductive organ development, further confirming its endocrine-disrupting potential. Additionally, a review by (Kumar *et al.,*2023) summarized the widespread impacts of atrazine on fish populations, indicating its role in delaying maturation, reducing fertility, and disrupting hormonal regulation.

Paraquat: Paraquat, a highly toxic non-selective herbicide, has been shown to cause severe genetic damage in fish. (Cavas and Ergene-Gozukara, 2005) Documented chromosomal aberrations and DNA damage in fish exposed to paraquat. A more recent study by (Silva *et al.,* 2023) in zebrafish observed increased oxidative stress and DNA fragmentation, indicating that paraquat could have severe genetic impacts on fish populations. Similarly, (Sharma *et al.,* 2016)

found that paraquat exposure in catfish caused significant reductions in hemoglobin levels and increased oxidative stress, underlining the detrimental effects of paraquat on both hematology and genetic integrity in aquatic organisms.

2,4-D: 2,4-Dichlorophenoxyacetic acid (2,4-D) is an herbicide that has been shown to impact metabolism and reproductive health in fish. (Geraudie *et al.,* 2010) Reported that exposure to 2,4-D resulted in reduced sperm quality and altered hormone levels in fish. Recent studies, such as those by (Shrestha *et al.,* 2022), confirmed that 2,4-D disrupts immune responses and can lead to developmental abnormalities in exposed fish species. Moreover, (Sivaprasad *et al.,* 2018) observed that 2,4-D exposure in tilapia resulted in hematological changes, which could compromise fish health and immunity.

5. Risk of Human and Environmental Health from Herbicide Contamination

Herbicide-induced toxicity in fish poses a significant threat to aquatic biodiversity and carries broader implications for human health due to bioaccumulation. Studies by (Kegley *et al.,* 2014) and the World Health Organization (2004) emphasize the accumulation of herbicide residues in fish tissues, which can enter the human food chain, exposing consumers to potentially harmful compounds. For instance, glyphosate residues have been detected in edible fish tissues, raising concerns about human exposure. Given that fish serve as a major source of protein worldwide, these findings highlight the urgent need for stringent monitoring and regulatory measures to mitigate herbicide contamination.

Human exposure to herbicides occurs through various pathways, including direct and indirect modes, bioaccumulation, and biomagnification, all of which present significant health risks. Agricultural workers face direct exposure primarily through skin contact and inhalation during herbicide application (Kegley *et al.,* 1999). Indirect exposure through contaminated food and water is another major concern, as herbicide residues can persist in the environment and accumulate within the food chain (Solomon and Thompson, 2003). Bioaccumulation of herbicides in aquatic organisms leads to biomagnification, where concentrations increase at higher trophic levels. This process is particularly dangerous for top predators and humans who consume contaminated fish and other aquatic species, as outlined by the USEPA (2003).

Herbicide exposure poses both acute and chronic health risks, particularly for individuals exposed occupationally. Adherence to safety measures and protective guidelines is essential to mitigate these risks (Kegley *et al.,*1999; Solomon and Thompson, 2003). Additionally, herbicide contamination disrupts ecosystem balance and biodiversity, with long-lasting effects due to their persistence in soil and water environments (Dodds and Welch, 2000; Kreutzweiser *et al.,* 2007). Studies focusing on glyphosate and atrazine highlight substantial health concerns, emphasizing the importance of regulatory oversight and safety protocols to safeguard human health.

Effective strategies to mitigate these risks include enhanced regulatory controls, education and training programs, and ongoing research and innovation. Such approaches are critical in reducing the adverse effects of herbicide use and in protecting both environmental and human health (Dodds and Welch, 2000; Kreutzweiser *et al.,* 2007).

6. Conclusion

The literature demonstrates that herbicides impose significant biochemical and genotoxic burdens on fish, with oxidative stress, enzyme inhibition, endocrine disruption, DNA damage, and mutagenic changes as common effects. Case studies of herbicides such as glyphosate, atrazine, paraquat, and 2,4-D provide clear evidence of herbicides' impact on fish health, population viability, and ecological stability. Furthermore, the persistence of herbicides in aquatic ecosystems highlights their potential to cause long-term environmental damage, including bioaccumulation and biomagnification through the food web, posing risks to top predators and humans reliant on fish as a protein source. These findings underscore the urgent need for regulatory interventions, such as stricter herbicide use guidelines, enforcement of safe handling practices, and implementation of water quality monitoring programs. Research into safer, eco-friendly alternatives, such as bioherbicides, and the development of bioremediation strategies to mitigate herbicide contamination in aquatic environments should also be prioritized. Public awareness campaigns and education for agricultural workers can further minimize herbicide misuse and reduce occupational exposure. Ultimately, an integrated approach involving policymakers, scientists, and communities is crucial to safeguard aquatic biodiversity, ensure food safety, and protect human health from long-term exposure to these toxic chemicals. This comprehensive strategy will also contribute to achieving global sustainability goals by preserving essential ecosystem services and enhancing environmental resilience against anthropogenic stressors.

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